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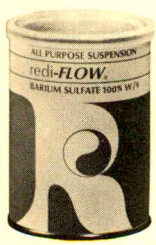
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VOL. 121, NO. 4

CONTENTS OF VOLUME 121

MAY, 1974, NUMBER 1

Review of Roentgenographic Bone Demineralization Studies of the Ge ini Space Flights. *George P. Vose, M.S.* 1
Cleidocranial Dysostosis: A Review of 40 New Cases. *J. Luther Jarvis, M.D., and Theodore E. Keats, M.D.* 5
Condensing Osteitis of the Clavicle: A New Entity. *Anne C. Brower, M.D., Donald E. Sweet, M.D., and Theodore E. Keats, M.D.* 17
Diabetic Osteoarthropathy: Clinical and Roentgenographic Observations in 90 Cases. *Melvin E. Clouse, M.D., Herbert F. Gramm, M.D., Merle Legg, M.D., and Thomas Flood, M.D.* 22
Similar Bone Manifestations of Scleroderma and Rheumatoid Arthritis. *Jack G. Rabinowitz, M.D., Jack Twersky, M.D., and Maria Guttadauria, M.D.* 35
Homocystinuria Due to Cystathionine Synthase Deficiency: Clinical-Roentgenologic Correlations. *Paula W. Brill, M.D., Harold A. Mitty, M.D., and Gerald .. Gaull, M.D.* 45
Complications of Total Hip Replacement: A Roentgen Eval ation. *Maurice F. Mullins, M.B., B.Ch., Richard N. Sutton, M.D., M.S.E.E., and Gwilym S odwick, M.D.* 55
The Painful Charnley Total Hip Replacement. *Car olinskas, M.D., Robert E. Campbell, M.D., and Richard H. Rothman, M.D., Ph.D.* 61
"Clipping Injury" Fracture of the Epiphysis in e Adolescent Football Player: An Occult Lesion of the Knee. *Lee F. Rogers, M.D., Stanley Jone , M.D., A. Ross Davis, M.D., and Geral Dietz, M.D.* 69
Arthrography of the Lateral Meniscus rray K. Dalinka, M.D., James F. Lally, M.D., and Vijay K. Gohel, M.D. 79
Synovial Recesses in Knee Ar aphy. *Charles E. Montgomery, M.D.* 86
Arthrography in Rupture of uprapatellar Bursa with Pseudocyst Formation. *Adah M. Duncan, M.D.* 89
Use of Xeroradiography i ruciate Ligament Injuries. *Harry J. Griffiths, M.D., and Carl J. D'Orsi, M.D.* 94
Lipohemarthrosis. *Peter H. Arger, M.D., Paul E. Oberkircher, M.D., and Wallace T. Miller, M.D.* 97
The Distal Anterior F emoral Metaphyseal Defect: An Anatomic Variant that May Simulate Disease. *Theodore E. K ats, M.D.* 101
Multifocal Oste Osteoma: A Case Report and Review of the Literature. *Adam Greenspan, M.D., Alberto T zzabel, M.D., and David Bryk, M.D.* 103
Calcificatio Cooper's Ligament. *John R. Steinfeld, M.D., K. E. Schuit, Ph.D., and Theodore E. Keats, M.D.* 107
Extravasa n of Calcium Gluconate As a Cause of Soft Tissue Calcification in Infancy. *Paul E. Berger, M.* *Kathleen P. Heidelberger, M.D., and Andrew K. Poznanski, M.D.* 109
Prima ymphangioma of the Calvarium. *Mordecai Kopperman, M.D., and John E. Antoine, M.D.* 118
Mul Calvarial "Doughnut Lesions": A Case Report. *Peter M. Royen, M.D., and M. B. Ozonoff, D.* 121
The axillary Artery in Craniofacial Pathology. *William E. Allen, III, M.D., E. Leon Kier, M.D., .d Stephen L. G. Rothman, M.D.* 124
Os Supra Petrosum of Meckel. *Guido Currarino, M.D., and Arthur Weinberg, M.D.* 139
Patterns of Injury and Significance of Uncommon Fractures in the Battered Child Syndrome. *Marvin S. Kogutt, M.D., Leonard E. Swischuk, M.D., and Charles J. Fagan, M.D.* 143
Pharmacologic Aids in Angiograpny of the Upper Extremity. *Harvey I. Wilner, M.D., Roger Kay, D.O., and Bradley A. Eisenbrey, M.D.* 150
Gangliosidosis (GM_1): A Re-Evaluation of the Vertebral Deformity. *Jack G. Rabinowitz, M.D., and Michael Sacher, M.D.* 155
Size of Pelvic Bony Outlet in Renal Agenesis. *Richard A. Fellows, M.D., Walter E. Berdon, M.D., and David H. Baker, M.D.* 159
Neonatal Transplacental Rubella Syndrome: Its Effect on Normal Maturation of the Diaphysis. *Joseph P. Whalen, M.D., Patricia Winchester, M.D., Lennart Krook, D.V.M., Ph.D., Neil O'Donohue, M.D., Renate Dische, M.D., and Eladio Nunez, Ph.D.* 166
Bilateral Pseudotumors of the Pelvis in a Patient with Christmas Disease: With Notes on Localization by Radioactive Scanning and Ultrasonography. *Charles D. Forbes, M.D., M.R.C.P., Brian Moule, M.B., B.Ch., D.M.R.D., F.F.R., Mary Grant, M.B., Ch.B., D.M.R.D., William R. Greig, M.D., Ph.D., F.R.C.P., and Colin R. M. Prentice, M.D., M.R.C.P.* 173

Technetium 99m Labeled Diphosphonate Bone Scanning in Paget's Disease. *Stephen W. Miller, M.D., Frank P. Castronovo, Jr., Ph.D., Henry P. Pendergrass, M.D., and Majic S. Potsaid, M.D.* 177
Comparison of Tc^{99m} Polyphosphate and F^{18} for Bone Imaging. *D. A. Weber, Ph.D., J. W. Keyes, Jr., M.D., S. Landman, M.D., an[illegible] G. A. Wilson, M.D.* 184
Officers 191
Editorial
Continuing Medical Education: Implications Concerning Recertification, Re-examination and Relicensure. *B. G. Brogden, M.D.* 192
News Items 197
Book Reviews 200
Books Received 201
Society Proceedings 202
Abstracts of Radiologic Literature 209

June, 1974, Number 2

Cervical Esophageal Webs—A Roentgen-a[illegible]atomic Correlation. *James L. Clements, Jr., M.D., George W. Cox, M.D., William E. Torres, M.D., and H. Stephen Weens, M.D.* 221
Enhanced Visualization of Esophageal Varices by [illegible]Buscopan. *Chung-I Liu, M.D.* 232
Nonobstructive Mucosal Diaphragms or Rings of t[illegible]astric Antrum in Adults. *Gary G. Ghahremani, M.D.* 236
Acquired Paraesophageal and Disparaesophageal Hernias Complications of Hiatal Hernia Repair. *Terry Hoyt, M.D., and Myo M. Kyaw, M.D.* 248
Spontaneous Reduction of Hernia "En Masse." *Louis S. Parvey, M.D., Elliot Himmelfarb, M.D., and Jack Rabinowitz, M.D.* 252
Diagnostic Value of Hypotonic Duodenography. *Charles L. Rennell, M[illegible]* 256
Hypotonic Roentgenography with Glucagon. *Roscoe E. Miller, M.D., [illegible]y M. Chernish, M.D., Jovitas Skucas, M.D., Bernard D. Rosenak, M.D., and Bruce E. Rodda, [illegible]* 264
Collagenous Sprue. *H. Walter Pepper, M.D., Lloyd L. Brandborg, M.D., Jon L. [illegible]hanser, M.D., Henry I. Goldberg, M.D., and Albert A. Moss, M.D.* 275
A Roentgenographic Classification of Diffuse Diseases of the Small Intestine Pre[illegible]enting with Malabsorption. *Timothy E. Tully, M.D., and Samuel B. Feinberg, M.D.* 283
Those Other Types of Enterocolitis. *Timothy E. Tully, M.D., and Samuel B. Feinberg, [illegible]M.D.* 291
Clindamycin (Cleocin) Colitis. *Leroy E. Cohen, M.D., Major, MC, Charles J. Smith, M.D., [illegible]Major, MC, James D. Pister, M.D., Captain, MC, and Ralph F. Wells, M.D., Colonel, MC* 301
Inflammatory Pseudotumor of the Small Intestine. *William L. Campbell, M.D., William [illegible]reen, M.D., and William B. Seaman, M.D.* 305
Frequency of Visualization of the Normal Appendix with the Barium Enema Examination. *Ka[illegible]ond P. Sakover, M.D., and Raymond L. Del Fava, M.D.* 312
Oral Examination of the Colon: "The Colonic Cocktail." *Rubem Pochaczevsky, M.D.* 318
The Water Double Contrast Barium Enema Study: A New Method of Roentgen Examination of Colon. *Rubem Pochaczevsky, M.D.* 326
Giant Duodenal Diverticula. *Jack R. Millard, M.D., Fred M. H. Ziter, Jr., M.D., and William [illegible] Slover, M.D.* 334
Giant Sigmoid Diverticulum. *Jack G. Rabinowitz, M.D., Jack Farman, M.D., Ch.B., M.Med., Serge Dallemand, M.D., Jack Twerskey, M.D., and Yale Rosen, M.D.* 338
Large Diverticulum of the Anal Canal: Case Report and Review of the Literature on Anal Canal and Rectal Diverticula. *A. F. Govoni, M.D., C.R.C.P.(C), and Jan J. Smulewicz, M.D.* 344
Evaluation of the Retrogastric Space: Normal Appearance and Variation. *Joseph P. Whalen, M.D., Lewis M. Bader, M.D., and Richard Wolfman, M.D.* 348
"Lower" Gastrointestinal Tract Hemorrhage: New Concepts Based on Arteriography. *William J. Casarella, M.D., Shelby J. Galloway, M.D., Richard N. Taxin, M.D., David A. Follett, M.D., Edward J. Pollack, M.D., and William B. Seaman, M.D.* 357
Ventral Portal Vein. *George Harell, M.D.* 369
Contrast Enhancement of the Mesenteric and Portal Veins Using Intra-arterial Papaverine. *Warren C. Widrich, M.D., David L. Nordahl, M.D., and Alan H. Robbins, M.D.* 374
Use of C-Terminal Octapeptide of Cholecystokinin in Cholecystography. *Jonathan A. Levant, M.D., and Richard A. L. Sturdevant, M.D.* 380
Intrahepatic Arterial-Portal Communications: Angiographic Study. *Yacov Itzchak, M.D., Rafael Adar, M.D., Harry Bogokowski, M.D., Mark Mozes, M.D., and Victor Deutsch, M.D.* 384

Pyogenic Liver Abscess: Angiographic Diagnosis and Treatment by Closed Aspiration. *Stanley B. Novy, M.D., Sidney Wallace, M.D., Arnold M. Goldman, M.D., and Yoram Ben-Menachem, M.D.* 388
Gallbladder Ectopia: A Review of Anomalies of Position. *David E. Blanton, M.D., Charles A. Bream, M.D., and Stanley R. Mandel, M.D., F.A.C.S.* 396
Macro-Regenerating Nodule in the Cirrhotic Liver: Radiologic Features and Differential Diagnosis. *Jack G. Rabinowitz, M.D., Mahesh Kinkabwala, M.D., and Sidney Ulreich, M.D.* 401
Localized Deformity of the Gallbladder Secondary to Hepatic Mass Lesions. *Norman Joffe, M.D., and George O. Babenco, M.D.* 412
Pitfalls of the Arteriographic Differentiation of Intrahepatic and Extrahepatic Masses. *Jon D. Shanser, M.D., Morton G. Glickman, M.D., and Alphonse J. Palubinskas, M.D.* 420
Officers 430
Editorial
Seventy-fifth Annual Meeting of the American Roentgen Ray Society. *Colin B. Holman, M.D.* .. 431
Advance Registration, American Roentgen Ray Society 433
Preliminary Program, American Roentgen Ray Society 436
Workshops on Federal Diagnostic X-Ray Standard. Department of Health, Education, and Welfare 441
News Items 443
Book Review 446
Books Received 446
Society Proceedings 448
Abstracts of Radiologic Literature 455

July, 1974, Number 3

Radiotherapy of Tumors of the Urinary Tract: Hickey Lecture, 1974. *J. A. del Regato, M.D.* 467
Megacalyces: Further Observations and Differentiation from Obstructive Renal Disease. *Lee B. Talner, M.D., and Ruben F. Gittes, M.D.* 473
Roentgenographic Findings in Urinary Tract Tuberculosis: A 10 Year Review. *Stephen A. Kollins, M.D., Glen W. Hartman, M.D., David T. Carr, M.D., Joseph W. Segura, M.D., and Robert R. Hattery, M.D.* 487
Xanthogranulomatous Pyelonephritis. *Michael C. Beachley, M.D., Klaus Ranniger, M.D., and Franz-Joseph Roth, M.D.* 500
Angiography in Unilateral Renal Bleeding with a Negative Urogram. *Harold A. Mitty, M.D., and Howard Goldman, M.D.* 508
Adrenal Tumors Simulating Intrarenal Lesions. *Gary G. Shrago, Major, MC, C. Malcolm McKinnon, Major, MC, and Robert Clark, Major, MC* 518
Diagnosis of Perirenal and Subcapsular Masses: Anatomic-Radiologic Correlation. *Morton A. Meyers, M.D., Joseph P. Whalen, M.D., and John A. Evans, M.D.* 523
Angiographic Features of Alport's Syndrome: Hereditary Nephritis. *Vincent P. Chuang, M.D., and Stewart R. Reuter, M.D.* 539
Acute Renal Failure after Angiography. *Frederich K. Port, M.D., Richard D. Wagoner, M.D., and Richard E. Fulton, M.D.* 544
Arteriography and Phlebography of the Limbs in Planning Shunt and Fistula Operations in Patients with Chronic Renal Failure. *M. Lea Thomas, A. S. Rappaport and A. J. Wing* 551
Congenital Subclavian Steal. *Michael J. Borushok, M.D., Robert I. White, Jr., M.D., Kook Sang Oh, M.D., and John P. Dorst, M.D.* 559
Angulated Views in the Sagittal Plane for Improved Accuracy of Cinecoronary Angiography. *Jacques Lespérance, M.D., Jacques Saltiel, M.D., Robert Petitclerc, M.D., and Martial G. Bourassa, M.D.* 565
New Lordotic Projection for Improved Visualization of the Left Coronary Artery and Its Branches. *Thomas A. Sos, M.D., Jack G. Lee, M.D., David C. Levin, M.D., and Harold A. Baltaxe, M.D.* .. 575
Coronary Arteriography in Left Main Coronary Artery Disease. *Josef Rösch, M.D., Henry De Mots, M.D., Ruza Antonovic, M.D., Shahbudin H. Rahimtoola, M.B., F.R.C.P., Melvin P. Judkins, M.D., and Charles T. Dotter, M.D.* 583
Angiographic Demonstration of the Absence of an Atrial Septal Defect in the Presence of Partial Anomalous Pulmonary Venous Connection. *Thomas A. Sos, M.D., Daniel Tay, M.D., Aaron R. Levin, M.D., M.R.C.P., David C. Levin, M.D., and Harold A. Baltaxe, M.D.* 591
Left Ventricular Angiographic Anatomy of Ostium Primum Defect in the Adult. *Patricia A. Randall, M.D., Daniel J. Goodman, M.D., and John S. Schroeder, M.D.* 597
The Importance of Selective Angiography of Systemic Arterial Supply to the Lungs in Planning Surgical Correction of Pseudotruncus Arteriosus. *David C. Levin, M.D., Harold A. Baltaxe, M.D.,*

Henry P. Goldberg, M.D., Mary Allen Engle, M.D., Paul A. Ebert, M.D., Thomas A. Sos, M.D., and Aaron R. Levin, M.D. ... 606
Bronchocutaneous Fistula As a Late Complication of Permanent Epicardial Pacing. *Charles J. Tegtmeyer, M.D., James G. Hunter, Jr., M.D., and Theodore E. Keats, M.D.* ... 614
Roentgenographic Evaluation of Dissecting Aneurysms of the Aorta. *Michael C. Beachley, M.D., Klaus Ranniger, M.D., and Franz-Joseph Roth, M.D.* ... 617
Continuous Ultrasound "B" Scanning of Abdominal Aortic Aneurysms. *F. Winsberg, M.D., C. Colebeuglet, M.D., and D. S. Mulder, M.D.* ... 626
Double Catheter Technique in Aortography. *John E. Antoine, M.D., Philip J. Middleton, M.D., and Philip W. Carmody, M.D.* ... 634
Radiation Reduction by Means of Low Pulse-Rate Fluoroscopy during Cardiac Catheterization and Coronary Arteriography. *J. H. Grollman, Jr., M.D.* ... 636
Hysterosalpingography in Infertility. *Heun Y. Yune, M.D., Eugene C. Klatte, M.D., Robert E. Cleary, M.D., and Loren Petersen, M.D.* ... 642
Officers ... 652
Editorial
Heuristic Gems from the American Radium Society. *Traian Leucutia, M.D.* ... 653
American Roentgen Ray Society, Section on Instruction ... 661
News Items ... 685
Book Reviews ... 687
Books Received ... 688
Society Proceedings ... 689
Abstracts of Radiologic Literature ... 696

August, 1974, Number 4

Quantitative Thyroid Scanning: A Reliable Thyroid Function Test. *L. Barry Arkles, M.R.A.C.P.* 705
An Unusual Concentration of Radiopertechnetate in a Patient with Thyroid Cancer. *Jacques Lamoureux, M.D., Ph.D.* ... 714
Diagnosis of Barrett's Esophagus with Radioisotopes. *Felipe Gordon, M.D., José Ramírez-Degollado, M.D., Rodrigo Muñoz, M.D., Alfredo Cuaren, M.D., and Luis Landa, M.D.* ... 716
Clinical Evaluation of Preoperative Liver Scintigraphy in Gastric Carcinoma Patients. *Katsushi Watanabe, M.D., Kenjiro Kawahira, M.D., and Keiichi Matsuura, M.D.* ... 720
The Use of Multiple Radionuclide Imaging to Differentiate the Focal Intrahepatic Lesion. *Lawrence R. Muroff, M.D., and Philip M. Johnson, M.D.* ... 728
The Value of Tc^{99m}-Diphosphonate (HEDSPA) as a Skeletal Scanning Agent. *J. Papadimitriou, M.D., M. Vezeridis, M.D., M. Constantinidis, Ph.D., E. Chiotelis, Ph.D., C. Constandinidis, M.D., and C. Tountas, M.D.* ... 735
Pitfalls in Tc^{99m} Polyphosphate Skeletal Imaging. *James H. Thrall, M.D., Nassar Ghaed, M.D., George E. Geslien, M.D., Steven M. Pinsky, M.D., and Merrill C. Johnson, M.D.* ... 739
Influence of Fractionation on Acute and Late Reactions in Vocal Cord Carcinoma. *R. Kenneth Loeffler, M.D.* ... 748
The Results of Split-Course Radiation Therapy in Cancer of the Lung. *Hugh Scruggs, M.D., Anas El-Mahdi, M.D., Sc.D., Richard D. Marks, Jr., M.D., and William C. Constable, M.B., Ch.B., D.M.R.T.* ... 754
Dual Camera Studies of Pulmonary Function with Computer Processing of Data. *Merle K. Loken, M.D., Ph.D., Richard A. Ponto, B.S., Richard S. Kronenberg, M.D., Lawrence E. Williams, Ph.D., and Marvin E. Goldberg, M.D.* ... 761
Extended Mantle Radiotherapy in Hodgkin's Disease and Malignant Lymphoma. *J. E. Marks, M.D., E. M. Moran, M.D., M. L. Griem, M.D., and J. E. Ultmann, M.D.* ... 772
Angiography of the Liver and Spleen in Hodgkin's Disease. *K. Jonsson, M.D., and A. Lunderquist, M.D.* 789
Kaposi's Sarcoma: Experience with Ten Cases. *Steven G. Mann, M.D.* ... 793
Urographic Manifestations of Metastatic Melanoma. *Harvey M. Goldstein, M.D., Stephen Kaminsky, M.D., Sidney Wallace, M.D., and Douglas E. Johnson, M.D.* ... 801
The Increased Incidence of Carcinoma of the Colon following Ureterosigmoidostomy. *Randall S. Preissig, M.D., William F. Barry, Jr., M.D., and Richard G. Lester, M.D.* ... 806
Detection and Localization of an Occult Vesicoenteric Fistula. *Edward K. Prokop, M.D., E. Ulric Buddemeyer, Sc.D., H. William Strauss, M.D., and Henry N. Wagner, Jr., M.D.* ... 811
Inferior Vena Caval Extension of Renal Carcinoma: A Lost Cause?: Roentgenographic and Pathologic Findings in Surgical Patients. *David L. McCullough, M.D., and Lee B. Talner, M.D.* ... 819

Pyogenic Liver Abscess: Angiographic Diagnosis and Treatment by Closed Aspiration. *Stanley B. Novy, M.D., Sidney Wallace, M.D., Arnold M. Goldman, M.D., and Yoram Ben-Menachem, M.D.* 388
Gallbladder Ectopia: A Review of Anomalies of Position. *David E. Blanton, M.D., Charles A. Bream, M.D., and Stanley R. Mandel, M.D., F.A.C.S.* 396
Macro-Regenerating Nodule in the Cirrhotic Liver: Radiologic Features and Differential Diagnosis. *Jack G. Rabinowitz, M.D., Mahesh Kinkabwala, M.D., and Sidney Ulreich, M.D.* 401
Localized Deformity of the Gallbladder Secondary to Hepatic Mass Lesions. *Norman Joffe, M.D., and George O. Babenco, M.D.* 412
Pitfalls of the Arteriographic Differentiation of Intrahepatic and Extrahepatic Masses. *Jon D. Shanser, M.D., Morton G. Glickman, M.D., and Alphonse J. Palubinskas, M.D.* 420
Officers 430
Editorial
Seventy-fifth Annual Meeting of the American Roentgen Ray Society. *Colin B. Holman, M.D.* .. 431
Advance Registration, American Roentgen Ray Society 433
Preliminary Program, American Roentgen Ray Society 436
Workshops on Federal Diagnostic X-Ray Standard. Department of Health, Education, and Welfare 441
News Items 443
Book Review 446
Books Received 446
Society Proceedings 448
Abstracts of Radiologic Literature 455

July, 1974, Number 3

Radiotherapy of Tumors of the Urinary Tract: Hickey Lecture, 1974. *J. A. del Regato, M.D.* 467
Megacalyces: Further Observations and Differentiation from Obstructive Renal Disease. *Lee B. Talner, M.D., and Ruben F. Gittes, M.D.* 473
Roentgenographic Findings in Urinary Tract Tuberculosis: A 10 Year Review. *Stephen A. Kollins, M.D., Glen W. Hartman, M.D., David T. Carr, M.D., Joseph W. Segura, M.D., and Robert R. Hattery, M.D.* 487
Xanthogranulomatous Pyelonephritis. *Michael C. Beachley, M.D., Klaus Ranniger, M.D., and Franz-Joseph Roth, M.D.* 500
Angiography in Unilateral Renal Bleeding with a Negative Urogram. *Harold A. Mitty, M.D., and Howard Goldman, M.D.* 508
Adrenal Tumors Simulating Intrarenal Lesions. *Gary G. Shrago, Major, MC, C. Malcolm McKinnon, Major, MC, and Robert Clark, Major, MC* 518
Diagnosis of Perirenal and Subcapsular Masses: Anatomic-Radiologic Correlation. *Morton A. Meyers, M.D., Joseph P. Whalen, M.D., and John A. Evans, M.D.* 523
Angiographic Features of Alport's Syndrome: Hereditary Nephritis. *Vincent P. Chuang, M.D., and Stewart R. Reuter, M.D.* 539
Acute Renal Failure after Angiography. *Frederich K. Port, M.D., Richard D. Wagoner, M.D., and Richard E. Fulton, M.D.* 544
Arteriography and Phlebography of the Limbs in Planning Shunt and Fistula Operations in Patients with Chronic Renal Failure. *M. Lea Thomas, A. S. Rappaport and A. J. Wing* 551
Congenital Subclavian Steal. *Michael J. Borushok, M.D., Robert I. White, Jr., M.D., Kook Sang Oh, M.D., and John P. Dorst, M.D.* 559
Angulated Views in the Sagittal Plane for Improved Accuracy of Cinecoronary Angiography. *Jacques Lespérance, M.D., Jacques Saltiel, M.D., Robert Petitclerc, M.D., and Martial G. Bourassa, M.D.* 565
New Lordotic Projection for Improved Visualization of the Left Coronary Artery and Its Branches. *Thomas A. Sos, M.D., Jack G. Lee, M.D., David C. Levin, M.D., and Harold A. Baltaxe, M.D.* .. 575
Coronary Arteriography in Left Main Coronary Artery Disease. *Josef Rösch, M.D., Henry De Mots, M.D., Ruza Antonovic, M.D., Shahbudin H. Rahimtoola, M.B., F.R.C.P., Melvin P. Judkins, M.D., and Charles T. Dotter, M.D.* 583
Angiographic Demonstration of the Absence of an Atrial Septal Defect in the Presence of Partial Anomalous Pulmonary Venous Connection. *Thomas A. Sos, M.D., Daniel Tay, M.D., Aaron R. Levin, M.D., M.R.C.P., David C. Levin, M.D., and Harold A. Baltaxe, M.D.* 591
Left Ventricular Angiographic Anatomy of Ostium Primum Defect in the Adult. *Patricia A. Randall, M.D., Daniel J. Goodman, M.D., and John S. Schroeder, M.D.* 597
The Importance of Selective Angiography of Systemic Arterial Supply to the Lungs in Planning Surgical Correction of Pseudotruncus Arteriosus. *David C. Levin, M.D., Harold A. Baltaxe, M.D.,*

Henry P. Goldberg, M.D., Mary Allen Engle, M.D., Paul A. Ebert, M.D., Thomas A. Sos, M.D., and Aaron R. Levin, M.D. ... 606
Bronchocutaneous Fistula As a Late Complication of Permanent Epicardial Pacing. *Charles J. Tegtmeyer, M.D., James G. Hunter, Jr., M.D., and Theodore E. Keats, M.D.* ... 614
Roentgenographic Evaluation of Dissecting Aneurysms of the Aorta. *Michael C. Beachley, M.D., Klaus Ranniger, M.D., and Franz-Joseph Roth, M.D.* ... 617
Continuous Ultrasound "B" Scanning of Abdominal Aortic Aneurysms. *F. Winsberg, M.D., C. Cole-Beuglet, M.D., and D. S. Mulder, M.D.* ... 626
Double Catheter Technique in Aortography. *John E. Antoine, M.D., Philip J. Middleton, M.D., and Philip W. Carmody, M.D.* ... 634
Radiation Reduction by Means of Low Pulse-Rate Fluoroscopy during Cardiac Catheterization and Coronary Arteriography. *J. H. Grollman, Jr., M.D.* ... 636
Hysterosalpingography in Infertility. *Heun Y. Yune, M.D., Eugene C. Klatte, M.D., Robert E. Cleary, M.D., and Loren Petersen, M.D.* ... 642
Officers ... 652
Editorial
Heuristic Gems from the American Radium Society. *Traian Leucutia, M.D.* ... 653
American Roentgen Ray Society, Section on Instruction ... 661
News Items ... 685
Book Reviews ... 687
Books Received ... 688
Society Proceedings ... 689
Abstracts of Radiologic Literature ... 696

August, 1974, Number 4

Quantitative Thyroid Scanning: A Reliable Thyroid Function Test. *L. Barry Arkles, M.R.A.C.P.* 705
An Unusual Concentration of Radiopertechnetate in a Patient with Thyroid Cancer. *Jacques Lamoureux, M.D., Ph.D.* ... 714
Diagnosis of Barrett's Esophagus with Radioisotopes. *Felipe Gordon, M.D., José Ramírez-Degollado, M.D., Rodrigo Muñoz, M.D., Alfredo Cuaren, M.D., and Luis Landa, M.D.* ... 716
Clinical Evaluation of Preoperative Liver Scintigraphy in Gastric Carcinoma Patients. *Katsushi Watanabe, M.D., Kenjiro Kawahira, M.D., and Keiichi Matsuura, M.D.* ... 720
The Use of Multiple Radionuclide Imaging to Differentiate the Focal Intrahepatic Lesion. *Lawrence R. Muroff, M.D., and Philip M. Johnson, M.D.* ... 728
The Value of Tc^{99m}-Diphosphonate (HEDSPA) as a Skeletal Scanning Agent. *J. Papadimitriou, M.D., M. Vezeridis, M.D., M. Constantinidis, Ph.D., E. Chiotelis, Ph.D., C. Constandinidis, M.D., and C. Tountas, M.D.* ... 735
Pitfalls in Tc^{99m} Polyphosphate Skeletal Imaging. *James H. Thrall, M.D., Nassar Ghaed, M.D., George E. Geslien, M.D., Steven M. Pinsky, M.D., and Merrill C. Johnson, M.D.* ... 739
Influence of Fractionation on Acute and Late Reactions in Vocal Cord Carcinoma. *R. Kenneth Loeffler, M.D.* ... 748
The Results of Split-Course Radiation Therapy in Cancer of the Lung. *Hugh Scruggs, M.D., Anas El-Mahdi, M.D., Sc.D., Richard D. Marks, Jr., M.D., and William C. Constable, M.B., Ch.B., D.M.R.T.* ... 754
Dual Camera Studies of Pulmonary Function with Computer Processing of Data. *Merle K. Loken, M.D., Ph.D., Richard A. Ponto, B.S., Richard S. Kronenberg, M.D., Lawrence E. Williams, Ph.D., and Marvin E. Goldberg, M.D.* ... 761
Extended Mantle Radiotherapy in Hodgkin's Disease and Malignant Lymphoma. *J. E. Marks, M.D., E. M. Moran, M.D., M. L. Griem, M.D., and J. E. Ultmann, M.D.* ... 772
Angiography of the Liver and Spleen in Hodgkin's Disease. *K. Jonsson, M.D., and A. Lunderquist, M.D.* 789
Kaposi's Sarcoma: Experience with Ten Cases. *Steven G. Mann, M.D.* ... 793
Urographic Manifestations of Metastatic Melanoma. *Harvey M. Goldstein, M.D., Stephen Kaminsky, M.D., Sidney Wallace, M.D., and Douglas E. Johnson, M.D.* ... 801
The Increased Incidence of Carcinoma of the Colon following Ureterosigmoidostomy. *Randall S. Preissig, M.D., William F. Barry, Jr., M.D., and Richard G. Lester, M.D.* ... 806
Detection and Localization of an Occult Vesicoenteric Fistula. *Edward K. Prokop, M.D., E. Ulric Buddemeyer, Sc.D., H. William Strauss, M.D., and Henry N. Wagner, Jr., M.D.* ... 811
Inferior Vena Caval Extension of Renal Carcinoma: A Lost Cause?: Roentgenographic and Pathologic Findings in Surgical Patients. *David L. McCullough, M.D., and Lee B. Talner, M.D.* ... 819

Calcification in Uroepithelial Tumors of the Bladder: Report of 5 Cases and Survey of the Literature. *Stephen W. Miller, M.D., and Richard C. Pfister, M.D.* ... 827
Radiation Therapy and Pelvic Node Dissection in the Management of Cancer of the Prostate. *Basil S. Hilaris, M.D., Willet F. Whitmore, Jr., M.D., Mostafa A. Batata, M.D., and Harry Grabstald, M.D.* 832
Percutaneous Needle Localization of Clustered Mammary Microcalcifications Prior to Biopsy. *Barbara Threatt, M.D., Henry Appelman, M.D., Richard Dow, M.D., and Terry O'Rourke, M.D.* ... 839
A Device for Precision Needle Biopsy of the Breast at Mammography. *Anders Mühlow* ... 843
Analysis of 462 Breast Carcinomas. *John N. Wolfe, M.D.* ... 846
A New Biopsy Needle and Its Clinical Use. *L. H. Lee, M.D.* ... 854
Thermal Enhancement of the Radiosensitivity Using Cultured Normal and Neoplastic Cells. *J. H. Kim, M.D., Ph.D., S. H. Kim, M.S., and E. Hahn, Ph.D.* ... 860
Computer Generated Isodose Curves for High Energy X-ray Machines: Experimental Verification and Time-Saving Strategies. *Karen Lane, B.A., Peter Bloch, Ph.D., and Lawrence W. Davis, M.D.* ... 865
Design of X-ray Targets for High Energy Linear Accelerators in Radiotherapy. *E. B. Podgoršak, Ph.D., J. A. Rawlinson, M.Sc., M. I. Glavinović, B.Sc., and H. E. Johns, Ph.D.* ... 873
Three-Dimensional Imaging of Abdominal Organs with Ultrasound. *S. Nørby Rasmussen, M.D., S. Skogstad Nielsen, M.Sc., Royal J. Bartrum, Jr., M.D., Bent Stigsby, M.D., and Hans H. Holm, M.D.* ... 883
Officers ... 889
Editorial
Fifty-sixth Annual Meeting of the American Radium Society. *Luther W. Brady, M.D.* ... 890
National Research Council: Picker Foundation Awards, 1974–1975 ... 897
National Council on Radiation Protection and Measurements (NCRP); Report No. 41 ... 900
I.A.E.A. Catalogue of Radiation Dosimetry Data ... 901
News Items ... 902
Book Reviews ... 905
Books Received ... 906
Society Proceedings ... 907
Abstracts of Radiologic Literature ... 914
Subject Index to Volume 121 ... 924
Author Index to Volume 121 ... 933

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VOL. 121 MAY, 1974 NO. 1

TABLE OF CONTENTS

Review of Roentgenographic Bone Demineralization Studies of the Gemini Space Flights. GEORGE P. VOSE, M.S. 1

Cleidocranial Dysostosis: A Review of 40 New Cases. J. LUTHER JARVIS, M.D., AND THEODORE E. KEATS, M.D. 5

Condensing Osteitis of the Clavicle: A New Entity. ANNE C. BROWER, M.D., DONALD E. SWEET, M.D., AND THEODORE E. KEATS, M.D. 17

Diabetic Osteoarthropathy: Clinical and Roentgenographic Observations in 90 Cases. MELVIN E. CLOUSE, M.D., HERBERT F. GRAMM, M.D., MERLE LEGG, M.D., AND THOMAS FLOOD, M.D. 22

Similar Bone Manifestations of Scleroderma and Rheumatoid Arthritis. JACK G. RABINOWITZ, M.D., JACK TWERSKY, M.D., AND MARIA GUTTADAURIA, M.D. 35

Homocystinuria Due to Cystathionine Synthase Deficiency: Clinical-Roentgenologic Correlations. PAULA W. BRILL, M.D., HAROLD A. MITTY, M.D., AND GERALD E. GAULL, M.D. 45

Complications of Total Hip Replacement: A Roentgen Evaluation. MAURICE F. MULLINS, M.B., B.CH., RICHARD N. SUTTON, M.D., M.S.E.E., AND GWILYM S. LODWICK, M.D. 55

The Painful Charnley Total Hip Replacement. CAROL DOLINSKAS, M.D., ROBERT E. CAMPBELL, M.D., AND RICHARD H. ROTHMAN, M.D., PH.D. 61

"Clipping Injury" Fracture of the Epiphysis in the Adolescent Football Player: An Occult Lesion of the Knee. LEE F. ROGERS, M.D., STANLEY JONES, M.D., A. ROSS DAVIS, M.D., AND GERAL DIETZ, M.D. 69

Arthrography of the Lateral Meniscus. MURRAY K. DALINKA, M.D., JAMES F. LALLY, M.D., AND VIJAY K. GOHEL, M.D. 79

Synovial Recesses in Knee Arthrography. CHARLES E. MONTGOMERY, M.D. . 86

Arthrography in Rupture of the Suprapatellar Bursa with Pseudocyst Formation. ADAH M. DUNCAN, M.D. 89

Use of Xeroradiography in Cruciate Ligament Injuries. HARRY J. GRIFFITHS, M.D., AND CARL J. D'ORSI, M.D. 94

Lipohemarthrosis. PETER H. ARGER, M.D., PAUL E. OBERKIRCHER, M.D., AND WALLACE T. MILLER, M.D. 97

The Distal Anterior Femoral Metaphyseal Defect: An Anatomic Variant that May Simulate Disease. THEODORE E. KEATS, M.D. 101

(Continued on page iv)

FACULTY COLLEGE OF MED LIBRARY

Multifocal Osteoid Osteoma: A Case Report and Review of the Literature. Adam Greenspan, M.D., Alberto Elguezabel, M.D., and David Bryk, M.D. 103

Calcification in Cooper's Ligament. John R. Steinfeld, M.D., K. E. Schuit, Ph.D., and Theodore E. Keats, M.D. 107

Extravasation of Calcium Gluconate As a Cause of Soft Tissue Calcification in Infancy. Paul E. Berger, M.D., Kathleen P. Heidelberger, M.D., and Andrew K. Poznanski, M.D. 109

Primary Lymphangioma of the Calvarium. Mordecai Kopperman, M.D., and John E. Antoine, M.D. 118

Multiple Calvarial "Doughnut Lesions": A Case Report. Peter M. Royen, M.D., and M. B. Ozonoff, M.D. 121

The Maxillary Artery in Craniofacial Pathology. William E. Allen, III, M.D., E. Leon Kier, M.D., and Stephan L. G. Rothman, M.D. . . . 124

Os Supra Petrosum of Meckel. Guido Currarino, M.D., and Arthur Weinberg, M.D. 139

Patterns of Injury and Significance of Uncommon Fractures in the Battered Child Syndrome. Marvin S. Kogutt, M.D., Leonard E. Swischuk, M.D., and Charles J. Fagan, M.D. 143

Pharmacologic Aids in Angiography of the Upper Extremity. Harvey I. Wilner, M.D., Roger Kay, D.O., and Bradley A. Eisenbrey, M.D. 150

Gangliosidosis (GM_1): A Re-Evaluation of the Vertebral Deformity. Jack G. Rabinowitz, M.D., and Michael Sacher, M.D. 155

Size of Pelvic Bony Outlet in Renal Agenesis. Richard A. Fellows, M.D., Walter E. Berdon, M.D., and David H. Baker, M.D. 159

Neonatal Transplacental Rubella Syndrome: Its Effect on Normal Maturation of the Diaphysis. Joseph P. Whalen, M.D., Patricia Winchester, M.D., Lennart Krook, D.V.M., Ph.D., Neil O'Donohue, M.D., Renate Dische, M.D., and Eladio Nunez, Ph.D. 166

Bilateral Pseudotumors of the Pelvis in a Patient with Christmas Disease: With Notes on Localization by Radioactive Scanning and Ultrasonography. Charles D. Forbes, M.D., M.R.C.P., Brian Moule, M.B., B.Ch., D.M.R.D., F.F.R., Mary Grant, M.B., Ch.B., D.M.R.D., William R. Greig, M.D., Ph.D., F.R.C.P., and Colin R. M. Prentice, M.D., M.R.C.P. 173

Technetium 99m Labeled Diphosphonate Bone Scanning in Paget's Disease. Stephen W. Miller, M.D., Frank P. Castronovo, Jr., Ph.D., Henry P. Pendergrass, M.D., and Majic S. Potsaid, M.D. . . . 177

Comparison of Tc^{99m} Polyphosphate and F^{18} for Bone Imaging. D. A. Weber, Ph.D., J. W. Keyes, Jr., M.D., S. Landman, M.D., and G. A. Wilson, M.D. 184

Officers 191

Editorial

Continuing Medical Education: Implications Concerning Recertification, Re-examination and Relicensure. B. G. Brogdon, M.D. 192

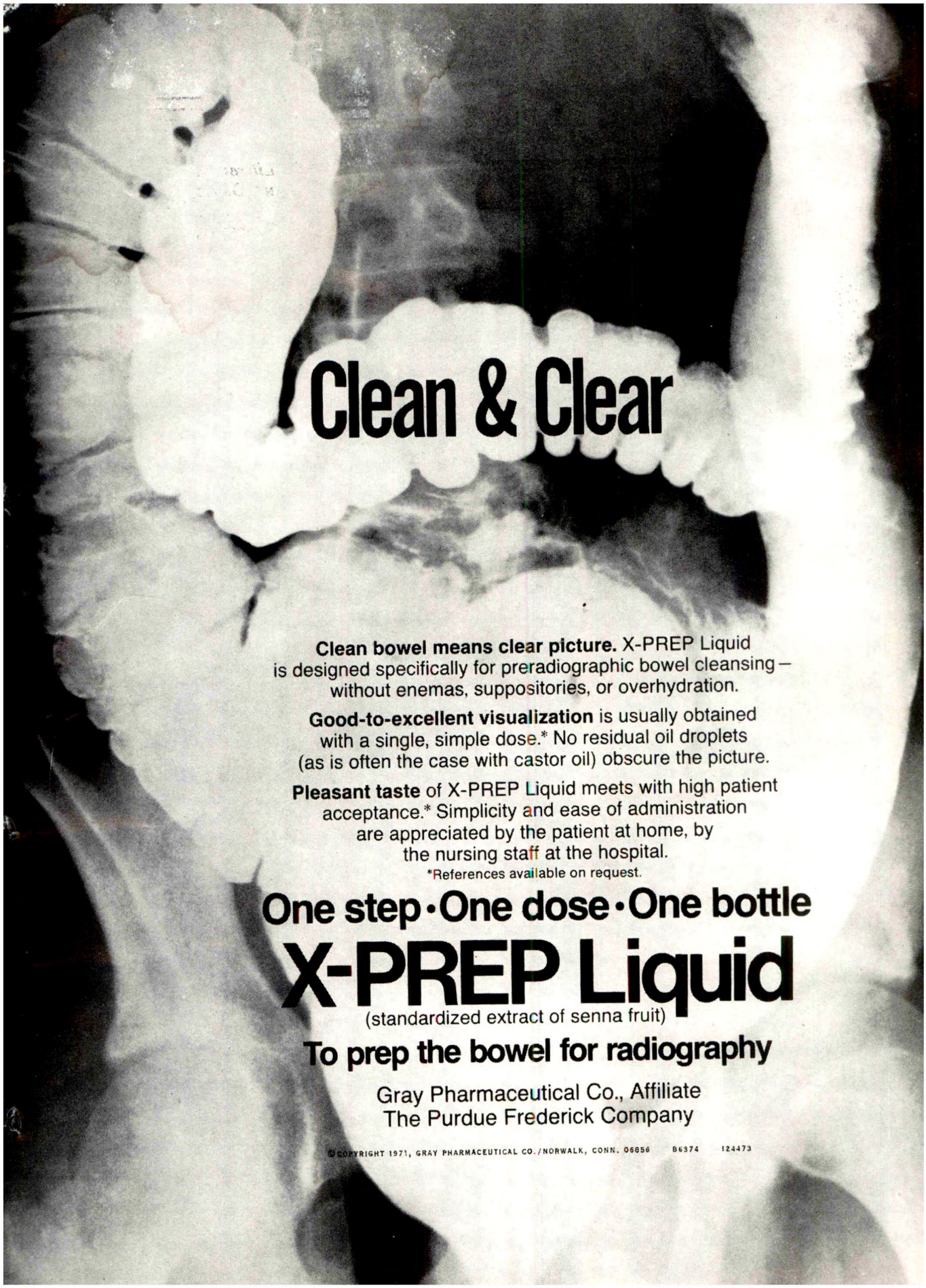
News Items 197

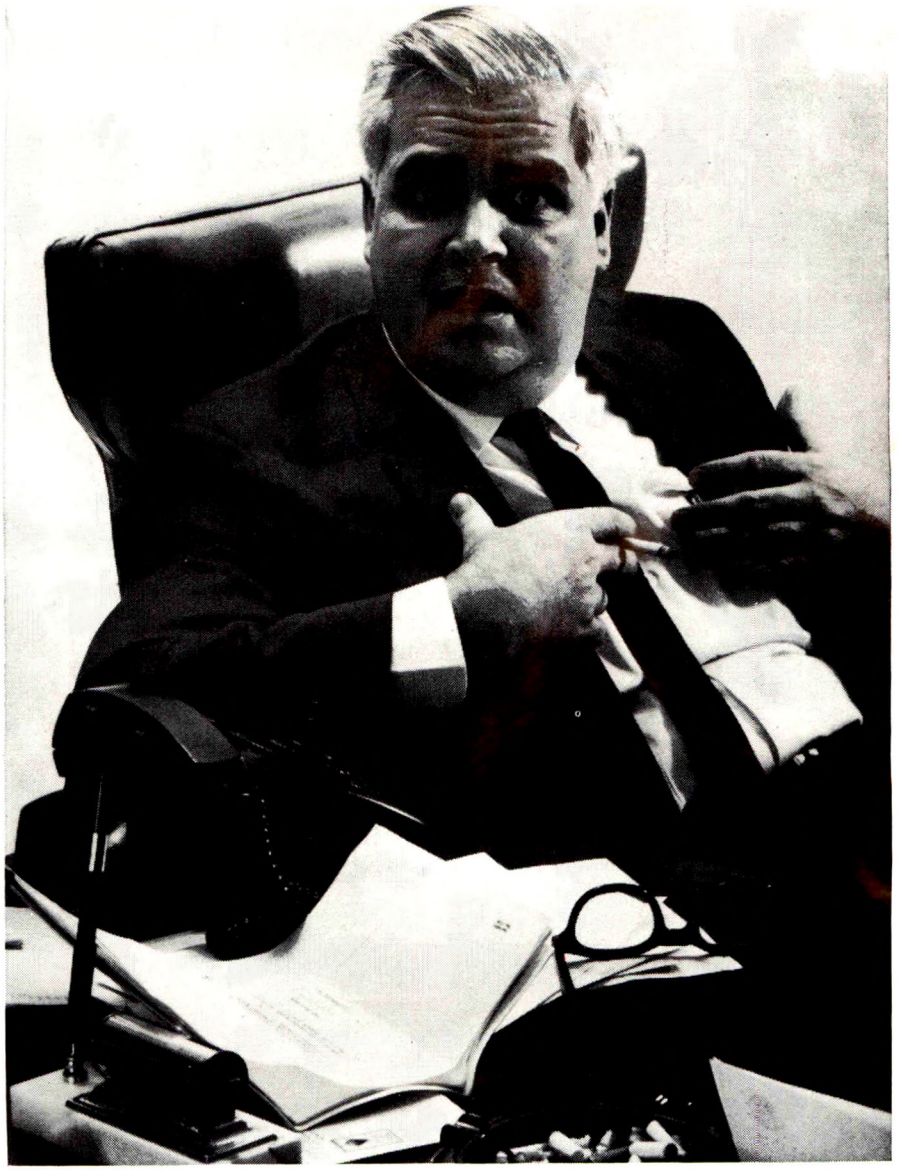
Book Reviews 200

Books Received 201

Society Proceedings 202

Abstracts of Radiologic Literature 209


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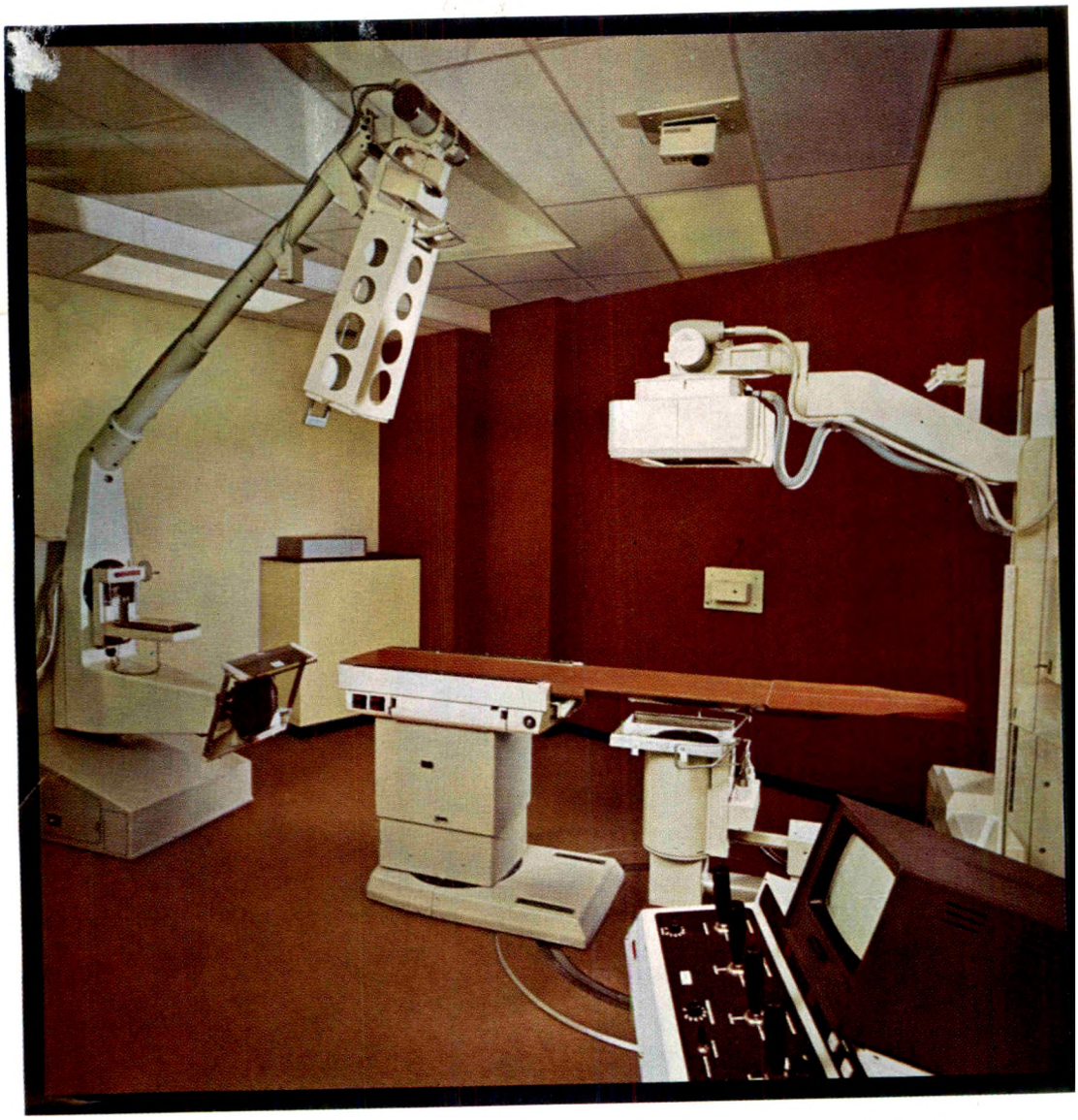
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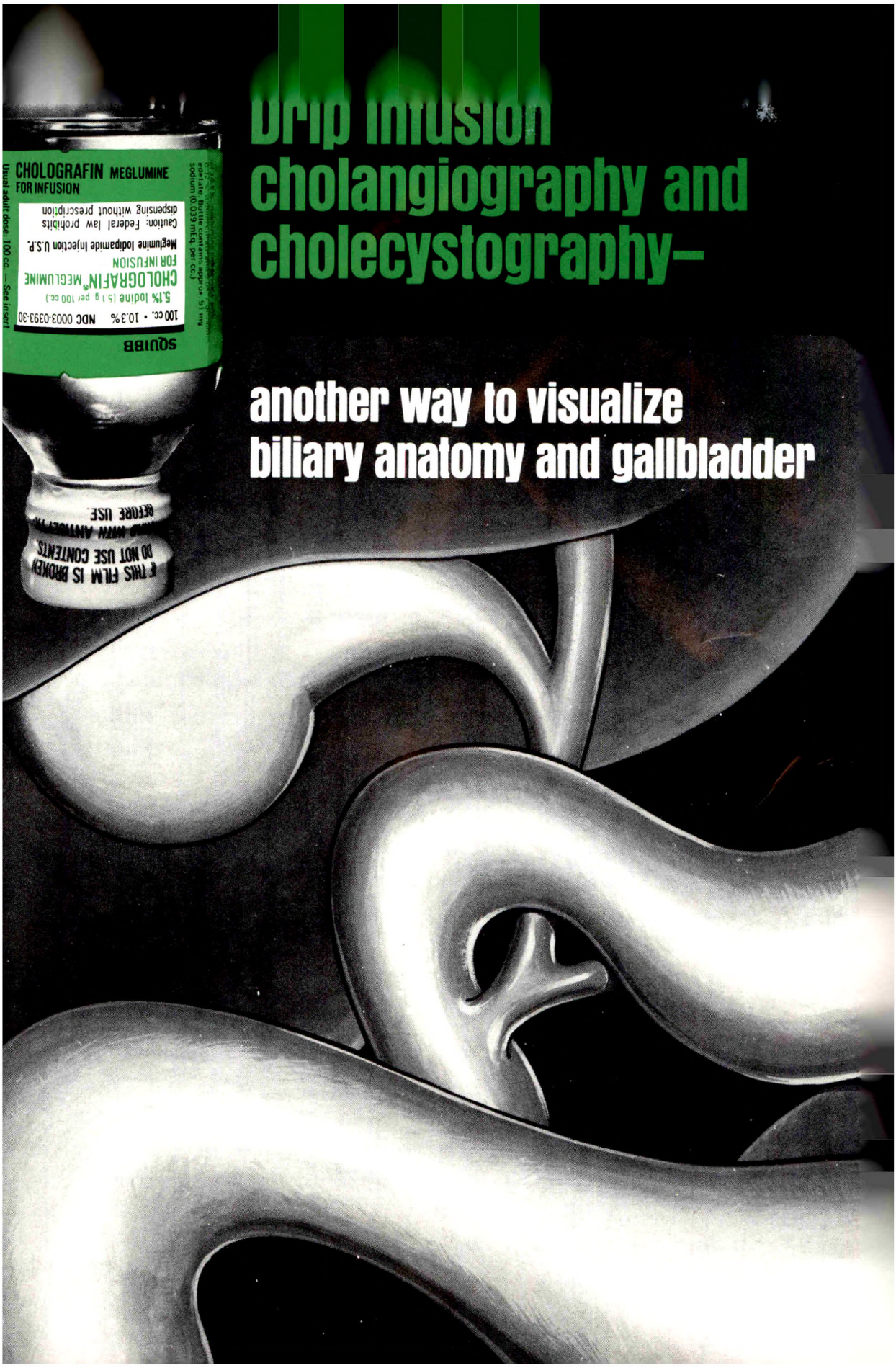
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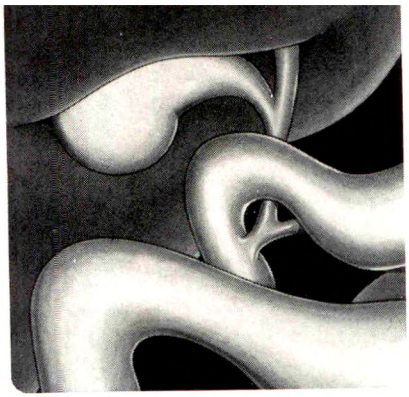
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Cholografin Meglumine (Meglumine Iodipamide Injection U.S.P.) is a sterile aqueous solution providing 52% meglumine iodipamide with 0.32% sodium citrate as a buffer and 0.04% disodium edetate as a sequestering agent. The bound iodine content is about 26% (5.2 g./20 ml.). *The solution contains approximately 0.91 mg. (0.039 mEq.) sodium per ml. (18.2 mg./20 ml.).*

CONTRAINDICATIONS: Contraindicated in patients hypersensitive to salts of iodipamide; in patients exhibiting sensitivity reactions to the test dose; in the presence of concomitant severe renal and liver function impairment.

WARNINGS: Intravenous or intra-arterial injections of contrast media have been shown to promote the phenomenon of sickling in persons homozygous for sickle cell disease. Perform thyroid function tests, if indicated, before administration of meglumine iodipamide since iodine-containing contrast media may alter results of such tests. A history of sensitivity to iodine *per se* or to other contrast agents is not an absolute contraindication to the use of meglumine iodipamide but calls for extreme caution in administration.

Usage in Pregnancy: The safety of meglumine iodipamide for use during pregnancy has not been established; therefore, it should be used in pregnant patients only when, in the judgment of the physician, its use is deemed essential to the welfare of the patient.

PRECAUTIONS: Diagnostic procedures involving use of contrast agents should be performed under the direction of personnel with prerequisite training and a thorough knowledge of the particular procedure. Appropriate facilities should be available for coping with situations which may arise as a result of the procedure and for emergency treatment of severe reactions to the contrast agent itself; competent personnel and emergency facilities should be available for at least 30 to 60 minutes after I.V. administration since severe delayed reactions have been known to occur. These severe life-threatening reactions suggest hypersensitivity to the contrast agent. A personal or family history of asthma or allergy or a history of a previous reaction to a contrast agent warrants special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 ml. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated. When the 52% meglumine iodipamide preparation is used, some clinicians feel it may be advisable to have a continuous I.V. infusion running prior to administration of the drug.

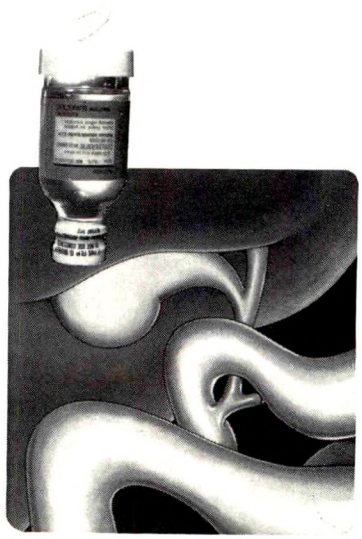
ADVERSE REACTIONS: Local reactions at the site of infusion or injection are not observed unless excessive amounts are extravasated during the infusion or injection. After too rapid administration, mild transient symptoms such as restlessness, sensations of warmth, sneezing, perspiration, salivation, flushing, pressure in the upper abdomen, dizziness, nausea, and vomiting may occur. These symptoms disappear when the infusion or injection has been completed. Hypersensitivity reactions may occur. In rare instances, despite the most careful sensitivity testing, anaphylactoid reactions may occur. Renal function tests may be altered and renal failure may occur.

For full information, consult package inserts.

HOW SUPPLIED: Cholografin Meglumine for Infusion (Meglumine Iodipamide Injection U.S.P.)—10.3%—is available in single-dose bottles of 100 ml. Cholografin Meglumine (Meglumine Iodipamide Injection U.S.P.)—52%—is available in single-dose vials and ampuls of 20 ml.

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SQUIBB HOSPITAL DIVISION



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The SAGITTAIRE is being used in some of the world's leading medical centres for sophisticated radiotherapy and high energy clinical research.

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Further information may be obtained by contacting our area representative.

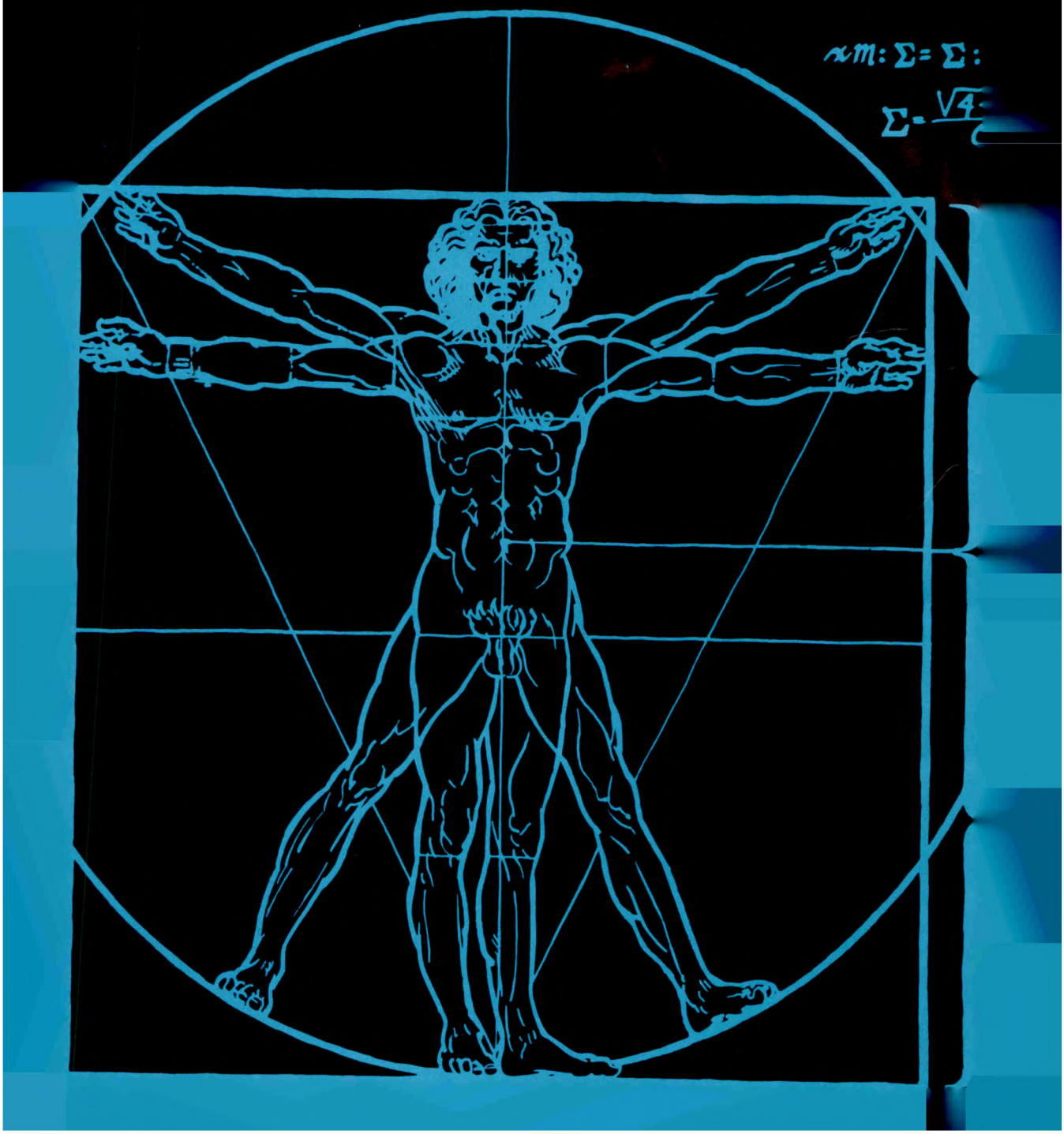
*The SAGITTAIRE, is the result of 18 years of experience in the field of particle accelerators by THOMSON CSF and CGR MeV.

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74.1 M

A new age of
adiology begins.



announcing 3M BRAND Trimax BRAND Rare Earth Imaging Systems

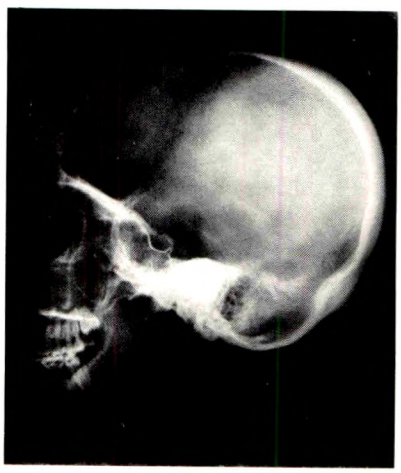
Publishing in RADIOLOGY in October, 1972,* researchers for Lockheed Research Institute described the properties of new and extremely efficient x-ray intensifying screens based on rare-earth technology. The potential for faster x-ray imaging systems was evident.

The missing link was a film so advanced it could match the capabilities of these rare-earth screens.

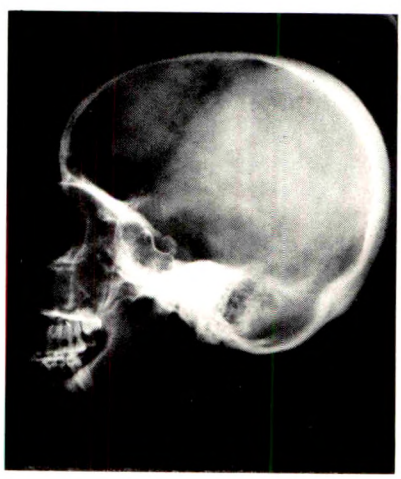
Today 3M technology has supplied that missing link in new TRIMAX Micro Dosage Imaging Systems.

What this means, very briefly, is a combination of film and screens up to *eight timer faster* than conventional medium-speed or detail-speed systems—with no equipment expenditures, no changes in chemistry, no changes in processing cycles, and no change in information.

Thus TRIMAX marks the beginning of very high speed, very high resolution radiography—the radiology of the future. Radiation levels are substantially reduced. Small focal work can now be done with relative ease and safety. Tubes will no longer be damaged by constant use at full capacity. Retakes required by patient movement will be virtually eliminated. Portables and low-powered equipment instantly become more efficient.



This x-ray was made at 70 Kvp-60 MaS with a conventional medium-speed imaging system.



This x-ray was made at 70 Kvp-7 ½ MaS with the new TRIMAX Micro Dosage Imaging System.

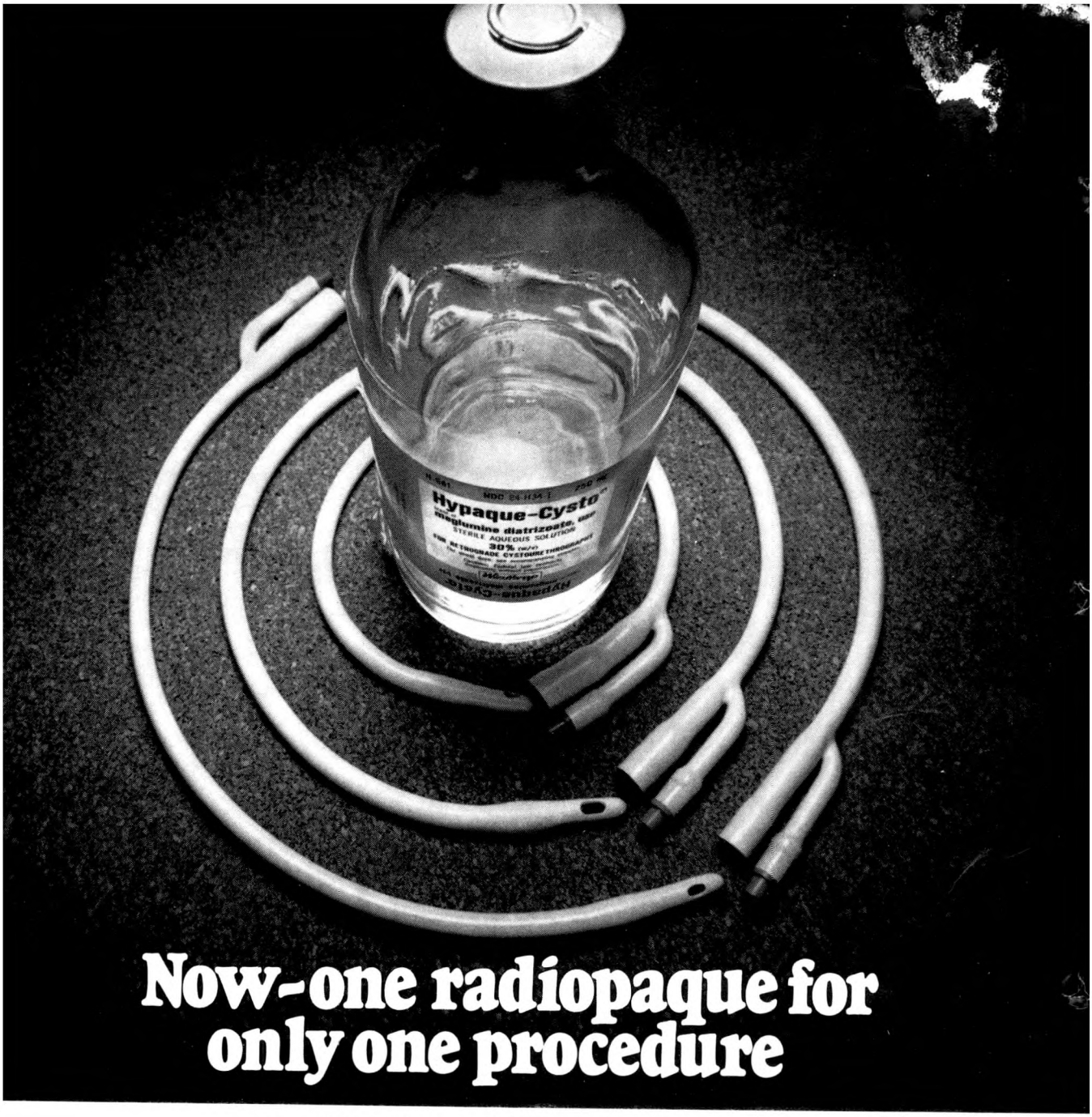
Thousands of radiologists and technologists throughout the country have expressed their interest in evaluating new 3M TRIMAX Micro Dosage Imaging Systems. Faced with this unprecedented demand for demonstrations, we can only ask your patience and forebearance.

Please believe we want to bring you and your patients the benefits of this "new age of radiology" as rapidly as possible.

*Radiology 105: 185-190, October, 1972. X-ray exposure reduction using Rare-Earth Oxysulfide Intensifying Screens; Robert A. Buchanan, PhD., Solon I. Finkelstein, M.D., and Kenneth A. Wickersheim, PhD.

PHOTOGRAPHIC
PRODUCTS DIVISION
3M Center.
St. Paul, Minnesota 55101

3M COMPANY



Now-one radiopaque for only one procedure

specifically designed for accurate well-tolerated retrograde cystourethrography

Specifically packaged. 250 ml. in calibrated 500 ml. bottle for dilution with sterile water or 5 per cent dextrose solution.

Complete in a single unit. For greater dilution capacity—calibrated bottle conveniently suited to 1 to 1 dilution. For flexible dosage range—concentration is easily adjusted over a wider range.

Well tolerated. No irritation or other undesirable effects have been reported following its administration in clinical investigations.*

*Data on file at Sterling-Winthrop Research Institute.

the specific for retrograde cystourethrography

New Hypaque-Cysto®

brand of

meglumine diatrizoate, USP

a logical addition to the expanding Hypaque® product line

New Hypaque-Cysto®

brand of
meglumine diatrizoate, USP

Sterile Aqueous Solution
For Retrograde Cystourethrography

Description: Hypaque-Cysto is a radiopaque medium which contains approximately 141 mg. organically-bound iodine per ml. It is a 30 per cent (weight/volume) sterile aqueous solution of the meglumine salt of 3,5-diacetamido-2,4,6-triiodobenzoic acid ($C_{18}H_{24}I_3N_3O_9$). The viscosity is 1.94 cps at 25° C. and 1.42 cps at 37° C. The pH is adjusted with hydrochloric acid or meglumine solution. Hypaque-Cysto is clear and colorless to pale yellow. It should be protected from strong light. The solution is relatively thermostable and may be autoclaved. Calcium disodium edetate 1:10,000 has been added as a sequestering stabilizing agent.

Action: Hypaque-Cysto is a diagnostic radiopaque medium which provides visualization of the lower urinary tract.

Indication: Hypaque-Cysto is indicated for retrograde cystourethrography.

Adverse Reactions: No irritation or other undesirable effects have been reported following the administration of Hypaque-Cysto in clinical investigations.

Dosage and Administration: After the bladder is emptied, Hypaque-Cysto is gently instilled without force, often beyond the first desire to micturate, but not beyond the point of urgency or mild discomfort. The volume required to fill the bladder to slightly less than capacity may vary from patient to patient.

Bladder capacity in normal adults is generally 200 to 300 ml., and rarely, up to 600 ml. Capacity at birth is 20 to 50 ml., and increases about 400 per cent in the first year. In children 3 to 5 years old, bladder capacity is 150 to 180 ml. In children older than 8 years, it is in the low adult range.

In disease, bladder capacity in adults may vary from 50 ml. in neurogenic "reflux" bladder to over 1000 ml. in autonomous bladder or chronic lower urinary tract obstruction.

Repeat examination may be required to detect reflux, or in function studies.

The concentration varies with technique and equipment used. Hypaque-Cysto may be diluted with sterile water or 5 per cent dextrose solution, as indicated in the following table.

To Make		Add	Final Solution Contains
Final conc.	Final volume	Sterile water or 5% dextrose solution	Iodine
30 %	250 ml.	—	141 mg./ml.
25 %	300 ml.	50 ml.	118 mg./ml.
21.4%	350 ml.	100 ml.	101 mg./ml.
20 %	375 ml.	125 ml.	94 mg./ml.
18.8%	400 ml.	150 ml.	88 mg./ml.
16.7%	450 ml.	200 ml.	78 mg./ml.
15 %	500 ml.	250 ml.	71 mg./ml.

How Supplied: Calibrated 500 ml. dilution bottles containing 250 ml. Hypaque-Cysto; rubber stoppered, with inner removable seal and screw neck.

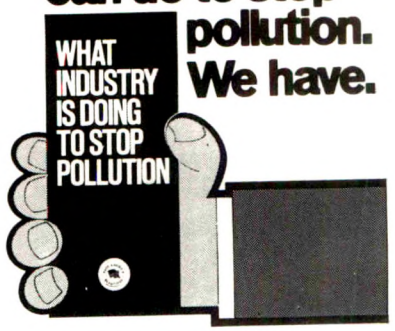


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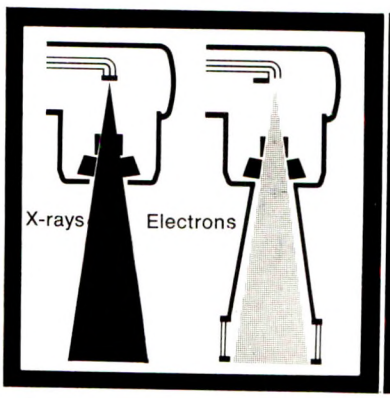
Philips Radiation Therapy Programme. Totally integrated.

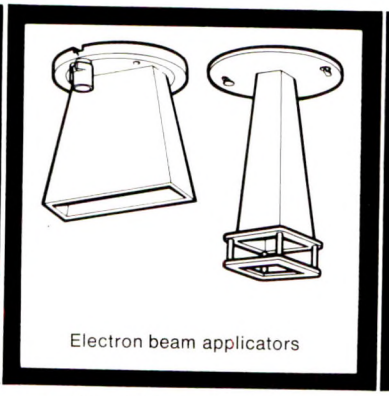
The many advantages offered by a complete and integrated system have long been recognised. That is why all components within the Philips programme are perfectly matched and have a common design philosophy. They are fully compatible: technically, visually and operationally.

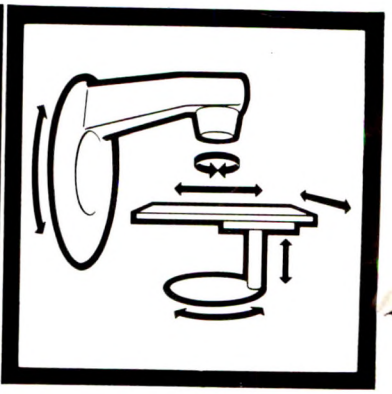
The Philips radiation therapy programme is unique. It offers the user versatile equipment in which he can have complete confidence. The result - greater clinical efficiency.

Linear Accelerator SL75-20

Within the Philips radiation therapy programme, the SL 75-20 is a total system within itself, with full provision for superficial and intermediate electron treatment and deep therapy with X-rays. Built-in select-and-confirm routines minimise errors in treatment; twin independent dosimetry channels prevent the pre-set maximum dose from being exceeded; touch guard and fail-safe interlocks ensure complete safety for patient and operator.




Electron beam applicators



Versatility

For electron therapy, the beam can be set in ten steps from 5 MeV to 20 MeV thus taking full advantage of the sharp depth dose cut-off. Specially designed electron applicators ensure maximum field flatness. For deep seated lesions, using arc or multiple field techniques, two alternative energy levels of 8 and 16 MeV with dose rates of more than 350 rads/min. at 100 cm provide short treatment times and optimum therapy with minimum skin reaction. The X-ray focal spot of 3 mm diameter together with the high precision diaphragm system provides a sharply defined beam.

Rigidity with flexibility

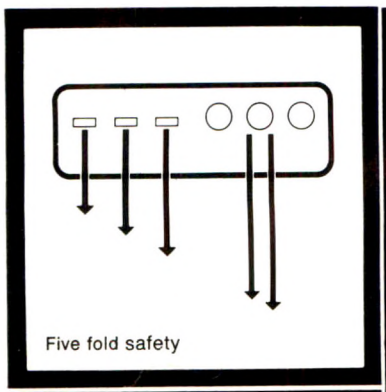
Advanced design and precision engineering have resulted in an extremely rigid structure and pin-point accuracy. The gantry rotation, treatment head and table movements are motorised with variable speed control. The table can be lowered to 46 cm for easy patient handling and, when the table top is removed, the ram can be completely retracted to permit other forms of patient support to be used.

Safety and efficiency

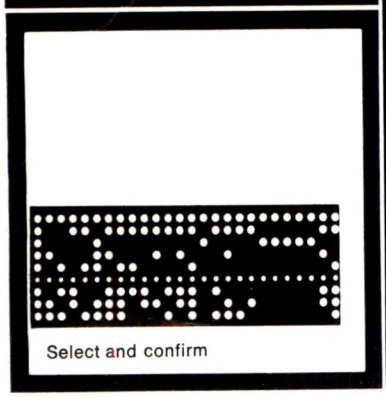
In all aspects the SL 75-20 is safe. Safe for the patients, safe for the operator. The patient is protected by five independent systems: a primary dosimetry channel, secondary dosimetry channel, back-up timer, high dose-rate safety cut-out and dose counter monitoring circuit. In addition, all movements of the head, gantry and table are controlled by fail-safe devices and interlocks. The standard control system incorporates selection and confirmation routines which minimise possibilities of erroneous treatment and reduce staff strain. Optional equipment is available for the automatic printout of treatment data and the automatic verification of the machine settings thereby assisting the setting up operation and ensuring fast, accurate record keeping.

PHILIPS

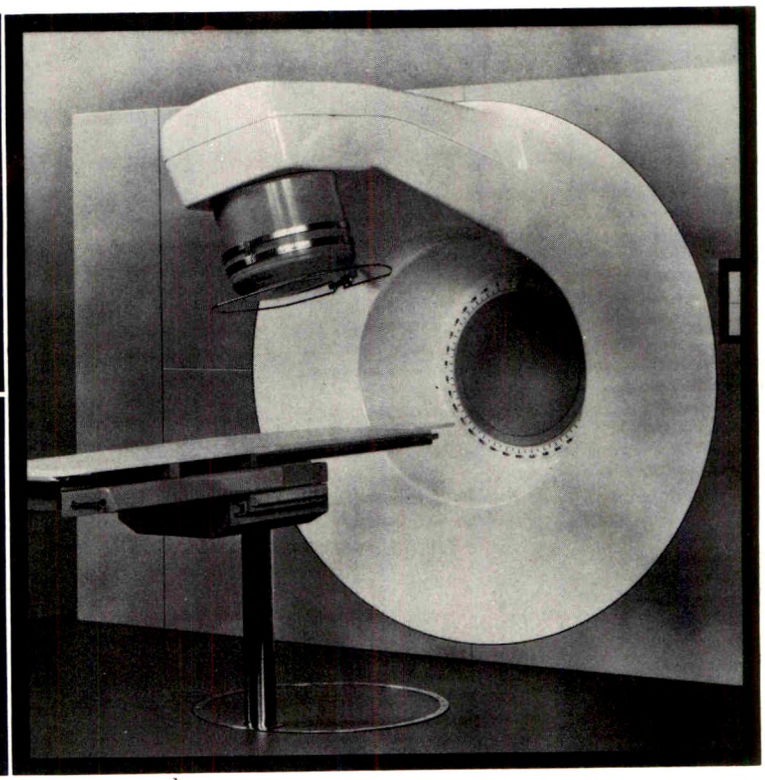
Philips in Radiation therapy
Safety Efficiency Reliability



Five fold safety



Select and confirm

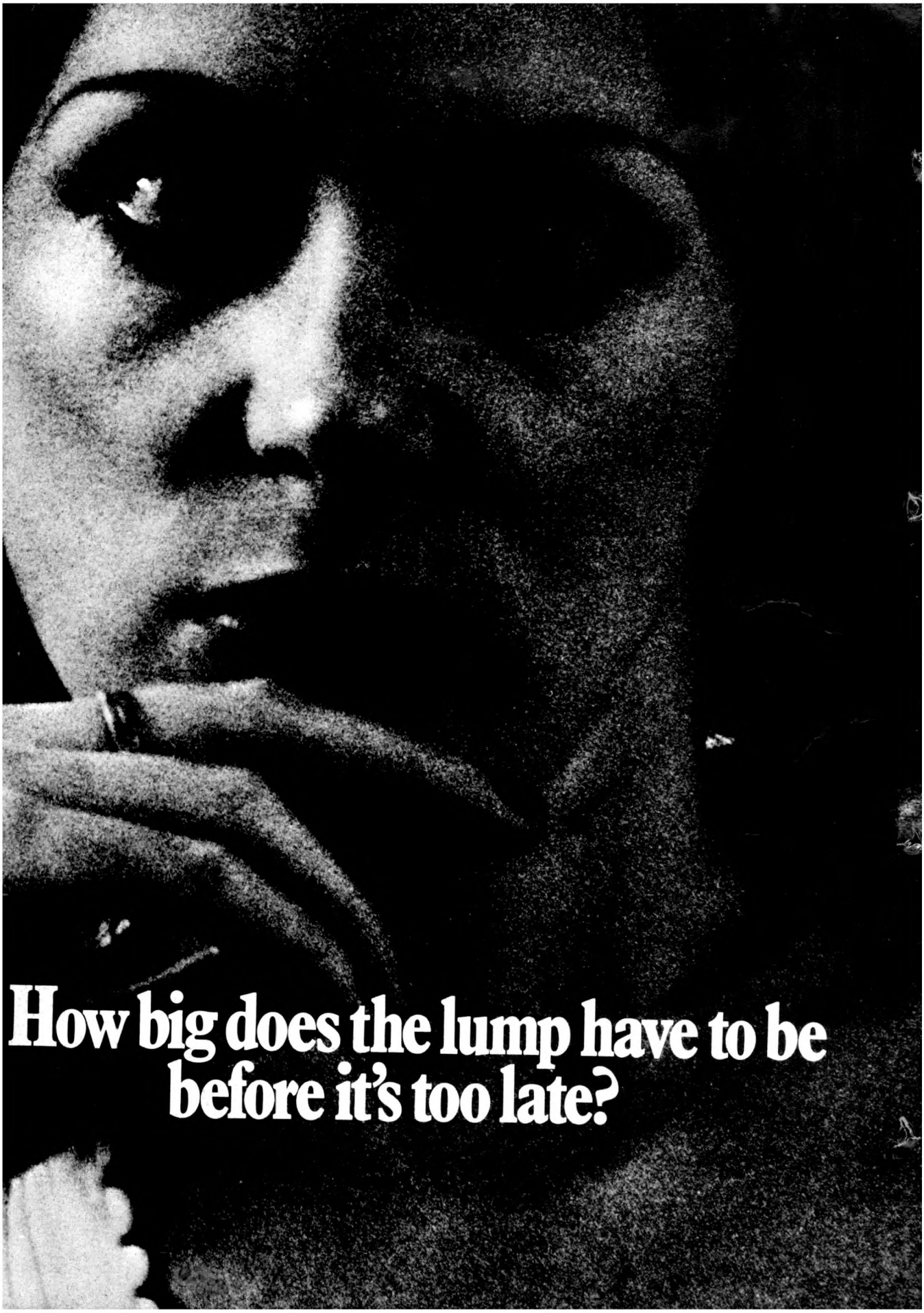


Reliability

The high degree of reliability achieved by the Philips Linear Accelerator SL 75-20 is obtained by attention to detail throughout design, development and manufacture. The result is a long trouble-free life with minimum maintenance.

For further information please contact your local organisation or R.G. Philips Medical Systems Division, Eindhoven, The Netherlands.

PHILIPS **Medical Systems**


How big does the lump have to be
before it's too late?

Recently, Profexray introduced a system that provides a new standard of excellence in the field of mammography.

We call it Gemini II.

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We feel strongly that effective mammography should be as important a part of today's cancer detecting examination as is the PAP test.

One question remains: How long should she be willing to wait?

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Contact your Profexray representative now for a comprehensive Gemini II presentation or write Profexray Marketing, 825 Nicholas Blvd., Elk Grove Village, Illinois 60007.

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Rapid R Type S, our new medium-speed film for 90-second processing, gives you something new . . . unique density characteristics that improve image detail. In simpler terms, our new film gives you radiographs that aren't too contrasty, aren't too flat, but are just right. In the same exposure and processing time you're already used to, Rapid R Type S gives you low-grain, crisp-looking films with a wide range of detail.

Rapid R Type S is just one of many new products we're working on because new products are a key part of our major expansion program. A program prompted by the fact that Ilford products are showing the greatest growth in the industry.



with new ideas for you Type S.

And before we're through, we plan to have all the capacity, inventory, people and new products to not only get well ahead of demand but to convince you too that Ilford should be your first choice for X-ray products.

Already, our new manufacturing plant in Brentwood is in operation. We've tripled our warehouse facilities in California. And we're doubling and fully automating our central warehouse. In addition, all warehouse facilities are being computer-linked to give us full flexibility to get material where it's needed *before* it's needed.

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If you don't use Ilford yet, keep an eye on us. On our full line of films for general radiography. On our new family of cine films. On our intensifying screens and chemicals. On our quality control monitor system. And most importantly, on the thing that puts them all together for you—The Ilford Man, an X-ray professional who knows your department's needs inside out.

Keep an eye on us because one day soon The Ilford Man will come calling. And when he does, he'll have a lot of new and exciting things to show you. He always does. Which explains why Ilford products are showing the greatest growth in the industry.

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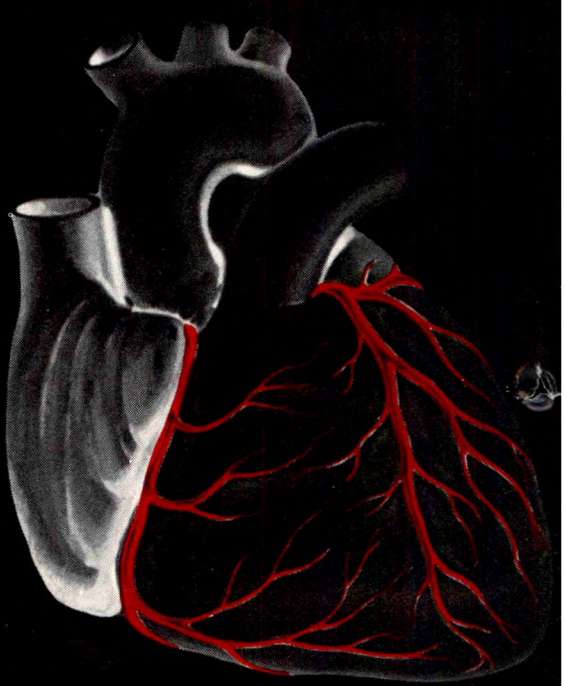
The Ilford Man is on the move.

In a single bottle...

enough contrast agent for selective coronary arteriography combined with left ventriculography







introducing
the new 200cc. single-dose bottle

RENOGRAFIN®-76 (MEGLUMINE DIATRIZOATE 66% AND SODIUM DIATRIZOATE 10% INJECTION)

- quantity sufficient for combined procedure—selective coronary arteriography with left ventriculography
- extra convenience—a single bottle to open
- cap may be removed or spiked

Also available: 20 cc. and 50 cc. single-dose vials for other procedures

Renografin-76 is indicated for excretion urography, aortography, pediatric angiocardiography, peripheral arteriography, selective coronary arteriography, and selective coronary arteriography combined with left ventriculography.

SQUIBB HOSPITAL DIVISION

For brief summary, see next page.

ew 200cc.
e-dose bottle

NOGRAFIN®-76

lumine Diatrizoate 66% and
ım Diatrizoate 10% Injection)

afin-76 is supplied as a sterile, aqueous
providing 66% meglumine diatrizoate
% sodium diatrizoate with 0.32% sodium
as a buffer and 0.04% disodium edetate
questering agent. The solution contains
mately 37% (370 mg./cc.) bound iodine
proximately 4.48 mg. (0.19 mEq.) sodium

ndications: In patients with a hypersen-
to salts of diatrizoic acid. Urography
ndicated in patients with anuria.

gs: A definite risk exists with the use of
t agents in excretion urography in pa-
vith multiple myeloma. There has been
with progressive uremia, renal failure
ath. This risk of the procedure in these
is not a contraindication; however,
dehydration in preparation for study is
ommended since it may predispose for
tation of myeloma protein in renal
. No therapy, including dialysis, has
successful in reversing this effect.
ia should be considered in persons over
re undertaking urographic procedures.

ises of known or suspected pheochro-
ma, if the physician feels that the pos-
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que materials should be administered
treme caution; however, an absolute
m of material should be injected, the
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rast media may promote sickling in
gous individuals when injected I.V. or
terially. Although a history of sensitivity
ie *per se* or to other contrast media is
absolute contraindication, administra-
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ases. Renografin-76 should be used in
nt patients only when the physician
its use essential to the welfare of the
since safe use during pregnancy has
n established. Perform thyroid function
rior to administration of Renografin-76
odine-containing contrast agents may
e test results.

h the inherent risks against necessity
forming *angiocardiography* in cyanotic
and patients with chronic pulmonary
sema. In *pediatric angiocardiography,* a
10 to 20 cc. may be particularly haz-
in infants weighing less than 7 kg.; this
probably significantly increased if these
have preexisting right heart "strain,"
eart failure, and effectively decreased
erated pulmonary vascular beds. Per-
rography with extreme caution in per-
vith severe concomitant hepatic and
isease. Perform *selective coronary ar-*
aphy only in selected patients and those
n expected benefits outweigh the pro-
risk.

tions: Diagnostic procedures involving
contrast agents should be performed
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aining and a thorough knowledge of the
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be available for coping with situations
may arise as a result of the procedure
r emergency treatment of severe re-
to the contrast agent itself; competent
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le for at least 30 to 60 minutes after I.V.
stration since delayed reactions have
nown to occur. These severe life-
ning reactions suggest hypersensitivity
ontrast agent. A personal or family his-
asthma or allergy or a history of a pre-

curately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 cc. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents; therefore, if known or suspected hepatic or biliary disorder exists, administration of Renografin-76 should be postponed following the ingestion of cholecystographic agents. Consider the functional ability of the kidneys before injecting Renografin-76. Use cautiously in severely debilitated patients and in those with marked hypertension. Bear in mind the possibility of thrombosis when using percutaneous techniques.

In *excretion urography,* adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be hazardous; this also applies to *pediatric angiocardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography,* hypotension or moderate decreases in blood pressure seem to occur frequently with intra-arterial (brachial) injections; this is transient and usually requires no treatment. Monitor blood pressure during the immediate 10 minutes after injection. It is recommended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and adequate facilities for immediate resuscitation and cardioversion.

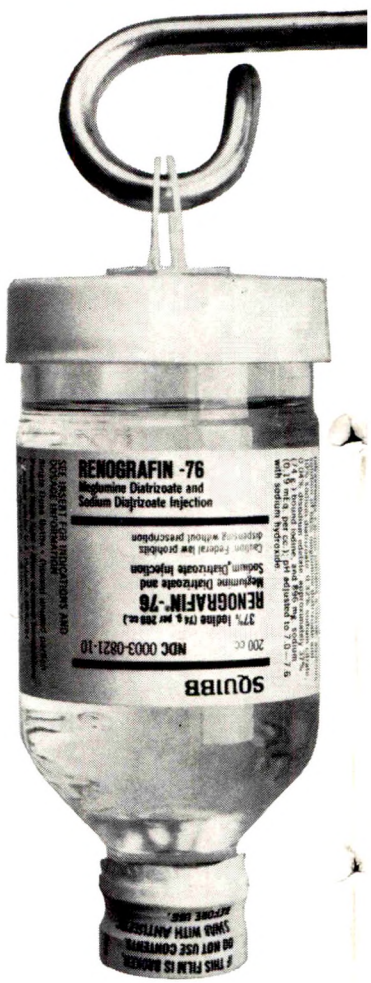
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Adverse reactions as a consequence of *excretion urography* include cardiac arrest, ventricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to generalized vasodilation. Risks of *aortography* procedures include injury to aorta and neighboring organs, pleural puncture, renal damage (including infarction and acute tubular necrosis with oliguria and anuria), accidental selective ease, retroperitoneal hemorrhage from translumbar approach, spinal cord injury and pathology associated with syndrome of transverse myelitis, generalized petechiae, and death following hypotension, arrhythmia, and anaphylactoid reactions. In *pediatric angiocardiography,* arrhythmia and death have occurred. During *peripheral arteriography,* hemorrhage from puncture site, thrombosis of the vessel, and brachial plexus palsy (following axillary artery injection) have occurred. During *selective coronary arteriography* and *selective coronary arteriography with left ventriculography,* transient ECG changes (most patients); transient arrhythmias (infrequent); ventricular fibrillation (from manipulation of catheter or administration of medium); hypotension; chest pain; myocardial infarction; transient elevation of creatinine phosphokinase (occurred in about 30% of patients tested); fatalities have been reported; hemorrhage, thrombosis, pseudoaneurysms at puncture site, dislogment of arteriosclerotic plaques, dissection of coronary vessels, and transient sinus arrest have occurred due to the procedure.

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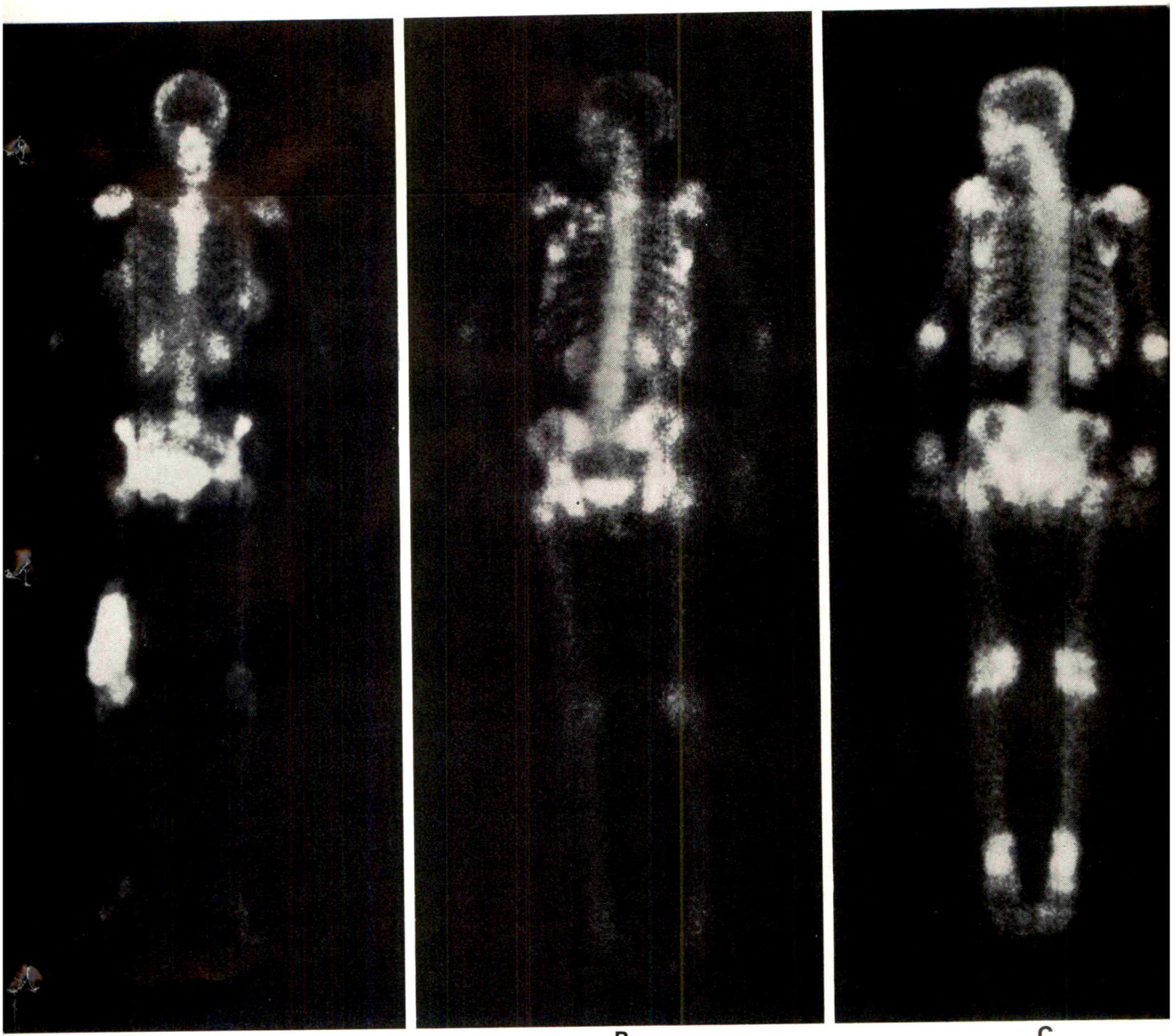
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B. 15 mCi ^{99m}Tc-OSTEOSCAN
Scanned 4 hr post injection
High Sensitivity Collimator
Speed: 32 cm/min, Length: 170 cm, Width: 60 cm
Posterior: 961,752 counts/1054.3 sec (17.6 min)
Comments: Cancer of breast. Polaroid image; posterior view taken with detector under table

C. 15 mCi ^{99m}Tc-OSTEOSCAN
Scanned 4 hr post injection
Low-Energy, All-Purpose Collimator
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Anterior: 927,833 counts/737.4 sec (12.3 min)
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OSTEOSCAN

(5.9 MG DISODIUM ETIDRONATE 0.16 MG STANNOUS CHLORIDE)

SKELETAL IMAGING AGENT

See following page for brief summary of package insert.

PROCTER & GAMBLE

OSTEOSCAN

(5.9 MG DISODIUM ETIDRONATE
0.16 MG STANNOUS CHLORIDE)
SKELETAL IMAGING AGENT



Brief summary of Package Insert. Before using, please consult the full Package Insert included in each kit.

DESCRIPTION

Each vial of OSTEOSCAN contains 5.9 mg disodium etidronate and 0.16 mg stannous chloride as active ingredients. Upon addition of ADDITIVE-FREE ^{99m}Tc-pertechnetate, these ingredients combine with ^{99m}Tc to form a stable soluble complex.

ACTIONS (CLINICAL PHARMACOLOGY)

When injected intravenously, ^{99m}Tc-labeled OSTEOSCAN has a specific affinity for areas of altered osteogenesis. Areas of bone which are undergoing neoplastic invasion often have an unusually high turnover rate which may be imaged with ^{99m}Tc-labeled OSTEOSCAN.

Three hours after intravenous injection of 1 ml ^{99m}Tc-labeled OSTEOSCAN, an estimated 40-50% of the injected dose has been taken up by the skeleton. At this time approximately 50% has been excreted in the urine and 6% remains in the blood. A small amount is retained by the soft tissue. The level of ^{99m}Tc-labeled OSTEOSCAN excreted in the feces is below the level detectable by routine laboratory techniques.

INDICATIONS

OSTEOSCAN is a skeletal imaging agent used to demonstrate areas of altered osteogenesis.

CONTRAINDICATIONS

None.

WARNINGS

This radiopharmaceutical should not be administered to patients who are pregnant or lactating unless the information to be gained outweighs the potential hazards.

Ideally, examinations using radiopharmaceuticals, especially those elective in nature, of a woman of childbearing capability should be performed during the first few (approximately 10) days following the onset of menses.

Radiopharmaceuticals should be used only by physicians who are qualified by specific training in the safe use and handling of radionuclides produced by nuclear reactor or particle accelerator and whose experience and training have been approved by the appropriate government agency authorized to license the use of radionuclides.

The ^{99m}Tc-generator should be tested routinely for molybdenum breakthrough and aluminum. If either is detected, the eluate should not be used.

PRECAUTIONS

Both prior to and following ^{99m}Tc-labeled OSTEOSCAN administration, patients should be encouraged to drink fluids. Patients should void as often as possible after the ^{99m}Tc-labeled OSTEOSCAN injection to minimize background interference from accumulation in the bladder and unnecessary exposure to radiation.

As in the use of any other radioactive material, care should be taken to insure minimum radiation exposure to the patient, consistent with proper patient management, and to insure minimum radiation exposure to occupational workers.

ADVERSE REACTIONS

None.

DOSAGE AND ADMINISTRATION

The recommended adult dose of ^{99m}Tc-labeled OSTEOSCAN is 1 ml with a total activity range of 10-15 mCi. ^{99m}Tc-labeled OSTEOSCAN should be given intravenously by slow injection over a period of 30 seconds within three (3) hours after its preparation. Optimum scanning time is 3-4 hours postinjection.

The patient dose should be measured by a suitable radioactivity calibration system immediately prior to administration.

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RADIUM THERAPY AND NUCLEAR MEDICINE

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REVIEW OF ROENTGENOGRAPHIC BONE DEMINERALIZATION STUDIES OF THE GEMINI SPACE FLIGHTS*

By GEORGE P. VOSE, M.S.
DENTON, TEXAS

THIS is a brief report compiled from some notes by the writer who participated in bone density studies in 3 of the Gemini space flights, the results of which were published a number of years ago in this Journal.[2]

The report involves computations which were not presented previously as well as some re-evaluations of old data in view of recent improvements in technology. It is felt that this information should be reported in order to clarify questions raised by some of the original data which reported unexplainably large losses in skeletal calcium during space flight.

BACKGROUND

In 1964 and 1965 an experiment designed to measure the effect of prolonged weightlessness on the skeleton was conducted in which pre- and post-flight roentgenograms were made of the Gemini 4, 5, and 7 astronauts for evaluation of possible bone mass changes using the Texas Woman's University system of bone densitometry. Following these investigations it was reported that surprisingly large mineral losses from the os calcis occurred in the Gemini 4 and 5 astronauts, while only moderate mineral changes occurred during the Gemini 7 orbital mission which were explainable on the basis of current knowledge of calcium mobility.[1,2]

Although the 14 day Gemini 7 flight was the longest duration in time undertaken in the Gemini series, the reported mineral changes were considerably less than those reported following the 4 day Gemini 4 flight and the 8 day Gemini 5 flight. In the Gemini 4 flight, density losses in the os calcis (in terms of aluminum wedge mass equivalency) were reported to be 7.8 per cent for the command pilot and 10.3 per cent for the pilot, and the reported losses in the Gemini 5 astronauts were 15.1 per cent for the command pilot and 8.9 per cent for the pilot. These losses are markedly more pronounced than the minimal 2.9 and 2.8 per cent losses in the astronauts of the 14 day Gemini 7 flight. In fact, the reported x-ray density changes in the earlier Gemini 4 and 5 flights, if wholly attributed to losses of

* From the Bone Metabolism Laboratory, Texas Woman's University, Denton, Texas.

bone mineral during the relatively short periods of time, are of such magnitude that they cannot be explained on the basis of current knowledge of bone physiology.

THEORETICAL

It should be noted that the per cent change in "x-ray aluminum equivalency" does not imply an equal change in bone mineral mass. We have determined that a decrease of 4 per cent in x-ray aluminum equivalency from the normal adult os calcis corresponds to a decrease of only 1 per cent of actual bone mineral.[3] Therefore, a loss of 15 per cent in x-ray aluminum equivalency in an 8 day period, such as that observed in the command pilot on Gemini 5, would indicate an actual mineral loss of 3.8 per cent. In the average adult male os calcis (containing 3,300 mg. of calcium) this would correspond to a loss of approximately 48 mg. of elemental calcium or a loss of about 6 mg. daily from that bone (bone mineral is approximately 38 per cent calcium). Extrapolating this to the skeleton as a whole (assuming that bone turnover rate is equal in all parts of the skeleton, and that the os calcis contains 1/500th of the total skeletal calcium), the massive daily loss of 3,000 mg. of calcium would be required.

Trabecular bone characteristically has a higher turnover rate than dense cortical bone which accounts for possibly 80 per cent of the skeletal mass. Considering only the 20 per cent of the skeleton which is composed of trabecular bone, a loss of 600 mg. of calcium a day would still be required to result in a bone mass loss of 15 per cent. It is improbable that such a severe negative calcium balance occurred during any space flight to date.

On the other hand, the losses on the order of −2.9 per cent by the Gemini 7 astronauts in 14 days represent losses of only 0.6 mg. of calcium a day from the os calcis which, on the basis of total skeletal trabecular bone, would represent a daily calcium loss of 65 mg. which may not be excessive under such unique environmental conditions. However, because the figures mentioned on the 2 earlier flights (Gemini 4 and 5) appear to be abnormally high, a search has been made for any causative factors which might be erroneously interpreted as bone mass losses of the reported magnitude.

EXPERIMENTAL

Of the 3 Gemini flights incorporating bone density experiments (Gemini 4, 5, and 7), the writer made roentgenograms immediately after astronaut recovery on the latter 2 flights. Different x-ray units were used prior to launch, on the recovery ship, and on follow-up tests. In all instances the KV. output was adjusted to produce a tabletop beam quality equal to that of the x-ray unit used at the L.B.J. Space Center in Houston—166 mr with 2 mm. added aluminum filtration.

Regardless of such careful attempts to assure a constant quality of x-radiation at all 3 examining sites, the only absolute method for assuring consistent reproducibility of bone density at each site is the precision of comparison of the living bone with a standard bone phantom made at the same time on all x-ray units. Accordingly, this procedure of control was adopted on the Gemini 5 and 7 roentgenograms.

The bone simulating phantom prepared by the writer for this test was a paraffin-embedded human os calcis sculptured to the dimensions of a foot. It is obvious that with sufficient measuring precision the roentgenographic density readings of this bone-simulating phantom would be the same at all 3 sites. However, densitometric evaluation of the phantom made following the first published reports of Gemini 5[1,2] indicated that the os calcis value obtained with the x-ray unit aboard the aircraft carrier U.S.S. Lake Champlain was 7 per cent lower than that obtained at the Kennedy Space Center. In addition, it was then determined that the calibration wedge used on the ship yielded a value 4 per cent lower than that of the auxiliary wedge used at the Kennedy Space Center prior to launch.

It is concluded from these determinations that, of the 7 per cent total error in x-ray measurements made aboard the aircraft carrier, 4 per cent was attributable to variations in intrinsic wedge densities or composition, and 3 per cent was due to unknown but consistent causes—perhaps dissimilar wave forms between the 2 x-ray units.

Knowing that such a 7 per cent error occurred, it became evident that the post-flight values obtained on the aircraft carrier in Gemini 5 were also 7 per cent too low. After correcting for this 7 per cent error the −15 per cent aluminum equivalency loss originally reported in the os calcis of the command pilot is reduced to −9.2 per cent, and the −8.2 per cent loss observed in the pilot is reduced to only −2.5 per cent. It is probable that the loss of −2.5 per cent in aluminum equivalency in the pilot is not a significant change because the over-all technique error is of approximately the same magnitude. The 9.2 per cent loss in the command pilot might be significant since it is well outside the experimental error of the technique; yet, it would correspond with a total daily calcium loss of 350 milligrams which is not physiologically impossible.

Although the calibration bone phantom was not roentgenographed concurrently with body roentgenograms on the Gemini 4 flight, tests made of the appropriate wedges on the same x-ray unit at a later date revealed that the same magnitude of error occurred on the unit aboard the U.S.S. Wasp as on the U.S.S. Lake Champlain. When the same 7 per cent correction factor is applied to the Gemini 4 data, the reported density losses in the os calcis of 7.8 and 10.3 per cent were reduced to 2.9 and 3.8 per cent in the command pilot and pilot, respectively.

Only on Gemini 7 was the same x-ray calibration wedge used throughout the study, and this was flown from the Kennedy Space Center to the aircraft carrier U.S.S. Wasp while the spacecraft was in orbit, and was also used in the initial and follow-up tests made at the L.B.J. Space Center in Houston. As mentioned previously, the changes in x-ray aluminum equivalency during this flight were on the order of only 2.5 per cent which strongly implies that much of the previously reported more marked losses in Gemini 4 and Gemini 5 are attributable to the use of different wedges pre- and post-flight.

Table 1 shows the values reported in 1967 representing per cent change in x-ray aluminum equivalency and the approximate corresponding calcium loss and the revised values.

TABLE I

PER CENT CHANGES IN X-RAY ALUMINUM EQUIVALENCY OF THE OS CALCIS FOR THE COMMAND PILOT AND PILOT AS ORIGINALLY REPORTED VS. THE CORRECTED VALUES*

	Originally Reported Values (1967)		Corrected Values	
	Command pilot	Pilot	Command pilot	Pilot
Gemini 4 (4 days)	−7.8% (−540)	−10.3% (−800)	−2.9% (−230)	−3.8% (−300)
Gemini 5 (8 days)	−15.1% (−600)	−8.9% (−350)	−9.2% (−350)	−2.5% (−100)
Gemini 7 (14 days)	−2.9% (−65)	−2.8% (−63)	—	—

* Values shown in parentheses are the approximate daily calcium losses in milligrams which would be necessary to correspond with the observed aluminum equivalency change.

SUMMARY

Several factors may have been conducive to misinterpretation of bone mineral changes in some of the Gemini astronauts which were reported previously by this laboratory. Evaluations of roentgenograms of a standard bone phantom made at the same times and on the same x-ray units indicate that the reported mass losses among the Gemini 4 and 5 astronauts were 7 per cent too high. This error is attributed to a 4 per cent inherent absorption difference between the 2 calibration wedges used prior to launch and after recovery, and 3 per cent of the error is attributable to variations in x-ray beam qualities among the 3 x-ray units used in the experiment.

The corrected density losses during the Gemini 4 flight are 2.9 and 3.8 per cent for the command pilot and pilot, respectively, instead of the 7.8 and 10.3 per cent values originally reported. Similarly, the corrected density losses during Gemini 5 were 9.2 and 2.5 per cent instead of the originally reported losses of 15.1 and 8.9 per cent for the command pilot and pilot, respectively.

Computed calcium losses necessary to yield the wedge equivalency changes originally reported in Gemini 4 and Gemini 5 would have had to be considerably greater than 600 mg./day, while in the Gemini 7 flight in which x-ray exposure conditions were more accurately controlled, a mean daily loss of skeletal calcium of 65 mg. would be required which is explainable under current knowledge of calcium mobility.

The modified data indicate that the observed changes in bone density pre- and post-flight were so close to the over-all experimental error that it is questionable whether any changes occurred at all. However, skeletal calcium losses which would concur with x-ray density changes of the observed magnitudes (63 to 350 mg. of calcium per day) would not be physiologically impossible.

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The Texas Woman's University
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TWV Station
Denton, Texas 76204

REFERENCES

1. Mack, P. B., Vose, G. P., Vogt, F. B., and LaChance, P. A. Experiment M-6, bone demineralization. In: Gemini Midprogram Conference. NASA SP-121, U. S. Government Printing Office 6606089, Washington, D. C., 1966, pp. 407–416.
2. Mack, P. B., LaChance, P. A., Vose, G. P., and Vogt, F. B. Bone demineralization of foot and hand of Gemini-Titan IV, V, and VII astronauts during orbital flight. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 503–511.
3. Vose, G. P. Estimation of changes in bone calcium content by radiographic densitometry. *Radiology*, 1969, *93*, 841–844.

CLEIDOCRANIAL DYSOSTOSIS*

A REVIEW OF 40 NEW CASES

By J. LUTHER JARVIS, M.D., *and* THEODORE E. KEATS, M.D.†
GASTONIA, NORTH CAROLINA AND CHARLOTTESVILLE, VIRGINIA

SKELETAL abnormalities reported previously in cleidocranial dysostosis involve virtually the entire osseous system. These observations, however, have been derived, for the most part, from small series of cases, due to the relative rarity of the condition. The material presented here is an assessment of 40 affected individuals from 8 unrelated family groups living in the Piedmont section of North and South Carolina, thus permitting an in-depth evaluation of the disease.

The purpose of this paper is: to record the pedigree of a large family, serving as a microcosm of cleidocranial dysostosis; to evaluate the known skeletal aberrations; and to present new features of the disorder. These include: skull changes considered specific for the condition; evidence of right clavicular selectivity; high incidence of spondylolysis of the lumbar vertebrae; characteristic lateral notching of the capital femoral epiphysis; and the danger of cephalopelvic disproportion, necessitating cesarean section.

MATERIAL

Forty white persons—18 males and 22 females, ranging in age from 14 months to 83 years— had roentgenographic examinations. In 1 family group, 22 members from 3 successive generations were examined; another kinship had 8 members from 5 of 6 generations known to have the syndrome; and 4 families had 2 members each—a parent and 1 child. Two boys had roentgen examinations but incomplete genealogic evaluation.

Twenty-seven patients had complete roentgenographic examinations of the following structures: skull, chest, thoracic spine, lumbar spine, pelvis, hands and knees. An additional 13 patients had some of these studies. All 40 individuals had roentgenograms of the clavicles taken.

GENETICS

The genealogy of the family groups in this study emphasizes the hereditary transmission of the syndrome by revealing 3 unusual kinships: (1) the passage of the disease through 6 successive generations of a kindred, apparently representing the longest familial linkage so far recorded; (2) the spontaneous mutational appearance of the disorder in a member of another family, present in her—but not in her 16 siblings—and transmitted to her daughter, suggesting that the mother represents the first phenotypic expression in this family of the dominant mutation causing cleidocranial dysostosis; and (3) the occurrence of mendelian dominance of high penetrance in 42 affected individuals from a 218 member kinship.

The pedigree of this large family is shown in Figure 1. The woman in the first generation died during the birth of her only child, and her every known descendant has been traced. At least 42 persons have cleidocranial dysostosis; almost surely other members will be recognized later, particularly in the sixth generation. Of these 42 affected individuals, 21 are males and 21 are females with 1 of the parents and roughly one-half of the brothers, sisters and children showing the trait. The disorder has passed equally from the mother or father to son and/or daughter, thereby fulfilling the requirements for mendelian dominance.

* From the Department of Radiology, Gaston Memorial Hospital, Gastonia, North Carolina.
† Professor and Chairman, Department of Radiology, University of Virginia, School of Medicine, Charlottesville, Virginia.

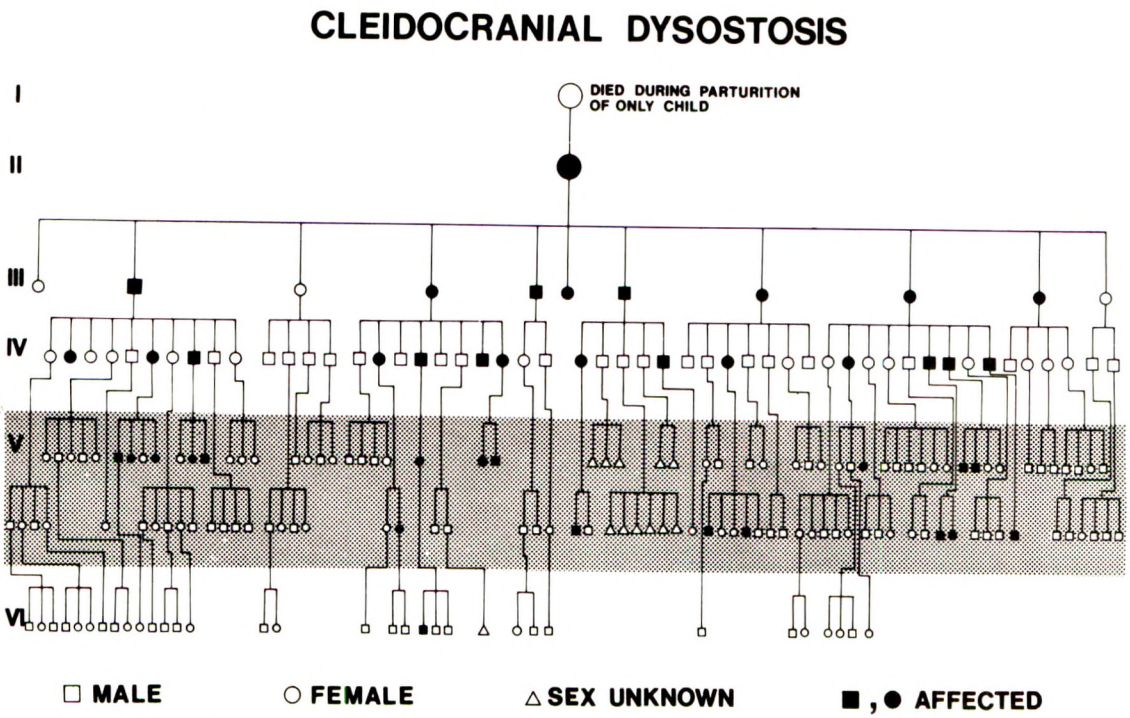


FIG. 1. Genealogy. The transmission demonstrates an autosomal dominant mode of inheritance in 42 affected individuals from a 218 member kinship.

ROENTGEN FINDINGS

SKULL

Familiar components of cleidocranial dysostosis in the skull (Table 1) include brachycephaly with wide interparietal diameter, hypertelorism, open metopic fontanelle and suture (Fig. 2, *A* and *B*), numerous wormian bones (Fig. 3, *A* and *B*) and delayed suture closure. In addition, the marked similarity of roentgen findings suggests a characteristic configuration in adults involving segmental calvarial thickening in the supra-orbital portion of the frontal bone, in the squama of the temporal bones and in the occipital bone above the inion, producing overlying eminences. Dysplastic changes occur in the basi-occiput as well, evidenced in part by consistent deformity of the foramen magnum.

The changes in the frontal bone consist of sclerosis of the diploic space with thickening of both tables of the calvarium. Marked sclerosis and thickening of the squamosal portion of the temporal bones, normally the thinnest part of the lateral wall of the cranial vault, produce a bony ledge at the squamoparietal suture, this shelf measuring nearly 1 cm. at times (Fig. 4, *A–C*). Also, the squama changes from its vertical orientation into somewhat of an oblique position.

Calvarial thickening also involves the squamosal portion of the occipital bone above the level of the external occipital protuberance, which is replaced by a large flat exostosis, the superior margin of which merges with the straightened planum occipitale. The inferior margin ends as a ledge which juts beyond the margin of the flattened basi-occiput for almost 1 cm., giving a "kyphotic base"[23] or a squared appearance to the occiput (Fig. 5).

Dental dysplasia occurred in almost all patients in this study (Table 1), manifested mainly by delayed eruption or failure of eruption of permanent dentition and by supernumerary teeth. Actually most of the permanent teeth in cleidocranial dysostosis

TABLE I

SKULL CHANGES IN CLEIDOCRANIAL DYSOSTOSIS

Ref. No.	Skull	Roentgen Studies	Yr. 2–11	Yr. 12–25	Yr. 26–	Total	Comment
9	Unossified metopic fontanelle	33	4	1	6	11	3 children under 3 years not roentgenographed
9	Unossified sutures						
	Metopic	33	3	5	5		
	Sagittal				3		
	Other			1	5	22	
9	Wormian bones	33	5	7	14	26	Extremely marked in 7
9	Hypoplastic sinuses	33	2	7	14	23	
15	Foramen magnum deformity	22	3	8	11	22	
9	Nasal bone calcification	32					
	Unossified		1		1	2	
	Less than 10 mm.		3	4	8	15	
	10–20 mm.		1	4	8	13	
	Over 20 mm.				2	2	
9	Interorbital distance	31					Hypertelorism in 27
20	Less than 30 mm.		1	2	3	6	=32 mm. average
	30 mm. and over		3	6	16	25	
	(Norm = 26 mm. ± 2 mm.)						
9	Dentition	40					Late or non-eruption of
12	Embedded teeth		2	10	17	29	permanent teeth; super-
14	Abnormal eruption by history		2		3	5	numerary teeth, partic-
	Primary dentition		4			4	ularly of the upper cen-
	Normal				1	1	tral incisors
	Unknown			1		1	
9	Calvarial thickening: See Discussion						
23							

patients fail to erupt or do so one at a time throughout adulthood, seemingly stimulated by the wearing of dentures.[23] An edentulous woman in this series cut a tooth under her plate at the age of 78 years, further emphasizing that permanent teeth may erupt at any time. Fracture of the jaw occurred in 1 woman during dental extraction, indicating that oral surgery for removal of supernumerary teeth usually is difficult.

CLAVICLES

The clavicles showed abnormalities in all 40 affected persons (Table II). Usually the dysplasias are obvious, but flaring of the normally tubular medial third—an underfunnelization type of modeling defect recognized now in the clavicle for the first time in cleidocranial dysostosis—and absence of the sternal end may be subtle in manifestation. Invariably the medial fragment is larger where 2 separate fragments are present, confirming previous observations.[6] Marked pseudarthrosis may occur (3 cases in this study), forming a false joint between the enlarged ends of the clavicles slightly lateral to the midpoint (Fig. 6). The sternal

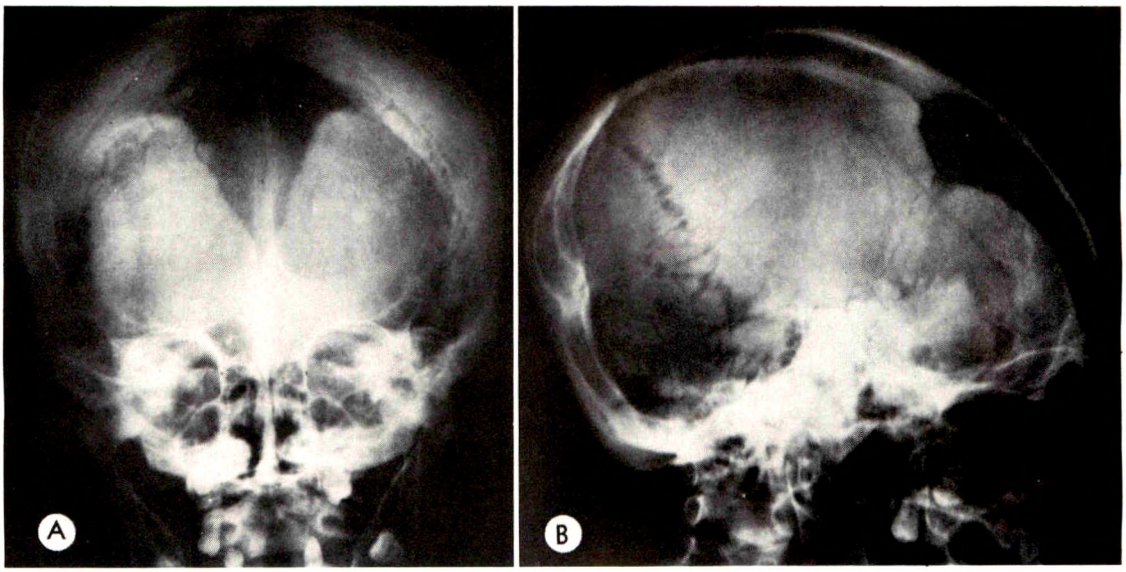


FIG. 2. (*A* and *B*) Open metopic fontanelle in a 75 year old woman. Note the sclerosis of the temporal squama, unossified sutures, squaring of the occipital bone and several unerupted teeth.

fragment is anterior and superior to the acromial fragment at the pseudarthrosis.

SPINE

Spinal abnormalities occur in most patients with cleidocranial dysostosis (Table III). Faulty development of the centers for the embryonic vertebral bodies and vertebral arches[2] accounts for the commonly observed neural arch defects, for hemivertebrae, for defects in the pars interarticularis and for posterior wedging of the thoracic segments (Fig. 7*A*). Spondylolysis (Fig. 8, *A* and *B*) and spondylolisthesis are now added to the wide variety of clinical and roentgen manifestations of this bizarre disorder.

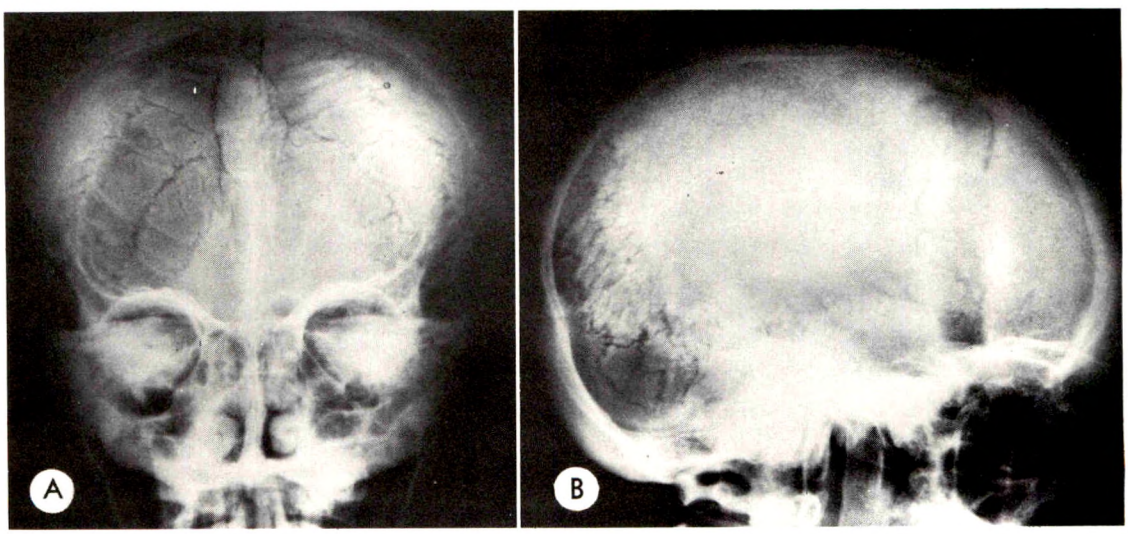


FIG. 3. (*A* and *B*) Numerous wormian bones in a 50 year old man. Note the thickening of the frontal bone.

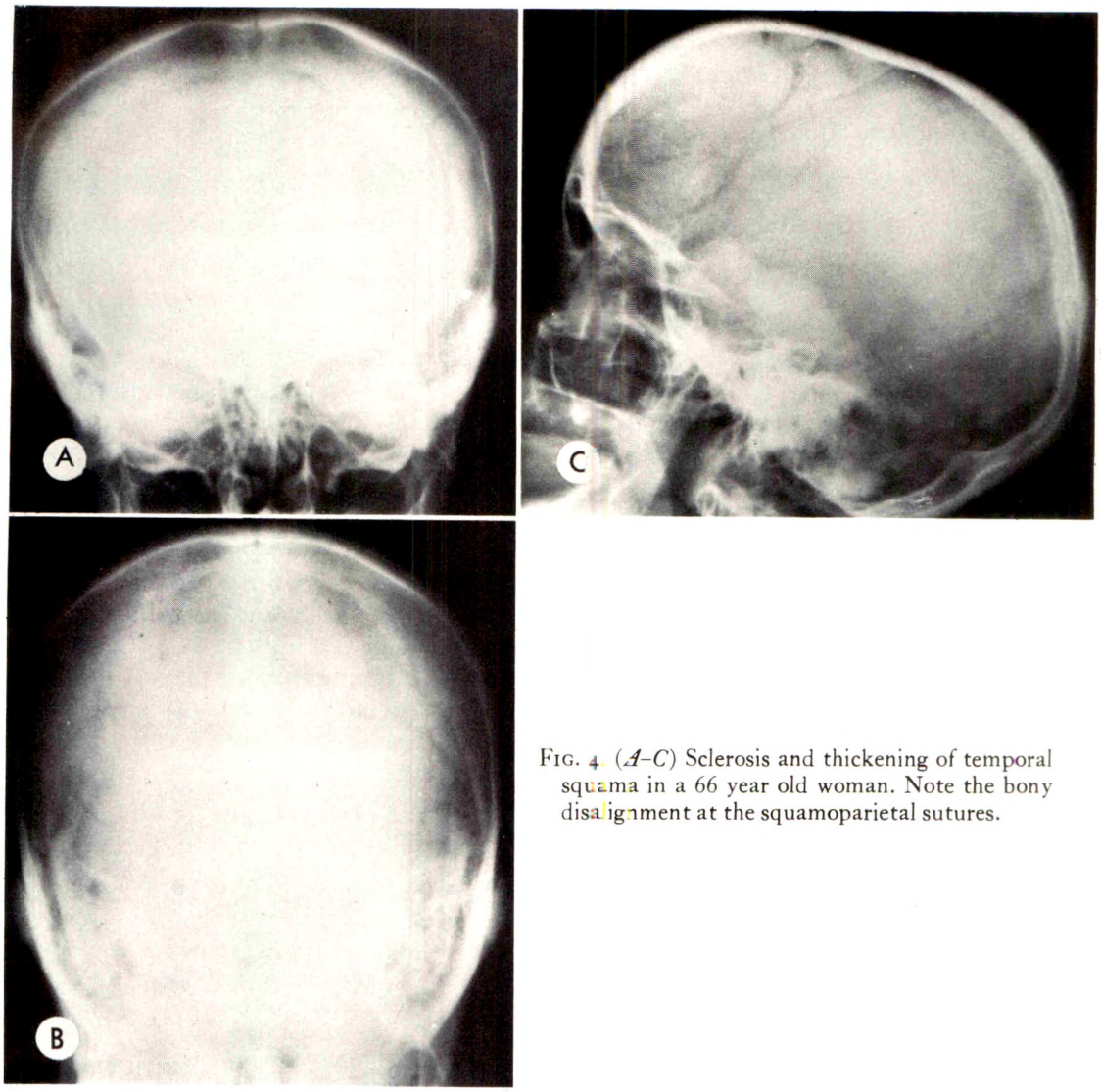


Fig. 4. (*A–C*) Sclerosis and thickening of temporal squama in a 66 year old woman. Note the bony disalignment at the squamoparietal sutures.

THORAX

The cone-shaped thorax (Fig. 7*B*), evident in all 40 patients, appears to be incident to hypoplasia or to delayed development of a segment of the posterior part of the vertebral body, the posterior wedging remaining in many adults as residual deformity. The separate centers for costal process development are apparently uninvolved, since no intrinsic rib abnormality has been observed thus far. Present at birth, the conical thorax remains throughout life as a distinguishing, but not diagnostic, mark of cleidocranial dysostosis.[18]

PELVIS

Pelvic involvement invariably is present in patients with the disorder (Table IV), the appellation pelvico-cleido-cranial dysplasia being used by some physicians to associate the pelvic abnormalities with the clavicular and cranial manifestations of the disease. Ossification of the pubic bone is delayed—considered to be a specific roentgenologic entity—until the age of around 25 years, then leaving residual sclerosis and distortion in most adults. Hypoplasia of the iliac wings results in a loss of normal ilial flaring, producing at times an almost vertical

lateral margin. Widening of the sacroiliac joints gives a "see through" appearance in some patients, presumably incident to the unusual pelvic architecture (Fig. 8*A*).

Five of the 14 parous women (from 4 kinships) had cesarean sections (Fig. 8, *A* and *B*), performed by 5 different obstetricians in 5 different hospitals; in each instance the fetus also had cleidocranial dysostosis. Abnormal labor occurred in several of the other 9 parturients with the disorder, vaginal delivery eventually taking place (babies were normal in 3 and affected in 6). A prenatal diagnosis based on the cranial defect has been reported;[3] a similar fetal abnormality is noted in retrospect in 2 of our patients (Fig. 8, *A* and *B*).

HIPS

Distinctive lateral notching of the proximal femoral ossification center occurred in 3 children near the age of 7 years (Fig. 9, *A* and *B*). The transverse diameter of the femoral neck measured over 4 cm. in about one-half of the affected adults (Table IV) contrasted to a 3.5 cm. average for normal individuals—presumptive evidence of widening of the femoral neck in the disease. Broadening of the femoral head generally accompanied the neck changes.

HANDS

Striking alterations of cleidocranial dysostosis occur in the hands (Table V). The terminal tufts of the phalanges show characteristic pointing, to a degree, resulting from failure of development of the lateral cancellous portion (Fig. 10); this contrasts to the overgrowth that occurs in this area in acromegaly.[11] These hypoplastic changes are particularly evident in the thumbs where shortening and broadening are present clinically. Clinodactyly and brachymesophalangia may occur as isolated findings or present in syndromes other than cleidocranial dysostosis.[19,22]

An accessory epiphysis at the base of the second metacarpal is common in the disorder, occurring occasionally in normal in-



FIG. 5. Segmental calvarial thickening of the occipital bone in a 13 year old boy. Note the loss of occipital curvature, flattening of basi-occiput and supernumerary teeth.

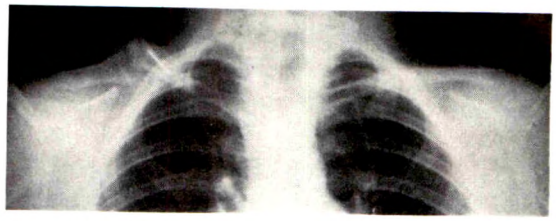


FIG. 6. Right clavicular dysplasia in a 12 year old boy. Note the pseudoarthrosis and the unsuccessful clavicular pinning.

TABLE II

CLAVICULAR ABNORMALITIES IN CLEIDOCRANIAL DYSOSTOSIS

Bilateral	
Two fragments	15
Single medial fragment present	5
Single lateral fragment present	1
Two right fragments and absent medial end of each clavicle	1
Two right fragments and single medial left fragment	1
	23
Right clavicle only	
Two fragments	9
Absent medial end	4
Failure of tubulation of medial end	3
Two fragments with absent medial end	1
	17
Left clavicle only	None

TABLE III

SPINE CHANGES

Ref. No.	Spine	Roentgen Studies	Yr. 2–11	Yr. 12–25	Yr. 26–	Total	Comments
5	Posterior thoracic wedging	24	1	7	9	17	
5	Spina bifida—cervico-thoracic	40	7	8	12	27	
0	Spondylolysis—lumbar	29		2	5	7	Two show spondylolisthesis. All 7 have posterior thoracic wedging and thoracic spina bifida
15	"Bone in bone" vertebrae	29	3			3	One of the 3 cases had normal vertebra at age 23

fants and frequently in hypothyroid children. Individuals in all age groups normally show phalangeal preponderance, the total length of the phalanges of the second finger measuring from 5 to 15 mm. more than the second metacarpal. In contrast, elongation of the second metacarpal frequently follows bony union of the extra epiphysis to the diaphysis (Fig. 10), the metacarpal-phalangeal ratio then approaching unity.

DISCUSSION

The etiology of cleidocranial dysostosis, a deep-rooted anomaly of skeletal development, is unknown. Presumably the cause of the defect, apparently carried in the genes of the affected individual, results in an

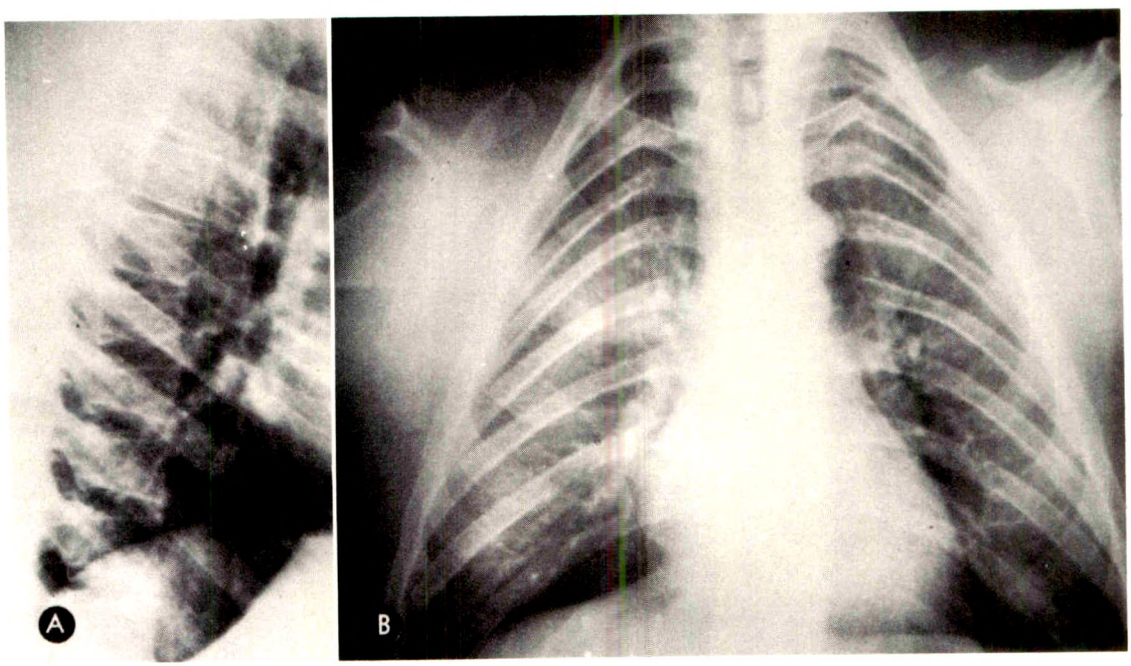


FIG. 7. (*A*) Posterior wedging of the thoracic vertebral bodies in a 33 year old man. (*B*) Cone-shaped thorax and absence of lateral end of each clavicle.

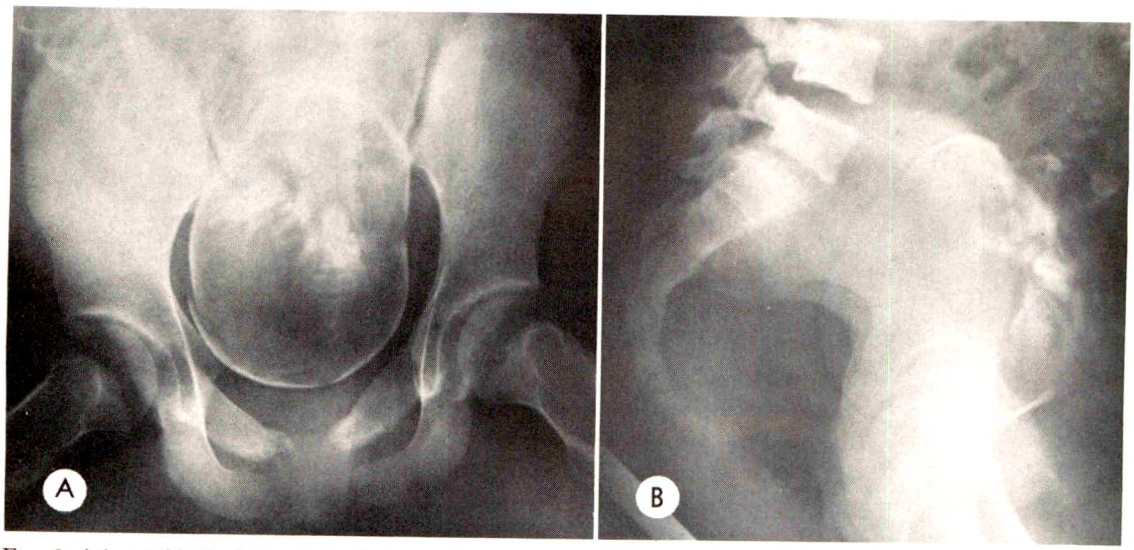


FIG. 8. (*A* and *B*) Cephalopelvic disproportion requiring cesarean section in a 19 year old girl (great granddaughter of woman shown in Figure 2). The affected fetus shows a large defect at the metopic fontanelle. Note the unossified symphysis pubis, hypoplasia of iliac wings, widening of the sacroiliac joints and spondylolysis of the fourth lumbar vertebra.

TABLE IV

PELVIS AND HIP OBSERVATIONS

Ref. No.	Pelvis and Hips	Roentgen Studies	Yr. 2–11	Yr. 12–25	Yr. 26–	Total	Comment
6	Unossified symphysis pubis	29	5	7	0	12	Progressive calcification on follow-up studies on 3 patients
9	Dysplastic symphysis (Sclerosis, incomplete fusion)	29	0	3	9	12	
24	Hypoplasia of iliac wing	29	3	9	5	17	
14	Wide sacroiliac joints	29	3	5	9	17	
0	Broad femoral necks						
	Under 3 cm.		1	0	0	1	
	3.0–3.9 cm.		2	4	4	10	
	4.0–4.7 cm.		0	4	10	14	
0	Notching of proximal femoral ossification center	29	3	0	0	3	
7	Cesarean sections:						
	Of 22 females:						
	4 are children						
	3 are childless						
	1 is unmarried						
	14 are parous—5 of these had cesarean sections (affected fetus in each case)						

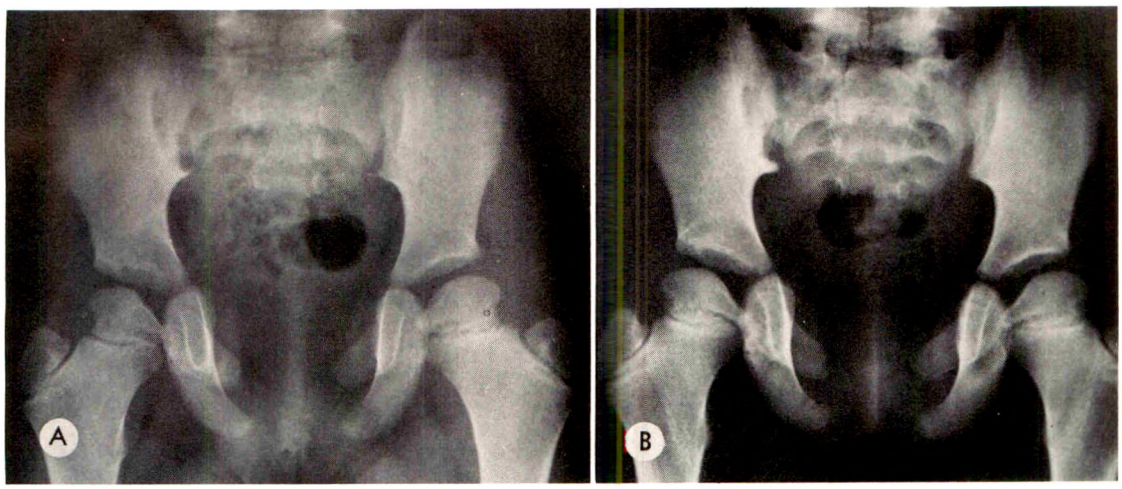


Fig. 9. (*A*) Lateral notching of capital femoral epiphysis in a 7 year old girl (with osteomyelitis of the right sacroiliac joint). (*B*) Partial ossification of the defect 22 months later. Note the unossified pubic bones and hypoplasia of the iliac wings.

early developmental aberration of the mesenchyma, beginning in the earliest weeks of embryonic life and producing retarded ossification of the membranous and cartilaginous precursors of bone. The clavicular manifestations of the syndrome have been known since 1765.[9] Following the advent of roentgen examinations in 1896, a wide variety of bone abnormalities have been recognized, many showing specific roentgenologic patterns at various phases of the disorder in different age groups.

Cleidocranial dysostosis is a hereditary disorder in which considerable disagreement exists concerning the mode of inheritance. Although most authorities consider the transmission pattern to represent the action of a mendelian dominant gene,[7,10,17] some consider it a recessive gene,[20] and others regard it as half dominant and half recessive in nature.[20] These differences in opinion spring from the lack of extensive pedigrees in this disease. The genealogic evaluation of the kinships in this study sup-

Table V

HAND CHANGES

Ref. No.	Hands	Roentgen Studies	Yr. 2–11	Yr. 12–25	Yr. 26–	Total	Comment
11	Pointing of terminal tufts	24	3	8	11	22	Aplasia of terminal tuft of thumbs in 3
19	Brachymesophalangia 2nd and 5th fingers	24	1	2	2	5	
22	Clinodactyly (ulnar) 5th finger	24	3	5	11	19	
11	Accessory epiphyses	24					
	2nd metacarpal base		1	4	7	12	
	2nd and 5th metacarpals		2	1	0	3	
23	Long 2nd metacarpal (metacarpal length = phalangeal length)	24	2	5	9	16	

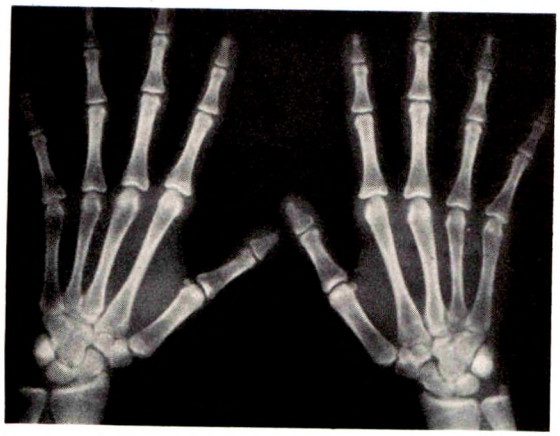


FIG. 10. Pointing of terminal phalangeal tufts in a 31 year old woman. Hypoplastic changes are most evident in the thumbs. Note the long second metacarpal and clinodactyly of the fifth fingers.

ports genetic dominance in both familial and spontaneous cases.

Striking similarity of the roentgen abnormalities observed in the skulls of our adult patients to the changes noted in a neanderthaloid skull[9] and in a necropsied case of cleidocranial dysostosis[23] suggests that the features in the temporal and occipital bones constitute a specific roentgenologic entity, consisting of: (1) a bony shelf at the squamoparietal suture, incident to the osteosclerosis and thickening of the temporal squama; and (2) squaring of the occipital bone, to a certain degree, resulting from both segmental calvarial thickening with ensuing loss of normal posterior curvature and from flattening of the basi-occiput. Basilar invagination apparently does not follow the flattening of the base, as it was not observed in our study.

Of particular interest is that in both cleidocranial dysostosis and osteogenesis imperfecta tarda, resulting from generalized deficiency of both membranous and endochondral bone, calvarial thickening may develop in the adult. The occipital contours show some similarities in these 2 conditions.[4,16,20]

Marked dissimilarity of expression of cleidocranial dysostosis occurs in the clavicles, involving both the site and appearance of the dysplasias. Bilateral abnormalities frequently alternate with right-sided involvement in successive generations. The dysplasias include various combinations of absence, lack of fusion, or incomplete modeling of any of the 3 ossification centers in each clavicle or in the right one alone.

Right clavicular selectivity is unexplained in 17 of the 40 patients in this study and in 58 of 65 cases (6 were bilateral; 1 was left-sided in association with dextrocardia) of congenital pseudarthrosis of the clavicle reported in the orthopedic literature.[1,8,13,21] Naturally the embryonic development of the clavicles is identical, occurring simultaneously;[2] thus an aberrant systemic gene would be expected to affect each clavicle.

The relationship of cleidocranial dysostosis and congenital pseudarthrosis of the clavicle needs further clarification. Eight of the patients reported by Gibson and Carroll[8] as congenital pseudarthrosis of the clavicle had hereditary transmission through 3 generations, were short of stature, and had irregular dentition, presumptive evidence of cleidocranial dysostosis. In addition, some of the reported cases of pseudarthrosis of the clavicle show merely unfused segments without formation of a false joint.

Posterior wedging of the thoracic vertebral bodies, and of the lumbar segments to a lesser degree, appears not to have been recognized as yet in other conditions; thus it is considered to be a specific roentgenologic entity. The frequent occurrence of spondylolysis of the lumbar vertebrae, present in 7 of 29 asymptomatic patients—an incidence of 24 per cent contrasted to 5 per cent in the general population—implies a congenital origin of the defects. Some puzzling aspects of this syndrome as it is manifested in the spine and related bone structures include the propensity of spina bifida defects to occur in the cervicothoracic spine, the invariable conical thorax and the usual short stature of most individuals with the disorder.

Cephalopelvic disproportion necessitated cesarean sections in 5 of 14 parous women

in this study. This gives a primary cesarean section rate of 36 per cent compared to a 3.5 per cent average for our area, and strongly emphasizes the danger of abnormal labor in cleidocranial dysostosis—a point not emphasized previously in this disease. Actually, such a high incidence of cesarean sections is expected in view of the unusual fetal passage and the frequently distorted maternal pelvis. The fetus—affected also with the disease in the 5 cases requiring operative delivery—contributes to the dystocia through its large head and wide parietal diameter. The dysplasias in the ilial and pubic bones often narrow the maternal pelvic diameters and alter the birth canal, these changes becoming critical in these small women.

High penetrance of cleidocranial dysostosis is strangely apparent in the 14 women delivering viable babies, the disorder passing in 11 instances to the offspring. The affected ratio of 78 per cent thus is higher than the 50 per cent incidence expected in mendelian dominance; yet the series obviously is too small for adequate statistical evaluation.

Lateral notching of the capital femoral epiphysis suggests a specific pattern not recognized before in the disorder. This unusual ossification center produces broadening of the femoral head and widening of the femoral neck in some of the older patients.

An interesting observation made during our investigation is the remarkable physical likeness exhibited by many persons with cleidocranial dysostosis. Characteristic facies (large head, small face, prominent furrowed brow, widely-spaced sunken eyes), sloping thorax, abnormal shoulder mobility, prominent knuckles, short thumbs, tapered fingers, rounded nails and usually curvature of the fifth finger—all contribute a degree of sameness to affected individuals, imparting kinship by family and by disease.

Cleidocranial dysostosis is an interesting disorder, challenging students of bone dysplasias to recognize clearly and to classify properly the various patterns produced by the disease process. Naturally the emphasis here is in locating the skeletal abnormalities—establishing where the bone aberrations have become visible on roentgen examinations. Later such information may be of value to cytogeneticists in determining how, when, and why the chromosomal complement in this syndrome departs from the normal, thereby producing a change in the genetic code and eventually affecting intrauterine and postnatal development.

SUMMARY

1. Forty new cases of cleidocranial dysostosis are presented in this genetic and roentgenologic evaluation.

2. Abnormalities observed previously in the disease are assessed including the following hallmark patterns: unossified fontanelles; posterior wedging of the thoracic vertebrae; delayed ossification of the pubic bones; and pointing of the terminal phalangeal tufts.

3. New aspects of the disorder recognized in this study include: distinctive segmental calvarial thickening in the temporal and occipital bones; right clavicular selectivity; high incidence of spondylolysis of the lumbar vertebrae; and characteristic lateral notching of the capital femoral epiphysis.

4. Cephalopelvic disproportion is emphasized as a potential cause of severe dystocia, necessitating cesarean sections in 5 of 14 parous women with the disorder.

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REFERENCES

1. Alldred, A. J. Congenital pseudarthrosis of clavicle. *J. Bone & Joint Surg.*, 1963, *45-B*, 312–319.
2. Arey, L. B. Developmental Anatomy. Seventh edition. W. B. Saunders Company, Philadelphia, 1965, pp. 406–410.
3. Bishop, P. A. Radiologic Studies of the Gravid Uterus. Harper and Row, New York, 1965, pp. 180–183.
4. Campbell, J. A. Roentgen aspects of cranial configurations. *Radiol. Clin. North America*, 1966, *4*, 11–31.

5. ELTORM, H. Case of cleidocranial dysostosis. *Acta radiol.*, 1945, *26*, 69–75.
6. FAIRBANKS, T. An Atlas of General Affections of the Skeleton. E. S. Livingstone, Ltd., Edinburg, 1951, pp. 164–168.
7. FORLAND, M. Cleidocranial dysostosis: review of syndrome and report of sporadic case, with hereditary transmission. *Am. J. Med.*, 1962, *38*, 792–799.
8. GIBSON, D. A., and CARROLL, N. Congenital pseudarthrosis of clavicle. *J. Bone & Joint Surg.*, 1970, *52-B*, 629–643.
9. GREIG, D. M. Neanderthaloid skull presenting features of cleidocranial dysostosis and other peculiarities. *Edinburgh M. J.*, 1933, *40*, 497–557.
10. HERNDON, C. N. Cleidocranial dysostosis. *Am. J. Human Genet.*, 1951, *3*, 314–324.
11. HOLT, J. F., and HODGES, F. J. Significant skeletal irregularities of hands. *Radiology*, 1945, *44*, 23–31.
12. JACKSON, W. P. U. Osteo-dental dysplasia: cleidocranial dysostosis. *Acta med. scandinav.*, 1951, *139*, 292–307.
13. JINKINS, W. J. Congenital pseudarthrosis of clavicle. *Clin. Orthoped.*, 1969, *62*, 183–186.
14. KALLIALA, E., and TASKINEN, P. J. Cleidocranial dysostosis: report of six typical cases and one atypical case. *Oral Surg., Oral Med. & Oral Path.*, 1962, *15*, 808–822.
15. KEATS, T. E. Cleidocranial dysostosis: some atypical roentgen manifestations. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *100*, 71–74.
16. KEATS, T. E. Diffuse thickening of calvarium in osteogenesis imperfecta: further observations. *Radiology*, 1966, *86*, 97–99.
17. LASKER, G. W. Inheritance of cleidocranial dysostosis. *Human Biol.*, 1946, *18*, 103–126.
18. MILES, P. W. Cleidocranial dysostosis: survey of 6 new cases and 126 from literature. *J. Kansas M. Soc.*, 1940, *41*, 462–468.
19. NEWCOMBE, D. S., and KEATS, T. E. Roentgenographic manifestations of hereditary peripheral dysostosis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *106*, 178–189.
20. NEWTON, T. H., and POTTS, D. G. Radiology of the Skull and Brain. Volume 1. Book 2: The Skull. C. V. Mosby Company, St. Louis, 1971, pp. 571–633; 653–661.
21. OWENS, R. Congenital pseudarthrosis of clavicle. *J. Bone & Joint Surg.*, 1970, *52-B*, 644–652.
22. POZNANSKI, A. K., PRATT, G. B., MANSON, G., and WEISS, L. Clinodactyly, camptodactyly, Kirner's deformity and other crooked fingers. *Radiology*, 1969, *93*, 573–582.
23. SOULE, A. B. Mutational dysostosis: cleidocranial dysostosis. *J. Bone & Joint Surg.*, 1946, *28*, 81–102.
24. STEEL, J. P., and WHITAKER, P. H. Case of cleidocranial dysostosis. *Brit. J. Radiol.*, 1937, *10*, 613–618.

Vol. 121, No. 1

CONDENSING OSTEITIS OF THE CLAVICLE: A NEW ENTITY*

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CONDENSING osteitis of the clavicle is a term we have applied to a previously undescribed entity with characteristic clinical, roentgenographic, and histologic findings. The disease is probably related to mechanical stress.

This report will describe our experience with 2 patients who presented with this lesion.

REPORT OF CASES

CASE I. A 26 year old woman, employed as an x-ray technician, was found to have sclerosis of the medial end of the left clavicle on routine chest roentgenograms (Fig. 1). Review of previous chest roentgenograms showed that the sclerosis was present 18 months previously. There was evidence of an increase in the amount of sclerosis during the interval.

On questioning, the patient stated that over a period of 3 years she had had pain along the top of her left shoulder when attempting to raise her left arm above her head.

On physical examination, a fusiform swelling of the medial end of the left clavicle was felt. No increase in temperature was noted, and no tenderness was elicited on palpation.

Laminagraphic sections through both clavicles showed amorphous sclerosis of the medial end of the left clavicle. The articular surface was intact and the joint space was maintained (Fig. 2).

The patient was admitted to the University of Virginia Hospital, and excisional biopsy was performed. A speciman roentgenogram showed the sclerosis to be thickened trabeculae. A possible osteophyte was seen inferiorly (Fig. 3). Histologic examination showed an increase in the amount and thickness of cancellous bone due to reinforcement of the pre-existing trabeculae. The posterior inferior surface showed the presence of periosteal reaction. The proximal end of this periosteal reaction was covered in part by a thin layer of cartilage contiguous with the adjacent articular surface of the clavicle. This pattern strongly suggests and is similar to the formation of an osteophyte (Fig. 4; and 5). These changes were felt to be consistent with a response to mechanical stress.

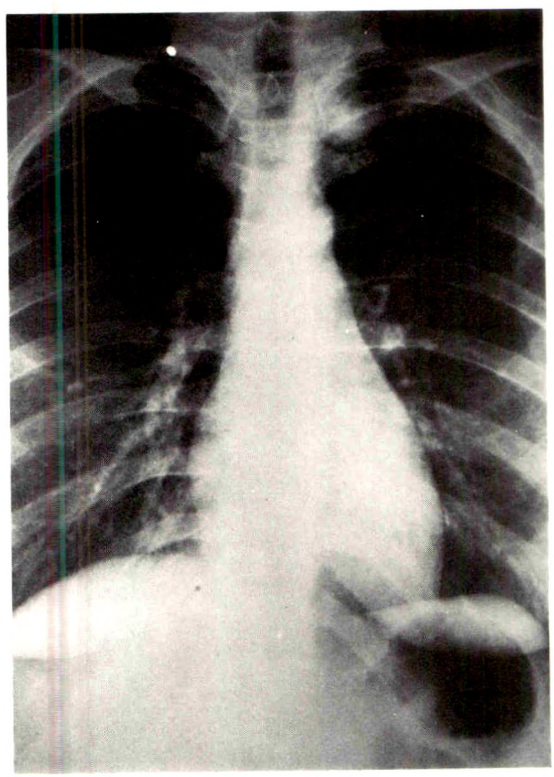


FIG. 1. Case 1. Posteroanterior view of the chest showing sclerosis of the proximal end of the left clavicle.

Since surgery the patient has done well, without recurrence of her pain.

CASE II. A 31 year old woman presented with a 2 year history of pain in the right supraclavicular area, radiating across the top of her right shoulder into the right upper arm. The pain was aggravated by raising her right arm above her shoulder. She played regularly on a women's softball team until the pain limited her ability to throw the ball.

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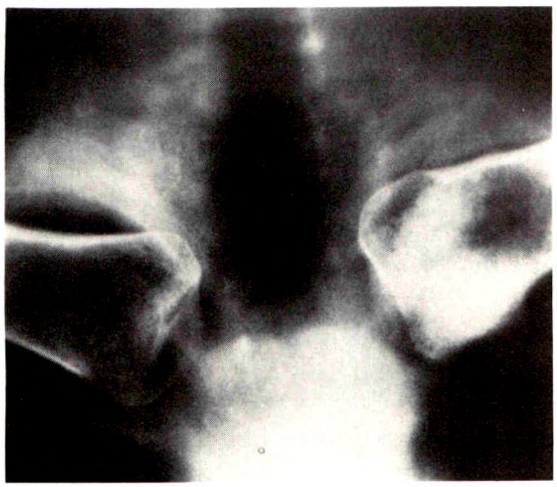


FIG. 2. Case 1. Laminagraphic cut through proximal end of both clavicles. Amorphous sclerosis of the proximal end at the left clavicle is seen. There is no narrowing of the joint space or destruction of the articular surface.

Physical examination showed fullness in the right clavicular area.

A chest roentgenogram revealed a long cervical rib on the right. There was also minimal sclerosis of the medial end of the right clavicle.

The patient was believed to have a right thoracic outlet compression syndrome with a cervical rib and was admitted to the University of Virginia Hospital for removal of that rib. At operation, there was apparent compression of the brachial plexus by the rib. The cervical rib and the first rib were removed and the brachial plexus freed. However, postoperatively, the patient experienced no relief of her symptoms.

One year later, because of persistent pain, a chest roentgenogram was obtained which showed an increase in sclerosis of the medial end of the right clavicle (Fig. 6). Laminagraphic sections through both clavicles showed increased density of the medial end of the right clavicle, amorphous in appearance (Fig. 7).

The patient was again admitted, and excisional biopsy was performed. Histologic examination showed an increase in amount and thickness of cancellous bone. Some periosteal reaction was also present. These changes were believed to reflect a response to mechanical stress.

Postoperatively, the patient did well and has had no recurrence of her symptoms.

DISCUSSION

The clinical, roentgenologic, and histologic findings in the 2 patients reported here are remarkably similar. Pain on abduction of the arm, fullness over the medial end of the clavicle, and sclerosis of the medial end of the clavicle by roentgenographic examination were identical features. The same histologic findings were present in both, consisting of increased amounts and thickness of cancellous bone and periosteal reaction. These findings of trabecular and cortical reinforcement, as well as the pattern of periosteal reaction were considered to be indicative of a response to mechanical stress.

The medial end of the clavicle is an unusual location for a stress response. Both patients carried out strenuous activity involving the sternoclavicular joint, and the possibility of a partial subluxation with altered stress forces across this joint has been given consideration. One lifted and manipulated patients and machinery, while performing her duties as a technician. The other threw a softball, while playing regularly with a team, until the pain prohibited her throwing.

The preoperative diagnostic possibilities considered were: low-grade osteomyelitis;

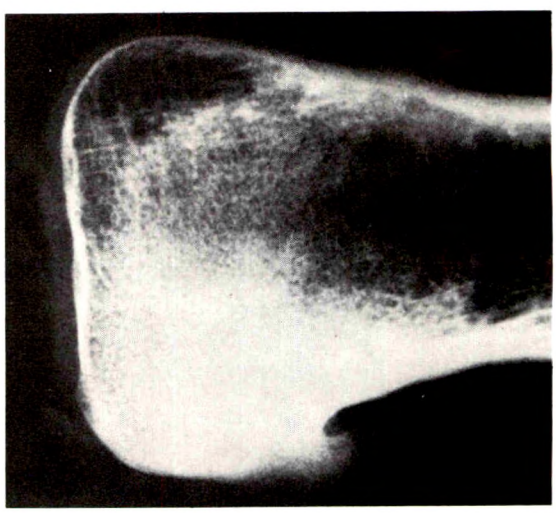


FIG. 3. Case 1. A specimen roentgenogram of the proximal end of the left clavicle showing increase in number and thickness of trabeculae.



Fig. 4. Case 1. Composite photomicrograph of the left clavicular head (4×). (*A*) There is progressive remodeling of the subarticular plate. This area also shows normal marrow activity, which is not seen in the more sclerotic portions of the clavicular head. (*B*) The cancellous bone trabeculae are thickened (see Fig. 5). This histologic feature accounts for the marked increase in density seen in the roentgenogram. (*C*) Reinforced periosteal new bone formation. The surface continuous with the articular surface is lined in part by fibrocartilage.

Paget's disease; a low-grade sclerosing osteosarcoma; degenerative arthritis; or a "condensing osteitis." The lack of bone destruction roentgenographically and the lack of inflammation histologically ruled out low-grade osteomyelitis. The absence of the typical histologic features of active osteoclastic resorption and osteoblastic production of pagetoid bone excluded Paget's disease. Again, lack of evidence of bone



FIG. 5. Case I. Photomicrograph of the cancellous bone trabeculae (30×). The cancellous bone trabeculae are markedly thickened due to appositional reinforcement with lamellar bone.

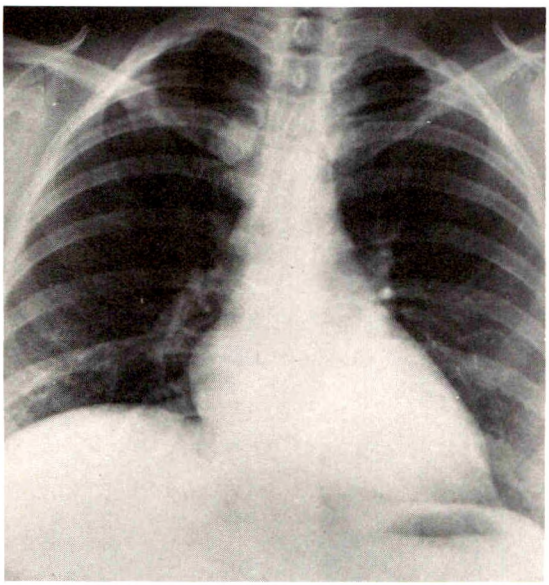


FIG. 6. Case II. Posteroanterior view of the chest showing sclerosis at the proximal end of the right clavicle.

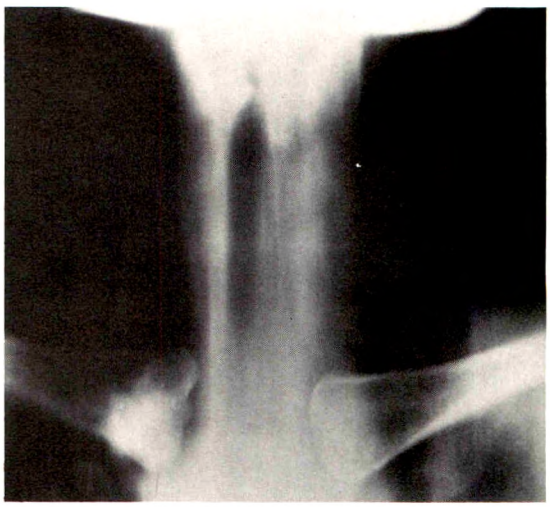


FIG. 7. Case II. Laminagraphic cut through the proximal end of both clavicles showing amorphous sclerosis of the proximal end of the right clavicle. Again, there is no narrowing of the joint space or destruction of the articular surface.

destruction roentgenographically and no evidence of tumor bone formation excluded a neoplastic process.

Roentgenographically, the usual finding of degenerative arthritis, *i.e.*, narrowing of the joint space, disruption of the articular surface, and cyst formation, were absent. However, histologically the presence of subchondral trabecular reinforcement and what appears to be osteophyte formation would be consistent with early changes of degenerative arthritis. Precise evaluation of the articular cartilage with respect to degenerative arthritis is difficult in view of the anatomic nature of the joint and surgical procedure necessary for disarticulation.

Realizing that some of the histologic features are compatible with degenerative arthritic changes, the roentgenographic lesion is perhaps best described as a "condensing osteitis" of the clavicle because of its similarities with condensing osteitis of the ilium and of the pubis. These entities also show an amorphous sclerosis of the bones adjacent to the joint, may be associated with pain and are found more frequently in women during the childbearing years. They are also believed to be a result of mechanical stress and may well represent degenerative changes. Biopsies of these areas have shown no qualitative abnormality; there is only a quantitative increase in the amount of normal bone.

SUMMARY

"Condensing osteitis" of the clavicle, a previously undescribed entity, is characterized by pain along the top of the shoulder, which is aggravated by abduction of the arm.

Roentgenographically, there is increased bone density of the medial end of the clavicle and, histologically, there is an increased quantity and thickness (reinforcement) of normal cancellous bone and sometimes expansion with associated periosteal reactive bone. These changes are felt to be the result of increased or altered mechanical stress across the sternoclavicular joint.

The symptoms are alleviated by surgical excision of the involved end of the bone.

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REFERENCES

1. Brenner, R. A. Monarticular, non-infective subacute arthritis of sterno-clavicular joint. *J. Bone & Joint Surg.*, 1959, *41B*, 749–753.
2. Gillespie, H. W., and Lloyd-Roberts, G. Osteitis condensans. *Brit. J. Radiol.*, 1953, *26*, 16–21.
3. Glay, A. Destructive lesion of clavicle. *J. Canad. A. Radiologists*, 1961, *12*, 117–125.
4. Kohler, A. The Clavicle. In: Borderlands of the Normal and Early Pathologic in Skeletal Radiology. Tenth edition. Grune & Stratton, Inc., New York, 1956, pp. 171–177.
5. Numaguchi, Y. Osteitis condensans ilii, including its resolution. *Radiology*, 1971, *98*, 1–8.
6. Wells, J. Osteitis condensans ilii. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *76*, 1141–1143.

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DIABETIC OSTEOARTHROPATHY*

CLINICAL AND ROENTGENOGRAPHIC OBSERVATIONS IN 90 CASES

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SINCE Jordan[10] first described Charcot's joint in diabetic neuropathy in 1936, case reports have appeared repeatedly in the literature. In 1962, Boehm[3] reviewed the literature and found only 52 cases. Others have reported cases with increasing frequency.[1,6,7,11] One must agree with Rodnan[12] that diabetic osteoarthropathy at least equals tabes dorsalis as a cause for neurotrophic joint disease and now probably exceeds it because of the decrease in frequency of tertiary syphilis and the increasing longevity of diabetics.

The purpose of this paper is to present the clinical and roentgenographic findings in 90 cases of diabetic osteoarthropathy, with roentgenographic findings suggestive of avascular necrosis in 2 cases and pathologic material in 3 cases, 1 of which demonstrated avascular necrosis.

MATERIAL AND METHOD

A review was made of 90 cases of diabetic osteoarthropathy seen at the New England Deaconess Hospital between 1959 and 1972. The criteria for patient selection were the presence of bone lesions of osteoarthropathy and diabetes mellitus. It is of interest that only 30 patients (33 per cent) with bone lesions were seen between 1959 and 1969, while 69 patients (67 per cent) were seen after 1969.

Roentgenograms of the ankle were obtained in the anteroposterior, lateral, and oblique projections; also roentgenograms of the foot in the anteroposterior, oblique, and, when necessary, the lateral projection. The roentgenographic findings were recorded on initial observation and during follow-up visits over a period of from 1 month to 7 years.

CLINICAL OBSERVATIONS

Of the 90 patients, 52 were male (56 per cent) and 38 female (42 per cent). In 41 patients (46 per cent) only the right foot was involved, in 33 (37 per cent) only the left foot, and in 16 (18 per cent) both feet were involved. On the initial examination, active osteoarthropathy was present in 13 of the 16 patients with bilateral involvement. In the remaining 3 patients, 1 presented at initial examination with stable disease in the left and active disease in the right foot, the second developed bilateral disease within 2 years, and the third patient within 3 years and 5 months. The age range was 25 to 78 years with an average of 55 years. Duration of diabetes was 8 months to 43 years, with a mean of 18 years. Eleven patients were treated with hypoglycemic agents and 79 were taking insulin averaging 45 units per day.

Fifty-five patients (61 per cent) had other complications of diabetes such as nephropathy, retinopathy, and peripheral vascular disease. All patients had peripheral neuropathy when first observed. In 42 patients (47 per cent), the onset of neuropathy was not documented. In the remaining 48 patients (53 per cent), neuropathy had been present from 3 months to 18 years.

ROENTGENOGRAPHIC FINDINGS

A total of 106 extremities were examined (Table 1). Vascular calcification, subluxation, and soft tissue swelling were found in a majority of cases, 78, 70, and 58 per cent, respectively.

The bone changes of osteoarthropathy developed within 1 to 12 months in 34 per cent of patients. Most frequently involved

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were the metatarsal bones (84 per cent) and tarsal bones (63 per cent). The tibia and fibula were involved in 11 per cent, the phalanges in 42 per cent, and all bones of the foot and ankle in 9 per cent.

The types of bone reactions observed were sclerosis in 72 per cent, fragmentation in 73 per cent, periosteal new bone formation in 60 per cent, and resorption in 38 per cent. In 15 per cent of extremities, a zone of osteoporosis was noted in the subchondral bone adjacent to the articular surface. The osteoporotic zone was most often seen in tarsal bones but was also seen in the base of the metatarsals at the tarsometatarsal joint; it varied from one to several millimeters in width with sharp to irregular borders. The articular surface could be identified in 50 per cent of these extremities; however, there were areas in 75 per cent where the articular surface was completely destroyed. Immediately adjacent to the osteoporotic zone, the bone was sclerotic (Fig. 1).



Fig. 1. J.B., a 41 year old female with neuropathy for 7 years, was hospitalized with swelling and deformity of both feet. An oblique projection shows sclerosis in the bases of the second, third, and fourth metatarsals with loss of articular surface and sclerosis in the tarsal bones. Note the osteoporotic zone in the subchondral bone of the cuboid and second and third cuneiform (arrow).

REPORT OF CASES

Case 1. J.L., a 36 year old man with 21 years of well controlled diabetes and 16 years of neuropathy, had mild pain in his right great toe for 10 months and deformity for 2 months. A roentgenogram of the right foot revealed dislocation of the proximal first phalanx with fracture of the sesamoid bones and a thin sclerotic band in the second metatarsal head (Fig. 2, *A* and *B*).

A roentgenogram 8 months later (Fig. 2*C*) revealed no change in the bone architecture of the great toe; however, there was flattening of the second metatarsal head with a lucent area at the articular surface, increased density more proximally in the head, and periosteal new bone formation in the neck and shaft. There was fracture with collapse of the third metatarsal head.

The right great toe was amputated because of an infected ulcer on the plantar surface.

Table I

ROENTGENOGRAPHIC FINDINGS IN 106 EXTREMITIES

	Per Cent
Vascular calcification	78
Subluxation	70
Soft tissue swelling	58
Bone involvement (developed within 1 year in 34 per cent)	
Tarsals	63
Metatarsals	84
Tibia and fibula	11
Phalanges	42
All bones	9
Type of bone reaction	
Sclerosis	72
Fragmentation	73
Periosteal new bone	60
Resorption	38
Subchondral osteoporosis	15

Case 11. F.A., a 61 year old man with 30 years of well controlled diabetes mellitus and neuropathy of unknown duration, had vague pains in the left ankle for 1 year and deformity with swelling for 1 month.

The initial roentgenogram (Fig. 3*A*) showed a fracture of the neck of the talus. A roentgenogram 19 months later showed complete dislocation of the head of the talus with marked increase in bone density (Fig. 3*B*). Re-examina-

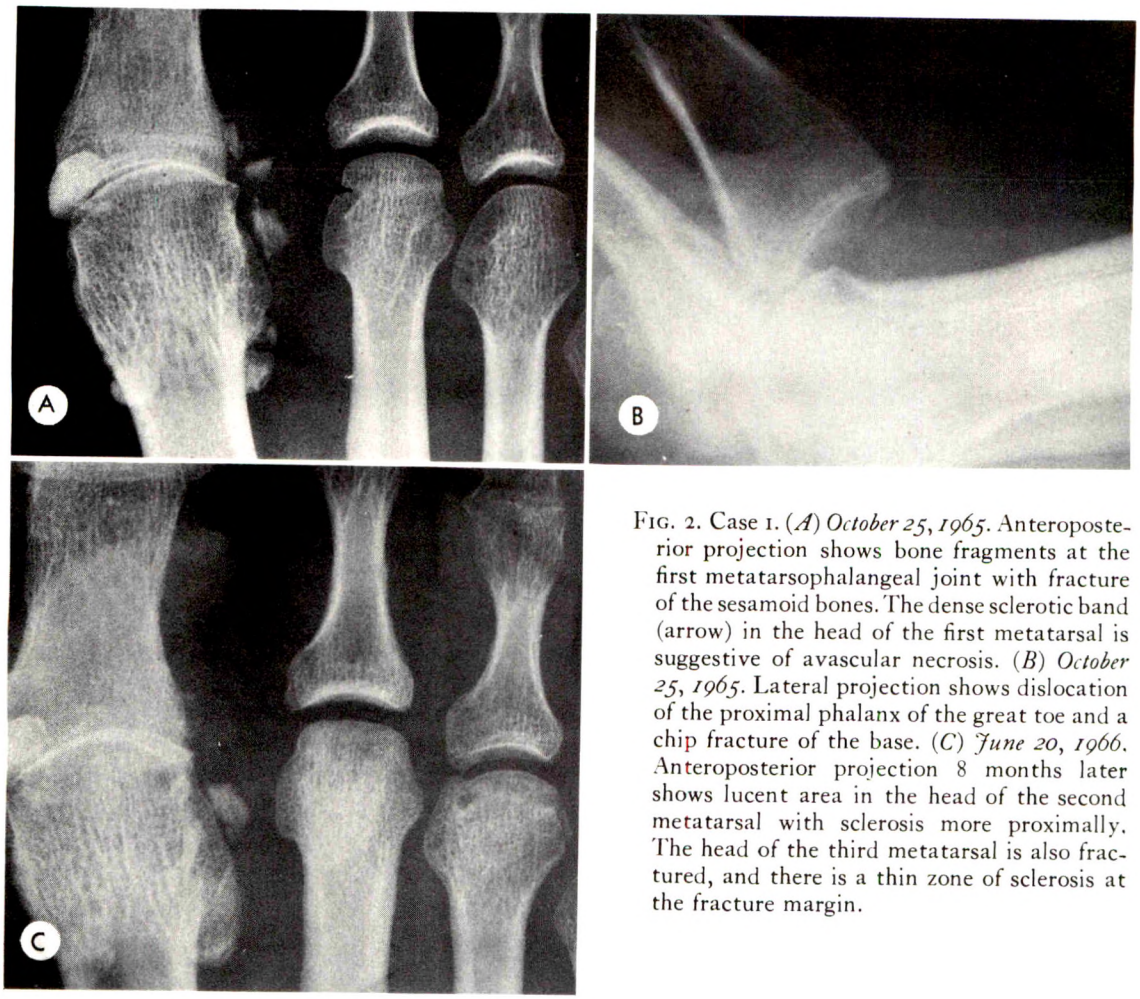


FIG. 2. Case I. (*A*) *October 25, 1965.* Anteroposterior projection shows bone fragments at the first metatarsophalangeal joint with fracture of the sesamoid bones. The dense sclerotic band (arrow) in the head of the first metatarsal is suggestive of avascular necrosis. (*B*) *October 25, 1965.* Lateral projection shows dislocation of the proximal phalanx of the great toe and a chip fracture of the base. (*C*) *June 20, 1966.* Anteroposterior projection 8 months later shows lucent area in the head of the second metatarsal with sclerosis more proximally. The head of the third metatarsal is also fractured, and there is a thin zone of sclerosis at the fracture margin.

tion 3 years and 2 months after the initial fracture showed considerable remodeling with a return to normal density. The trabecular pattern was more clearly defined (Fig. 3*C*).

CASE III. J.W., a 59 year old male diabetic of 30 years with neuropathy of unknown duration. His diabetes has been controlled with 50 mg. of orinase twice daily. Six months before hospitalization, an ulcer developed on the medial aspect of the interphalangeal joint of the right big toe. A roentgenogram revealed normal joint cartilage with erosion of the first proximal phalanx at the joint margin (Fig. 4*A*). Roentgenograms 3 months later showed loss of joint cartilage, subluxation of the distal phalanx, and irregular erosion on the plantar aspect of the head of the proximal phalanx (Fig. 4, *B* and *C*).

The unhealed ulcer (Fig. 4*B*) became infected 2 weeks before hospitalization. Roentgenograms before amputation of the first digit did not reveal evidence of osteomyelitis.

The pathologic specimen (Fig. 4*D*) shows the major bone changes to be cortical rather than medullary. Centrally there are Howship's lacunae with active resorption of large bone spicules. The irregular outline of cortex results from active resorption, focal areas of collapse, fibrosis and reactive new bone formation without inflammation. Clusters of osteoblasts, sometimes associated with osteoid rims, are scattered subperiosteally. Dense fibrous tissue around the bone is most abundant in foci related to the greatest distortion of normal bone contour.

CASE IV. G.F., a 58 year old man with diabetes for 33 years and neuropathy for 3 years. His diabetes has been controlled with 45 units of NPH insulin. A bluish discoloration appeared

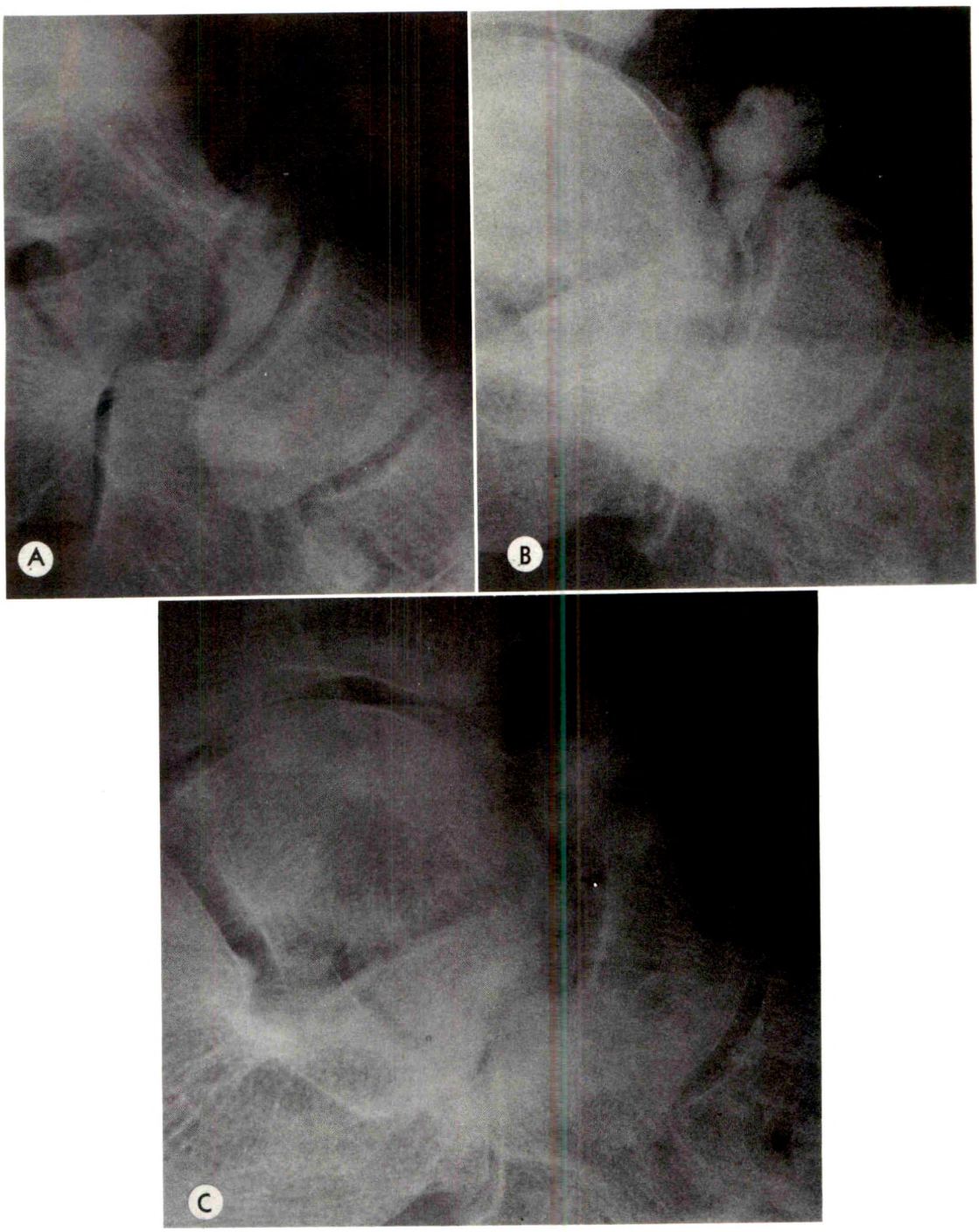


FIG. 3. Case II. (*A*) *May 1, 1962*. Note fracture through the neck of the talus with the articular surfaces congruous. (*B*) *November 8, 1963*. Lateral projection shows complete dislocation of the major portion of the head of the talus. The bone shows marked increase of density with several small lucent areas suggestive of healing avascular necrosis. (*C*) *August 22, 1965*. Re-examination shows remodeling with return to normal density and normal trabecular pattern.

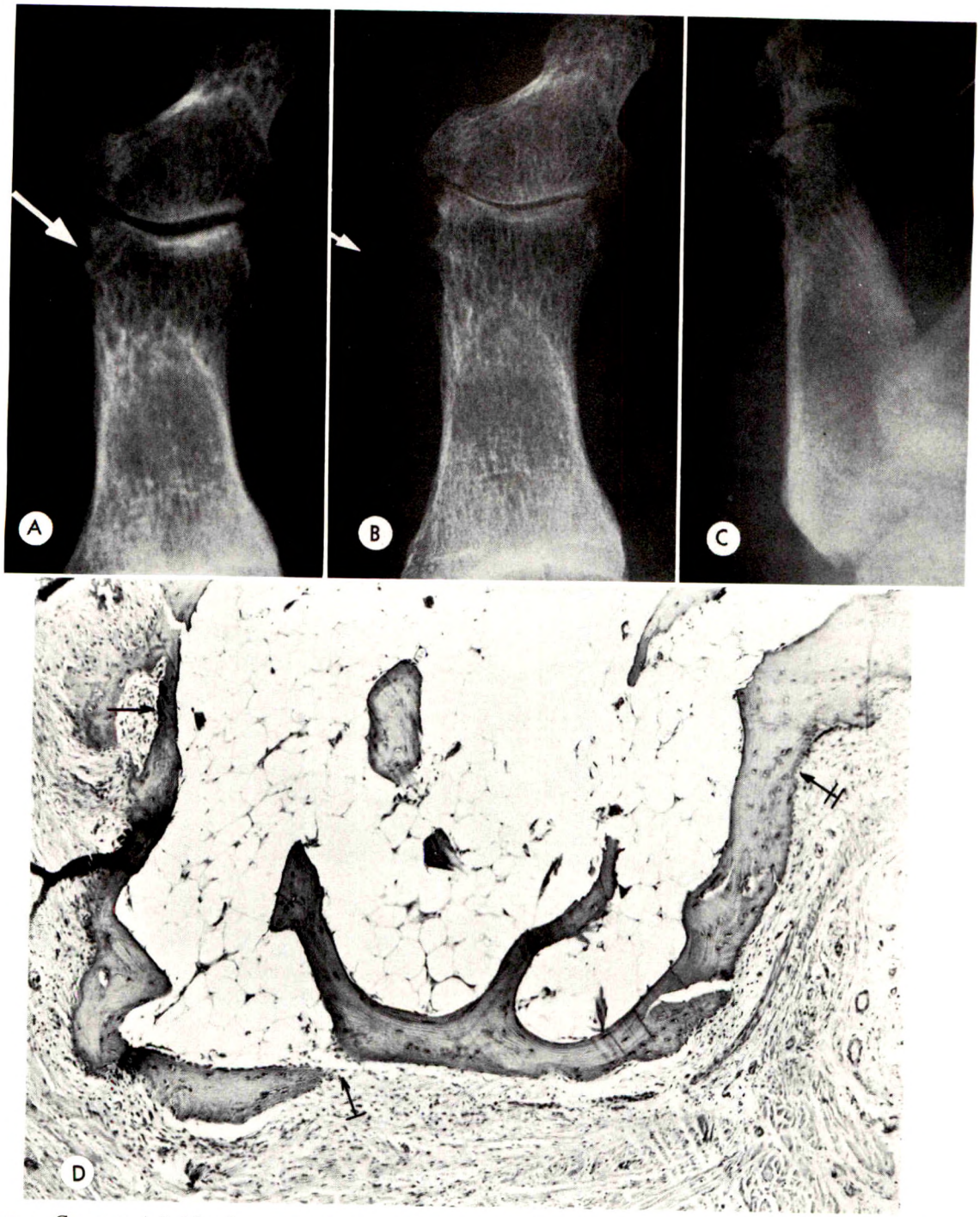


FIG. 4. Case III. (*A*) *March 3, 1972.* Anteroposterior projection shows normal articular cartilage and bone trabeculae. Note small erosion at lateral margin of the head of the first proximal phalanx (arrow). (*B*) *June 6, 1972.* Three months later, there is narrowing of joint cartilage, erosion on the lateral margin of the joint, and soft tissue defect (arrow) representing an ulcer. (*C*) Note subluxation and lucent areas in head of first phalanx. (*D*) Photomicrograph (×85) (section at junction of the head and neck). Note osteoclastic activity with resorption (→), fibrosis and new bone formation (⊢→), and osteoblastic activity subperiosteally (⊩→).

over the left big toe with ulceration within 1 week. Gangrene developed within 2 weeks, and the big toe was amputated. Roentgenograms showed complete disruption of the first metatarsophalangeal joint with fragmentation of the bone and loss of articular cartilage (Fig. 5*A*). The pathologic specimen shows superimposed osteomyelitis.

Ten months later osteoarthropathy developed in the second, third, and fourth metatarsophalangeal joints. A trans-metatarsal amputation was performed because a gangrenous ulcer had developed under the second metatarsophalangeal joint. A roentgenogram (Fig. 5*B*) showed subluxation of the second metatarsophalangeal joint, periosteal new bone formation, bone resorption of the third and fourth metatarsal heads and healed fractures of the third, fourth, and fifth metatarsal necks.

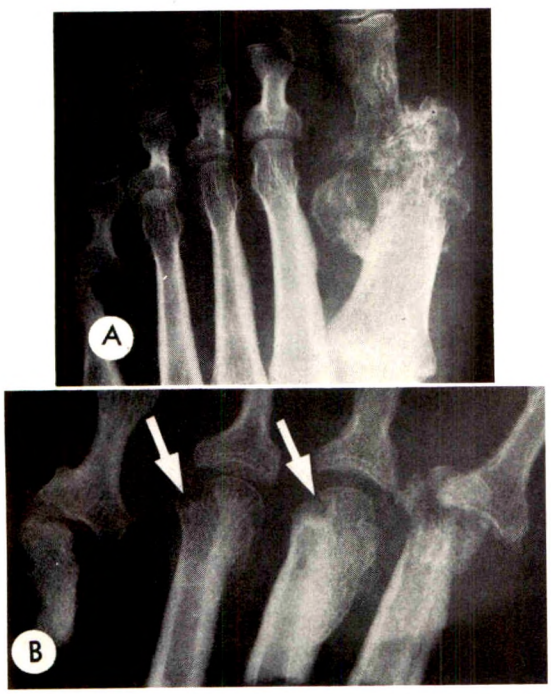


Fig. 5. Case IV. (*A*) *July 20, 1971.* Anteroposterior view of left foot reveals bone fragmentation and complete disruption of first metatarsophalangeal joint with soft tissue swelling. In addition, there is resorption of the fifth metatarsal head. *Pathologic diagnosis*: superimposed osteomyelitis. (*B*) *May 12, 1972.* Specimen shows: periosteal new bone formation, second and third metatarsals; subluxation, second metatarsal head with fragmentation; change in alignment of third and fourth metatarsal heads with their shafts; an area of resorption on lateral aspect of third and fourth metatarsal necks (arrows); and partial resorption of fifth metatarsal head.

The second metatarsal head contained granulation tissue with small clusters of inflammatory cells in the central portion of the head. Diagnosis was superimposed osteomyeltis. In the neck and proximal shaft there was a thick layer of periosteal new bone with no inflammation and reconstructed older bone (Fig. 5*C*).

In *the third metatarsal head*, there was separation of the articular cartilage from reconstructed bone by a layer of fibrous tissue indicative of avascular necrosis (Fig. 5*D*). In the center of the metatarsal head, cement lines were disrupted and at right angles to each other indicating repeated bone replacement and reconstruction (Fig. 5*E*). Laterally, at the junction of the head and neck of the third metatarsal, there was extensive osteoclastic activity (Fig. 5*F*).

The fourth metatarsal head contained foci of irregular bone spicules with occasional osteoclasts, encroaching fibrous tissue, and osteoblastic activity (Fig. 5*G*).

Case V. C.K., a 72 year old man with 24 years of diabetes and neuropathy of unknown duration. Diabetes was controlled with 60 units of NPH insulin daily. He was hospitalized because of an infected ulcer on the right foot which had developed 3 months previously. The ulcer healed with bed rest, soaks, and Keflin, 500 mg., 4 times daily.

A roentgenogram of the right foot revealed deformity of the head of the first metatarsal with central osteoporosis and peripheral sclerosis (Fig. 6*A*).

A punch biopsy in the area of osteoporosis showed a spicule of bone that had been partially resorbed with dense fibrous tissue, predominant osteoblastic activity and new bone formation (Fig. 6*B*).

DISCUSSION

Osteoarthropathy in diabetes has been attributed to primary disease of the bone, ischemia, and neuropathy. A primary metabolic bone defect is an unlikely explanation as there is no evidence of impaired osteoblastic function and healing results in a normal structural bone pattern which may occur spontaneously or when the patient is treated on a strict regimen of

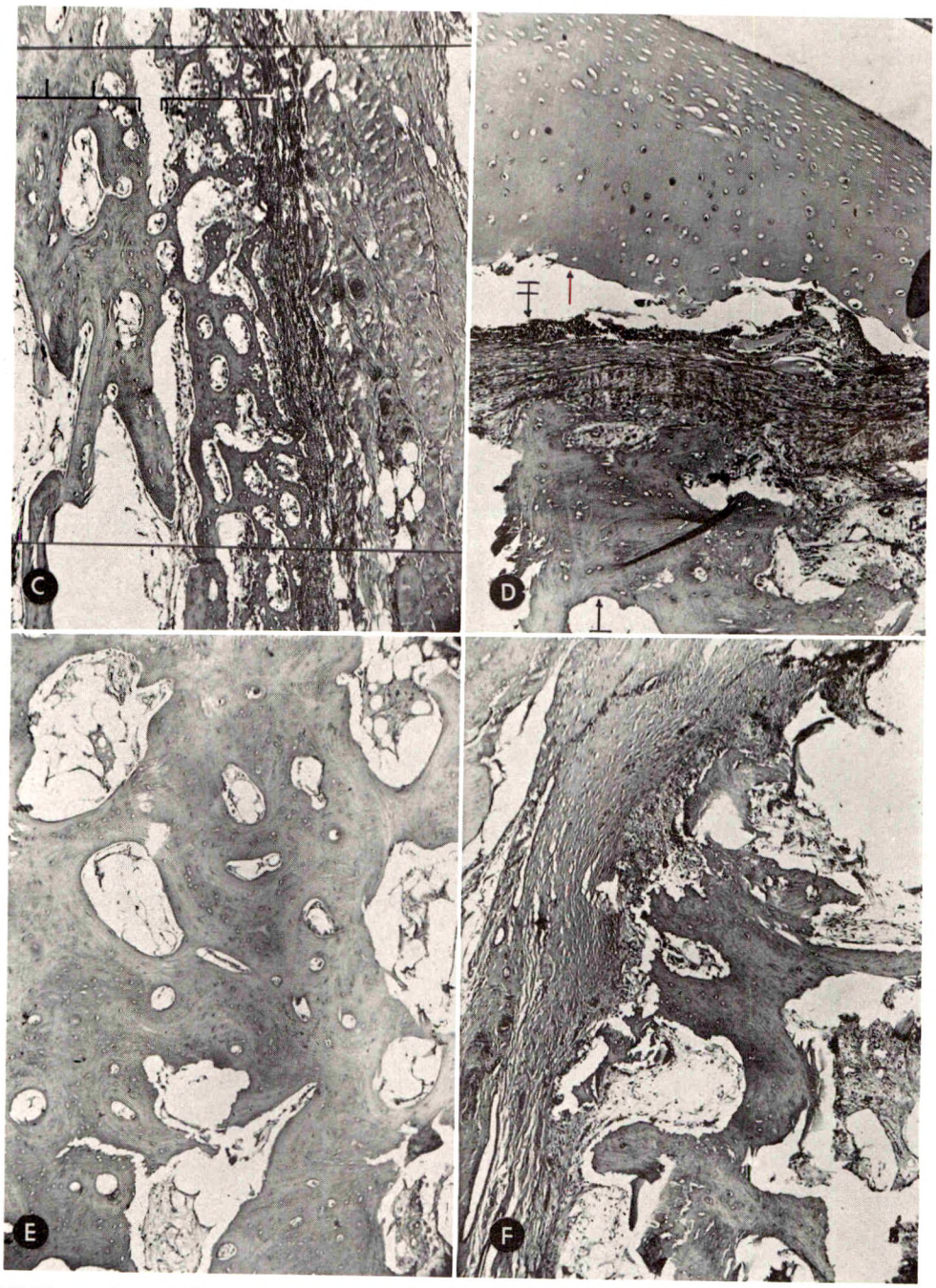
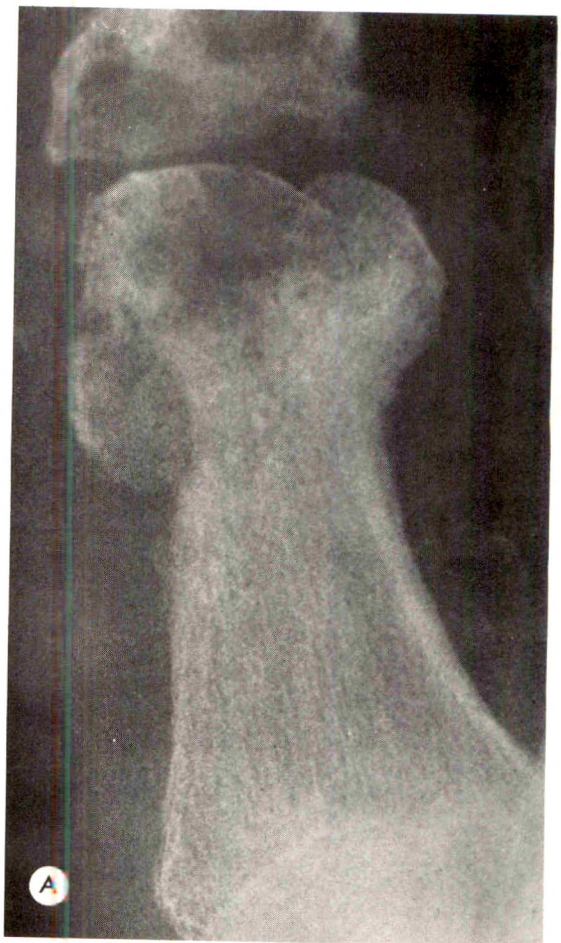
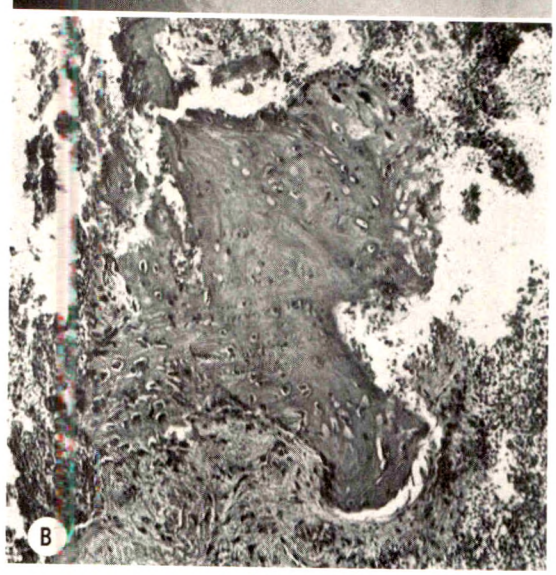


FIG. 5. (*C*) Photomicrograph (×90) of second metatarsal neck shows periosteal new bone formation (⋹) and reconstructed older bone (⋹). (*D*) Photomicrograph (×90) of third metatarsal head. The articular cartilage (→) is separated from the reconstructed bone (⊢→) by a layer of fibrous tissue (⊩→), consistent with avascular necrosis. (*E*) Photomicrograph (×80) of head of third metatarsal. The cement lines are disrupted and are at right angles to each other, indicating repeated bone replacement and reconstruction with a change in the lines of stress. (*F*) Photomicrograph (×80) of lateral aspect of the junction of the head and neck of the third metatarsal (see arrow, Figure 5*B*). Note extensive osteoclastic activity with bone resorption.

Fig. 6. Case v. (*A*) Note deformity of the first metatarsal head and the base of the proximal phalanx. There is central osteoporosis and resorption with sclerosis in the remaining cortical bone. (*B*) Photomicrograph (×130). Punch biopsy in central portion of the first metatarsal head. The spicule of bone has partially been resorbed. There is dense fibrous tissue in addition to predominant osteoblastic activity and new bone formation.





non-weight bearing (Fig. 7, *A* and *B*).

The hypothesis of ischemia is not tenable because ischemic bone does not change in density. Areas of osteoporosis and sclerosis as well as areas of new bone formation such as those seen in osteoarthropathy require good circulation.

Peripheral diabetic neuropathy with trauma is the only common denominator of the disease. Ellenberg[5] has shown that neuropathy is an integral feature of the syndrome of diabetes mellitus rather than a complication of the disease. Woltman and Wilder[14] introduced the hypothesis of luminal narrowing of the vasa nervorum as a cause for neuropathy. Dolman[4] and Greenbaum *et al.*[8] did not demonstrate an anatomic basis for the hypothesis of vascular disease producing neuropathy. They



Fig. 5. (*G*) Photomicrograph (×80) of the lateral aspect of the junction of the head and neck of the fourth metatarsal (see arrow, Figure 5*B*). Note predominant osteoclastic activity with bone resorption and encroaching fibrous tissue.

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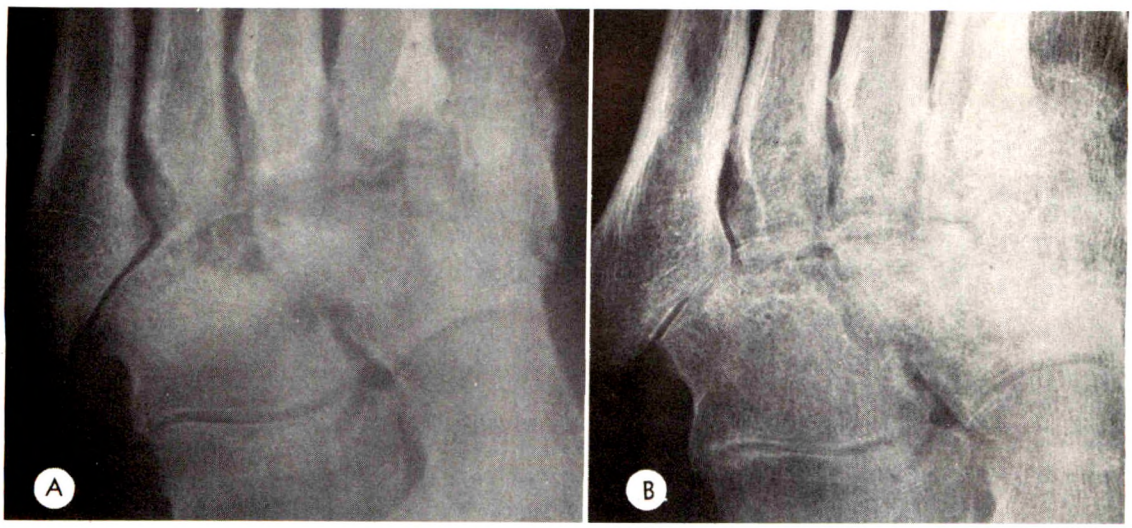


FIG. 7. E.L., a 51 year old man with diabetes for 24 years had a normal roentgenogram of the left foot in January, 1959. He was hospitalized with swelling of the left foot on October 26, 1959. (*A*) *October 26, 1959.* Note sclerosis in the tarsal bones with an area of subchondral osteoporosis in the tarsal and metatarsal bones at the joint interface. (*B*) *October 24, 1966.* Treated with non-weight bearing. Roentgenogram 7 years later shows complete bone reconstitution of the areas of subchondral osteoporosis.

considered it primary neuronal degeneration affecting both motor and sensory nerves, anterior horn cells, and posterior root ganglia.

The bone and soft tissue changes in diabetic osteoarthropathy are identical to those seen in other pathologic entities which combine sensory impairment of the extremity with trauma such as leprosy, syringomyelia, tabes dorsalis, scleroderma, and hereditary sensory peripheral neuropathy.

Clinically, the diabetic patient has a swollen, warm, nontender, almost painless foot or ankle. If only a phalanx or metatarsal is involved, the patient may be asymptomatic, and the changes may be discovered as an incidental roentgenographic finding. These clinical features correlated with roentgenographic findings of soft tissue swelling and joint effusion which were present in the majority of our patients (Fig. 8, *A* and *B*). As injury to the ligamentous structures progresses, subluxation with disruption of the articular surface occurs. These changes are more easily demonstrated in tarsometatarsal and intermetatarsal joints than when they occur in the tarsal area.

When joint surfaces become incongruous, bone destruction becomes more severe with fracture and fragmentation resulting in bony spicules in the synovium and soft tissues (Fig. 9, *A–C*). At this stage of the disease a zone of subchondral osteoporosis is frequently seen (Fig. 1). This zone is identical to that in Seewald's [13]case report with biopsy material. It most likely represents fracture in the subchondral bone, because it was subsequently replaced by bone when the lesion healed. This also explains Seewald's finding of normal articular cartilage in the early stage when the articular surface is intact. In several cases treated with non-weight bearing, the osteoporotic zone was replaced by trabecular bone and the sclerotic areas returned to normal density (Fig. 7, *A* and *B*).

In areas which have progressed, the articular cartilage is completely destroyed and masses of sclerotic bone are separated by irregular zones of soft tissue density.

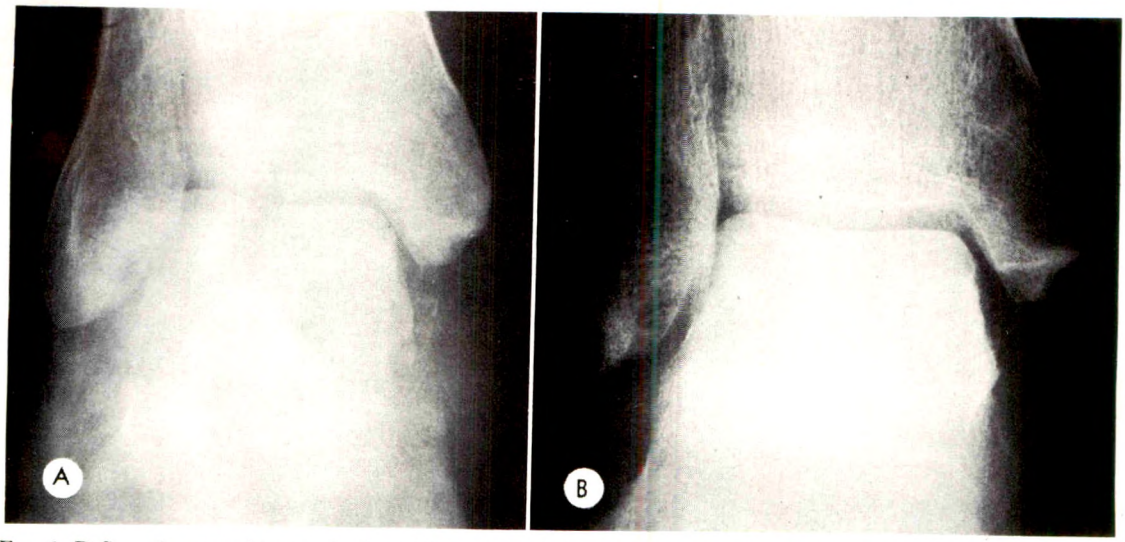


Fig. 8. D.S., a 62 year old man, had swelling of the right ankle for 7 weeks. He was treated with non-weight bearing and the swelling decreased, but increased with ambulation. (*A*) *February 6, 1971.* There is soft tissue swelling and calcification in the posterior tibial artery but intact bone architecture. (*B*) *June 9, 1971.* Four months later the swelling has subsided and the bone architecture remains intact.

Healing can only occur with bone union obliterating the joint.

Sclerosis was seen most frequently in the tarsal bones, and its exact cause is difficult to explain. Increases in density also develop as bone conforms to Wolffs' law by increased deposition with increased stress or when there is a change in the lines of stress, as occurs with disruption of the tarsal arch.

The band of increased density in the second metatarsal head noted in Case III (Fig. 2, *A–C*) is identical to the changes of avascular necrosis in the femoral neck of rabbits described by Bobechko and Harris.[2] Histologically, changes of avascular necrosis were present in the head of the third metatarsal, as in Case IV (Fig. 5*D*), with separation of the articular cartilage from reconstructed bone by a layer of fibrous tissue.

The changes of increased density in the talus of Case II (Fig. 3*B*) support the concept of an avascular incident with laying down of new bone on dead trabeculae. The bone returned to normal density when there was healing with remodeling.

There was no evidence of renal osteodystrophy, secondary hyperparathyroidism, elevated serum calcium, phosphorus, or elevated blood urea nitrogen to explain osteosclerosis, although several patients had marked proteinuria. There was no correlation between the patients with proteinuria and those with osteosclerosis.

Periosteal new bone was not seen in the tarsal bones because they heal primarily by endosteal repair. As expected, periosteal new bone production was seen almost exclusively in the metatarsals and phalanges, since long bones heal primarily by periosteal new bone production.

The "sucked candy" or "sharpened pencil" deformity that occurs at the distal end of the metatarsal bones is more difficult to explain. It most likely results from a combination of neuropathy and repeated episodes of trauma manifested by a series of fractures with attempts at healing without success, resulting in concentric resorption of bone. The changes are identical to those seen in non-united fractures in long bones; concentric resorption produces a smooth tapering with attempts to form a false joint at the fracture site (Fig. 10).

The findings in Figure 5*E* indicate attempts at healing with collagen fibers laid

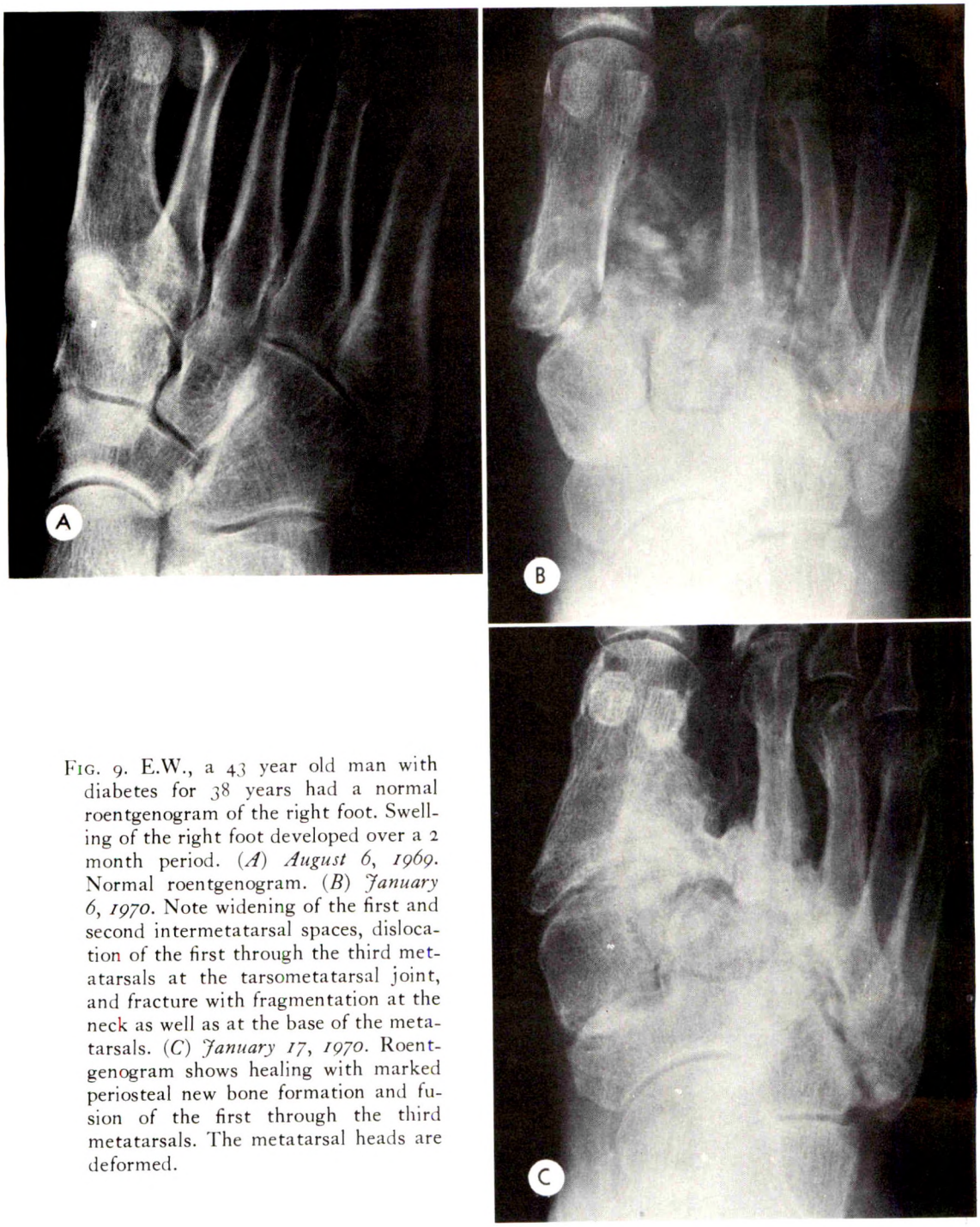


FIG. 9. E.W., a 43 year old man with diabetes for 38 years had a normal roentgenogram of the right foot. Swelling of the right foot developed over a 2 month period. (*A*) *August 6, 1969.* Normal roentgenogram. (*B*) *January 6, 1970.* Note widening of the first and second intermetatarsal spaces, dislocation of the first through the third metatarsals at the tarsometatarsal joint, and fracture with fragmentation at the neck as well as at the base of the metatarsals. (*C*) *January 17, 1970.* Roentgenogram shows healing with marked periosteal new bone formation and fusion of the first through the third metatarsals. The metatarsal heads are deformed.

down in one plane. When there is refracture or change in the lines of stress, new collagen fibers are laid down at right angles to the previous ones, resulting in a mosaic-like pattern. The large number of osteoblasts and osteoclasts seen in Figure 5*F* indicate rapid resorption and deposition as re-fracture and change in lines of stress occur.

Superimposed on these events, one must postulate or suspect avascular episodes with necrosis and healing after revascularization, as evidenced by the fibrous tissue replacement shown in Figures 5*G* and 6*B*, and separation of the articular cartilage and fibrous tissue demonstrated in Figure 5*D*.

Soft tissue infection may play a secondary role; however, it is difficult to visualize it as the sole cause as suggested by Hodgson *et al.*[9]

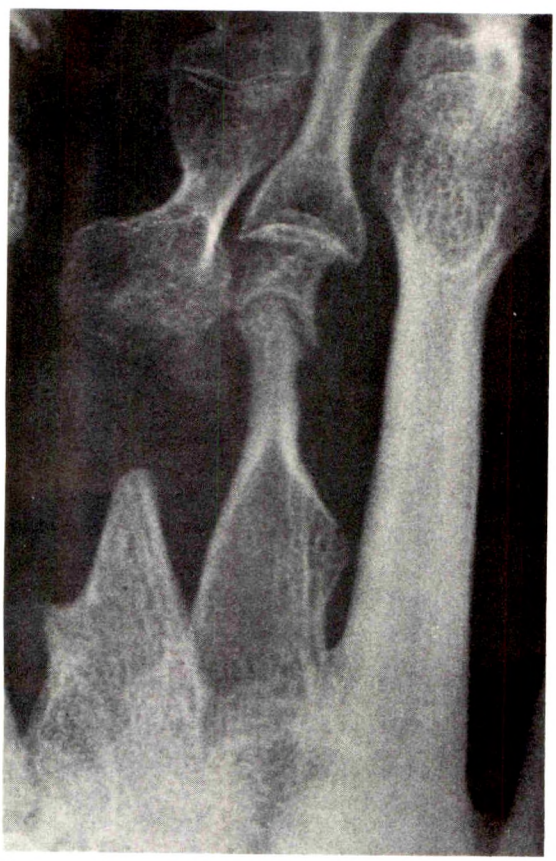


FIG. 10. S.C., a 61 year old woman with 9 years of diabetes mellitus and several years of neuropathy entered with advanced bone changes. There is resorption of the head of the third metatarsal with pseudoarthrosis and secondary hypertrophy of the fourth metatarsal because of increased stress.

SUMMARY

The clinical and roentgenographic findings in 90 cases of diabetic osteoarthropathy are described with emphasis on 2 case reports resembling avascular necrosis, and pathologic correlation in 3 cases, 1 of which is consistent with avascular necrosis.

The roentgenographic manifestations are: swelling of the joint; subluxation; fracture fragmentation; a zone of subchondral osteoporosis; and sclerosis in the adjacent bone. The bone may heal and retain its normal architecture at this stage. If the disease progresses, the articular surface is completely destroyed with resorption and dissolution of the subchondral bone. Healing, if it occurs, is by bone fusion with loss of the joint.

The "sucked candy" or "sharpened pencil" deformity of the metatarsal head most likely results from a combination of neuropathy with repeated episodes of trauma manifested by a series of fractures with attempt at healing without success. There may be superimposed episodes of avascular necrosis. The end result is concentric resorption. Infection may play a minor role.

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REFERENCES

1. AZERAD, E., STUHL, L., LUBETZKI, J., and SLOTING, M. Étude radiologique des ostéopathies du diabète sucré, "le pied diabétique." *Ann. de radiol.* 1963, *6*, 421–436.
2. BOBECHKO, W. P., and HARRIS, W. R. Radiographic density of avascular bone. *J. Bone & Joint Surg.*, 1960, *42-B*, 626–632.
3. BOEHM, H. J. Diabetic Charcot joint: report of case and review of literature. *New England J. Med.*, 1962, *267*, 185–187.
4. DOLMAN, C. L. Morbid anatomy of diabetic neuropathy. *Neurology*, 1963, *13*, 135–142.
5. ELLENBERG, M. Diabetic neuropathy: consideration of factors in onset. *Ann. Int. Med.*, 1960, *52*, 1067–1075.
6. FRIEDMAN, S. A., and RAKOW, R. B. Osseous

lesions of foot in diabetic neuropathy. *Diabetes*, 1971, *20*, 302–307.

7. GONDOS, B. Roentgen observations in diabetic osteopathy. *Radiology*, 1968, *91*, 6–13.
8. GREENBAUM, D., RICHARDSON, P. C., SALMON, M. V., and URICH, H. Pathological observations on six cases of diabetic neuropathy. *Brain*, 1964, *87*, 201–213.
9. HODGSON, J. R., PUGH, D. G., and YOUNG, H. Roentgenologic aspects of certain lesions of bone: neurotrophic or infectious. *Radiology*, 1948, *50*, 65–70.
10. JORDAN, W. R. Neuritic manifestations in diabetes mellitus. *Arch. Int. Med.*, 1936, *57*, 307–366.
11. POGONOWSKA, M. J., COLLINS, L. C., and DOBSON, H. L. Diabetic osteopathy. *Radiology*, 1967, *89*, 265–271.
12. RODNAN, G. P. Neuropathic joint disease (Charcot's joint). In: Arthritis and Allied Conditions. Seventh edition. Edited by J. L. Hollander *et al.* Lea & Febiger, Philadelphia, 1966, pp. 1109–1119.
13. SEEWALD, K. Zur Osteoarthropathia diabetica—Bericht über einen Fall in Verbindung mit einer Studie über die Belastungsverhältnisse des menschlichen Fusses. *Wien. klin. Wchnschr.*, 1969, *81*, 53–54.
14. WOLTMAN, H. W., and WILDER, R. M. Diabetes mellitus: pathological changes in spinal cord and peripheral nerves. *Arch. Int. Med.*, 1929, *44*, 576–603.

SIMILAR BONE MANIFESTATIONS OF SCLERODERMA AND RHEUMATOID ARTHRITIS*

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JOINT involvement occurs frequently in scleroderma and is often mistaken for rheumatoid arthritis. In addition, positive serologic studies considered characteristic for rheumatoid arthritis are also found in scleroderma.[7] Since the hands and wrists are predominantly involved in both diseases, roentgenographic examination[5] should provide a useful means of differentiation. In the majority of patients, the features are distinctive. However, abnormalities considered pathognomonic for arthritis are also found in scleroderma. After reviewing the roentgenographic changes of the hand and wrist in patients with scleroderma, we were impressed with the difficulty in distinguishing the 2 diseases in some patients. Unfortunately, most textbooks as well as the radiologic literature stress the classical features encountered in scleroderma and minimize the frequency of coexisting changes.[2,6]

In this report we will: (1) review all the roentgenographic findings noted within the hands and wrists in our patients with scleroderma; (2) compare some of these features to those seen in rheumatoid arthritis; and (3) note any differentiating findings that may be used in those cases with overlapping manifestations.

MATERIAL AND METHOD

The roentgenographic findings of the hands and wrists in 24 patients with proven scleroderma were reviewed and compiled. Eighteen of these patients were females and 6 were males. The diagnosis of scleroderma was established by reviewing the nature of the clinical findings and the development of the disease. All patients had symptoms of arthralgia or mild arthritis. Only 1 patient (I.S.) clinically demonstrated joint changes that resembled rheumatoid arthritis. Serologic studies, *e.g.*, latex fixation, were performed in 14 patients and were positive in 3. For one reason or another, no studies were obtained on the other 7. Roentgenographic examination was performed on a random basis and only sporadic cases are available. The routine views obtained were those of the hand and wrist exposed in the frontal, lateral and oblique positions using screen cassettes. Although early and subtle changes may have been missed by not utilizing a non-screen technique, the final analysis is probably not altered. Esophagography was performed in 20 patients and 12 of these demonstrated classical changes in the distal esophagus.

ROENTGENOGRAPHIC FINDINGS

Typical roentgenographic manifestations of scleroderma were encountered in the majority of cases (Table 1). Absorption of the ungual tuft was found in 8 patients, soft tissue calcification in 13 patients and skin atrophy in 9 patients. These findings were minimal in some and extensive in others. Osteoporosis was present in 10 patients, and was minimal in 4, diffuse in 4, and juxta-articular in 2. (Determination of osteoporosis was purely subjective and therefore not accurate. However, a similar subjective estimate was used in studying patients with rheumatoid arthritis.)

Joint narrowing was encountered in 11 patients (46 per cent). This was predominantly localized to the wrist with the carpal bones and the radiocarpal joints the most affected (Fig. 1). The interphalangeal joints were also involved, but in a random and asymmetric manner. The affected individ-

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TABLE I

ROENTGENOGRAPHIC MANIFESTATIONS OF SCLERODERMA

Patient	Sex	Osteoporosis	Joint Narrowing	Bone Destruction	Bone Erosion	Mutilation	Acro-Osteolysis	Calcium	Skin Atrophy
H.G.	F	+ generalized	−	−	−	−	−	+ minimal	−
L.F.	M	−	+ carpal bones	−	+	−	−	+	+
P.M.	F	−	−	−	−	−	−	+ ungual	+
H.R.	F	+ generalized	+ all joints	+	+	+	?	+ not extensive	+
L.W.	M	−	+ minimal, radio-carpal	−	+	−	−	−	−
S.D.	F	−	+ minimal, distal phalangeal	−	−	−	+	+ massive	−
Y.M.	F	−	−	−	+	−	−	−	−
62270	M	−	−	−	−	−	+ minimal, single tuft	−	+
I.S.	F	−	+ radio-carpal	−	+	−	−	−	−
V.P.	F	−	+ radio-carpal, carpal and others	−	+	−	−	−	minimal
A.H.	F	−	−	−	−	−	−	−	−
A.J.	F	+ juxta-articular	−	−	−	−	−	−	−
P.P.	F	−	−	−	−	−	+ left 2nd digit	−	−
W.H.	M	+ minimal	+	+	−	+ distal	−	−	−
L.L. 2 years later	F	− + minimal	−	− minimal	+ +	− −	− −	− + massive	− −
A.S.	F	+ juxta-articular	+	−	−	−	+ all distal phalanges gone	+	+
S.C.	F	−	+	minimal	+	−	−	+	−
M.B. 2712	F	−	−	−	?	+	+	−	+
L.V.	M	−	−	−	+	−	−	+ one ungual tuft	−
L.O.	M	+ minimal diffuse	+	+	+	++	−	+	−
M.R.	F	−	−	−	−	−	+ most ungual tufts	minimal	−
A.P.	F	+ diffuse	+ minimal, carpo-metacarpal	−	+	−	−	+++	
S.S.	F	+ minimal	−	−	+	−	+ index finger 3rd and 4th fingers	minimal	+
F.W.	F	+	?	−	−	+ 2nd digit		+	+
Total 24	F-18 M-6	10	11	5	13	4	8	13	9

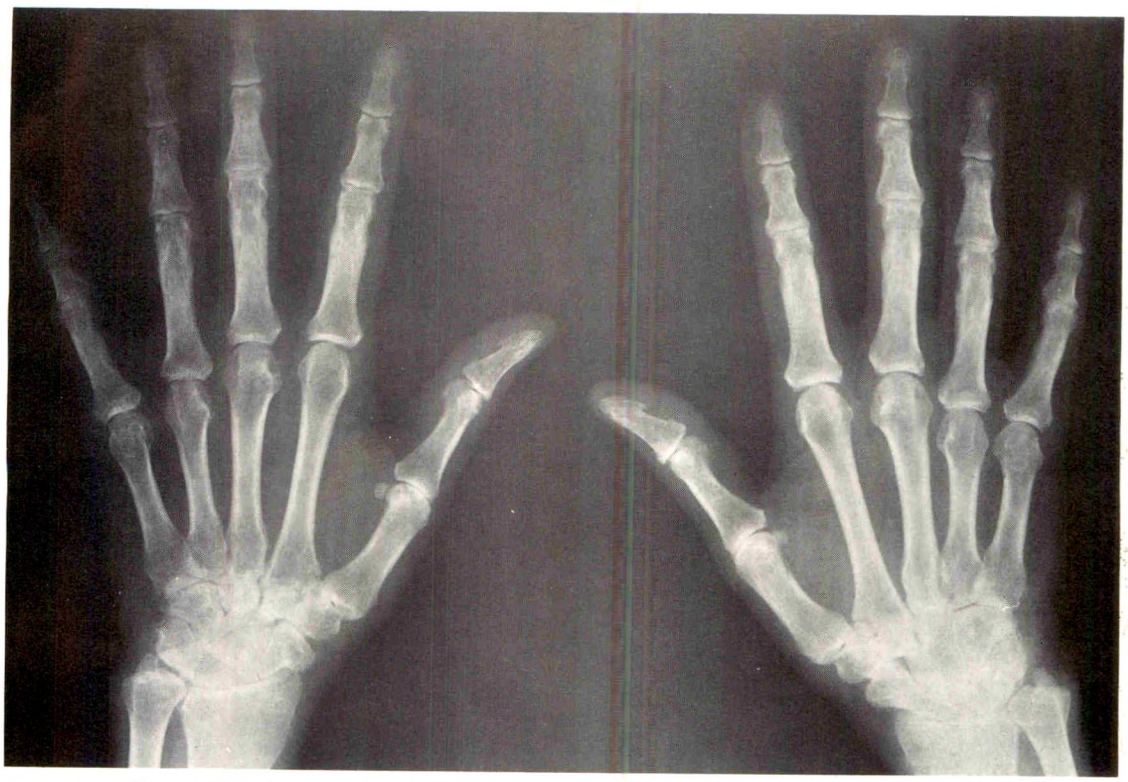


FIG. 1. V.D. Moderate arthritic changes are noted in the wrist. The overlying skin appears atrophic, but no other changes are apparent.

ual joint was for the most part smoothly narrowed, although irregularities were occasionally detected on the cartilaginous surface. The distal interphalangeal joints of the thumbs in 1 patient were markedly eroded (Fig. 2). In this case, as in 2 others, dislocation of all metacarpo-phalangeal joints was present. An irregular surface associated with splaying and flattening of the distal portion of the radius and the carpal bones was found in some cases (Fig. 1–4). Minimal joint effusion was also encountered. This was neither diffuse nor symmetric and involved the interphalangeal joints only occasionally.

Bone erosions of varying size and location were encountered in 13 patients (Fig. 1–6). A large, broad, notch-like erosion was evident within the distal radio-ulnar joint in 6 patients (Fig. 2–4). The navicular bone demonstrated a smooth defect along its medial border in 4 patients (Fig. 5, *A* and *B*). Marginal erosions were seen in the greater multiangular and lunate bones, the metacarpals, and the phalanges (Fig. 6, *A–C*). These erosions were small and sharply demarcated.

BONE AND JOINT CHANGES COMMON FOR SCLERODERMA AND RHEUMATOID ARTHRITIS

(Tables II and III)

In 7 patients (29 per cent), the roentgenograms of the hand and wrist demonstrated many roentgenographic features encountered in rheumatoid arthritis. This was quite evident in the wrist. Radio-ulnar apposition probably associated with destruction of the triangular fibrocartilage was frequently observed. This accounted for the broad irregular compression erosion along the medial aspect of the radius (Fig. 2–4). Alteration of the ulnar styloid process was also noted. This was either eroded, irregular, or completely destroyed (Fig. 2 and 3). Both these changes have been re-

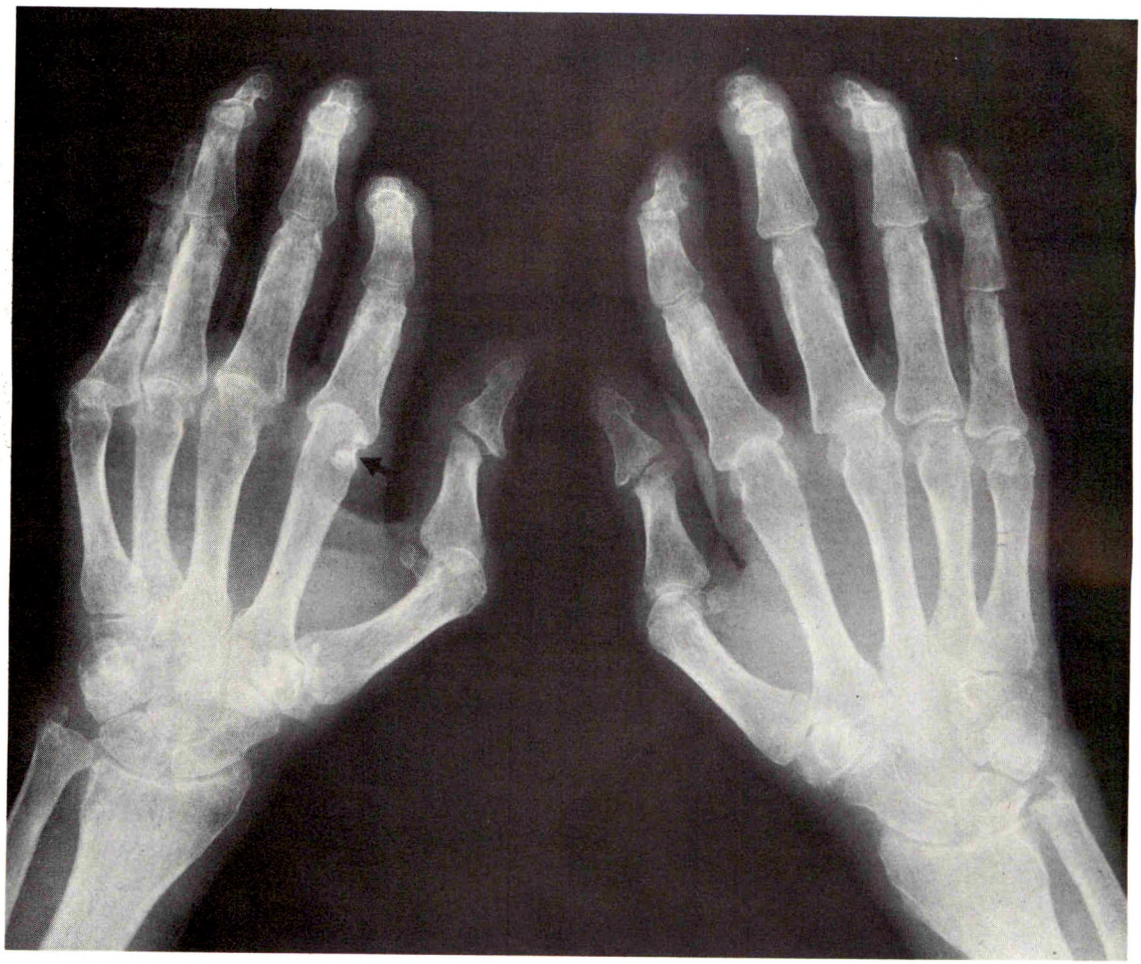


FIG. 2. L.D. Bilateral joint dislocations are present within the metacarpo-phalangeal and interphalangeal joints. The interphalangeal joints of the thumb are sheared and dorsally flexed. Note the alterations of the wrists, particularly on the right. A sharply defined marginal erosion (arrow) is present in the distal 2nd left metacarpal.

garded as early and characteristic manifestations of rheumatoid arthritis.

The radio-carpal joint as well as most joint surfaces in the wrist were narrowed and irregular in both diseases (Fig. 1–6). In 1 patient, ankylosis of one wrist was encountered (Fig. 3). This feature has not been previously described in scleroderma. Widening and splaying of the distal radius were also visualized, with the navicular bone encompassed within the base of the radius (Fig. 2–4). As in rheumatoid arthritis, this alteration may be due to an underlying joint instability with resulting bone contact.[4]

Marginal bone erosions, another roentgenographic feature presently considered pathognomonic for rheumatoid arthritis, was also observed in these 6 patients as well as in other patients with manifestations typical for scleroderma. According to Martel *et al.*[5] the erosions in rheumatoid arthritis occur at those margins where the capsule attaches to bone adjacent to the articular cartilage. The erosions are particularly prominent at sites, the so-called "bare areas," where a portion of the bone within the joint is not covered by articular cartilage but by a reflection of the synovial membrane. Involvement of the carpal

bones occurs in both entities. The navicular bone was frequently affected in our cases along the medial margin, adjacent to the attachment of the radial collateral ligament.

Marginal erosions were also present within the metacarpals and the phalanges. No specific distribution was demonstrated, although a larger series of patients may demonstrate a more definitive pattern. In the metacarpals, erosions were found in both the proximal and distal part of the bone. In 9 patients, erosions were found in the phalanges. The proximal phalanx of the 3rd finger appeared to be the most affected, although other phalanges were almost equally involved. In 1 patient, progression of the disease was witnessed by further development of erosions and soft tissue calcification within a year.



Fig. 3. L.R. Diffuse osteoporosis is visualized with ankylosis of the right carpal bones. Severe deformity is present within radio-carpal and radio-ulnar joints. Dislocation of the metacarpo-phalangeal joints and associated phalangeal deformities are present. Only differentiating feature from rheumatoid arthritis is the soft tissue calcification (arrow) lateral to the ulna.



Fig. 4. S.A. All the distal phalanges are absent. Soft tissue calcification is present in the first digit. These findings are classic for scleroderma. The wrist demonstrates a large erosion in the medial aspect of the radius (arrow).

In 2 patients with scleroderma, the joints of the hands and wrists demonstrated severe malalignment and subluxation (Fig. 2 and 3). The terminal phalanx of the thumb was dorsally displaced and in 1 patient both phalangeal surfaces had a sheared appearance (Fig. 2). Flexion deformities involving the bones at the metacarpo-phalangeal joints were also encountered.

DIFFERENTIAL DIAGNOSIS

In the majority of our cases with scleroderma (79 per cent) the phalangeal changes consisted of bone absorption, skin atrophy, and soft tissue calcification. These features are classic, and their presence alone should suggest the correct diagnosis (Fig. 4). In some cases, however, the joint changes predominate. In these cases the wrist is frequently involved. The interphalangeal joints


A


B

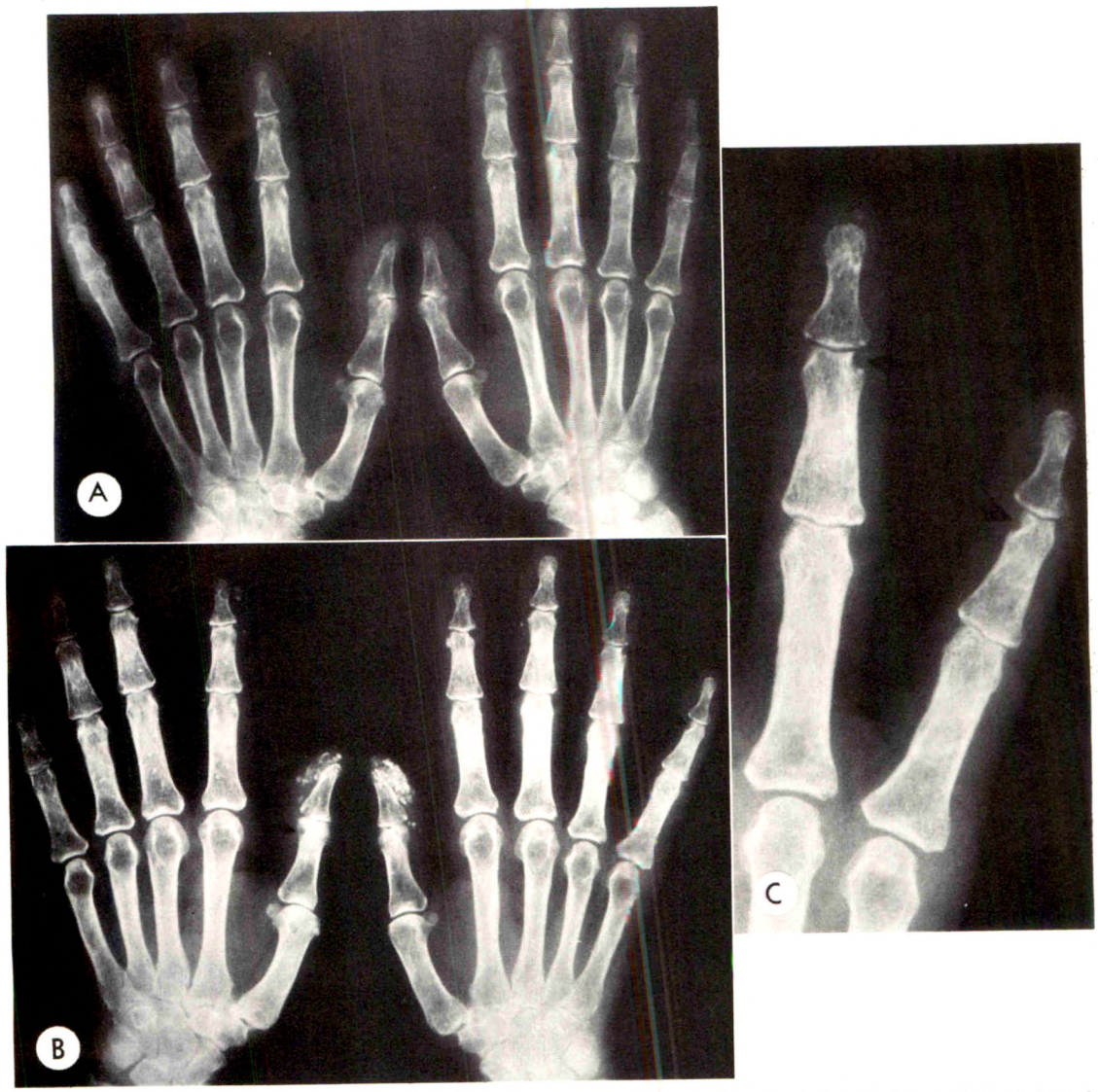


Fig. 6. L.L. (*A*) A single sharply defined erosion is present within the distal middle phalanx of the right 4th digit. The remaining bones appear normal. (*B*) Two years later, additional erosions are now present within the distal middle and proximal phalanges of the right 5th and 1st digits, respectively. Soft tissue calcifications are obvious. (*C*) A detailed view of the 4th and 5th digit demonstrates sharp marginal erosions as described above. Note the small deposit of calcification distal to the terminal phalanx of the 4th digit.

Fig. 5. I.S. (*A*) The wrist demonstrates narrowing of the radio-carpal joint and a sharply defined erosion in the navicular bone. The remainder of the hand appears normal. (*B*) One year later, additional erosions are present in both greater multiangular bones and the distal right 1st metacarpal.

TABLE II

HANDS OF SCLERODERMA PATIENTS RESEMBLING RHEUMATOID ARTHRITIS

Patient	Osteoporosis	Joint Narrowing	Bone Destruction	Bone Erosion	Mutilation	Acro-Osteolysis	Calcium	Skin Atrophy
L.F.	−	1. carpal bones 2. metacarpal phalangeal (1st)	−	+	−	−	+	+
L.R. (*Fig. 3*)	+ generalized	1. all joints 2. fusion carpal bones (unilateral)	+	+	+	?	+ minimal	+
I.S.	−	radio-carpal	−	+	−	−	−	−
V.P.	−	1. radio-carpal 2. carpal 3. carpal-metacarpal 4. metacarpal-phalangeal	−	+	−	−	−	minimal ?
S.A. (*Fig. 4*)	+ juxta-articular and all carpal bones	1. radio-ulnar 2. radio-carpal 3. carpal-metacarpal	−	+	+ with dislocation of multiangular bones	+ all	+	+
L.O.	minimal, diffuse	1. all joints except proximal interphalangeal 2. ulnar styloid	+	+	++	−	minimal	−
L.L.	minimal	carpal-metacarpal	−	+	−	−	+	−

are also affected, but to a lesser degree and in an asymmetric manner.

The pattern of osteoporosis can be used as a differential point since juxta-articular osteoporosis was found in only 2 cases. This form of osteoporosis is seen quite frequently in the early stages of rheumatoid arthritis. However, because of immobility or existing hyperemia, the osteoporosis eventually becomes more or less uniform and is then no longer of diagnostic significance.

The presence of soft tissue calcification is extremely important. Aside from patients receiving excessive vitamin D or high calcium diets, this is presently rarely seen in rheumatoid arthritis.[1] When identified, soft tissue calcification is therefore considered diagnostic for scleroderma and in some patients was the only differential feature (Fig. 3; and 6, *A–C*).

DISCUSSION

The articular complaints in scleroderma as in rheumatoid arthritis are pain, swelling, redness, and warmth. These symptoms similarly undergo remissions and exacerbations. Synovial biopsies performed in these patients with joint involvement, have revealed an inflammatory reaction characterized initially by infiltration of lymphocytes and plasma cells.[7] Later in the course of the disease, fibrosis is produced which can, in some advanced cases, completely encompass and obliterate the vascular structures. The remaining vessels demonstrate thickened walls and narrow lumina.

It appears that the majority of the joint changes demonstrated roentgenographically in progressive systemic sclerosis are related basically to this synovial inflammation and not to the changes occurring in the overlying skin. The synovitis encountered in these patients with joint symptoms is not unlike that encountered in mild cases of rheumatoid arthritis and accounts for the striking clinical and roentgenographic similarity noted in both diseases. The marginal erosions may be attributed to the inflamed

TABLE III

BONE EROSIONS

Patient	Wrist	Carpal Bones	Metacarpal Bones	Phalanges
S.A.	radio-ulnar joint, large	navicular, medial	—	proximal phalanx, dorsal lateral base
I.S. 1 year later (*Fig. 5, A and B*)	radio-ulnar joint, minimal radio-ulnar joint, minimal	navicular, medial greater multiangular, bilateral		mid-phalanx, subperiosteal erosion—5th digit proximal phalanx, base 2nd, 3rd, 4th digit proximal phalanx, distal
V.P.	radio-ulnar joint, minimal	triquetrum	distal	proximal phalanx, lateral base
L.O.	1. radio-ulnar joint 2. ulnar styloid process	—	—	—
L.F.	radio-ulnar joint	navicular, lunate	—	—
L.R.	radio-ulnar joint	—	proximal	—
L.L. (*Fig. 6, A–C*)	—	capitate	—	mid-phalanx, distal 2nd, 3rd, 4th, 5th digit, proximal phalanx, thumb
S.S.	—	—	—	mid-phalanx 3rd digit, base mid-phalanx 5th digit, distal
R.W.	—	—	—	mid-phalanx distal
L.V.	—	—	—	mid-phalanx 3rd digit, distal
S.C.		navicular, medial		proximal phalanx, thumb, distal proximal phalanx, distal
R.W.	—	—	—	mid-phalanx distal
L.V.	—	—	—	mid-phalanx, distal
S.C.		navicular, medial		proximal phalanx, thumb, distal proximal phalanx, distal

hyperplastic synovial tissue that covers the "bare areas." Erosions have also been described in other bones; *e.g.*, along the superior aspect of the posterior ribs.[3] The changes occurring along the joint surface are probably similarly caused by the inflammatory process destroying the articular cartilage. However, the absence of a well developed pannus in scleroderma explains why only minimal bone destruction and little ankylosis is encountered. In 1 patient this developed sufficiently in one wrist to cause bone ankylosis. Clinically, this patient did not manifest an overt clinical picture of rheumatoid arthritis.

As in rheumatoid arthritis, it appears that muscular forces acting on demineralized bone may also play an important role in producing bone destruction. This factor may account for the subluxation occurring at the metacarpo-phalangeal joints, the sheared bone ends of the phalanges, the splayed ends of bones and the relatively severe deformities.

Since bone reacts to insult in only a limited amount of ways and since some of the pathologic processes in both diseases are similar, it is not surprising to see many of the osseous features previously considered pathognomonic for rheumatoid arthritis also present in scleroderma. Whether both diseases were concomitantly present is still open to question, although this was clinically excluded in our patients. Despite the fact that many overlapping clinical and roentgenographic manifestations do exist, the over-all evolution of both diseases still remains distinct.

SUMMARY

The hands and wrists of 24 patients with scleroderma were reviewed.

Although, in the majority of cases, the phalangeal alterations of scleroderma were quite typical, many features similar to those encountered in rheumatoid arthritis were noted; *e.g.*, bone erosions, narrowing of the joints, alteration of the ulnar styloid process, subluxations, and even ankylosis of the wrist. Many of these overlapping features were originally considered pathognomonic for rheumatoid arthritis.

The presence of synovitis in scleroderma, similar to that encountered in early rheumatoid arthritis, probably accounts for many of these manifestations.

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REFERENCES

1. DALINKA, M. K., and WUNDER, J. F. Unusual manifestations of rheumatoid arthritis. *Radiology*, 1970, *97*, 393–395.
2. GONDOS, B. Roentgen manifestations in progressive systemic sclerosis (diffuse scleroderma). AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *84*, 235–247.
3. KEATS, T. E. Rib erosions in scleroderma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *100*, 530–532.
4. MARTEL, W. Pattern of rheumatoid arthritis in hand and wrist. *Radiol. Clin. North America*, 1964, *2*, 221–234.
5. MARTEL, W., HAYES, J. T., and DUFF, I. F. Pattern of bone erosion in hand and wrist in rheumatoid arthritis. *Radiology*, 1965, *84*, 204–213.
6. MESZAROS, W. T. Regional manifestations of scleroderma. *Radiology*, 1958, *70*, 313–325.
7. RODMAN, G. P. Nature of joint involvement in progressive systemic sclerosis: diffuse scleroderma. *Ann. Int. Med.*, 1962, *56*, 422–439.
8. SCHACHERL, M., and HOLZMANN, H. Zur Polyarthritis bei Progressive Sclerodermie. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1967, *107*, 485–493.

HOMOCYSTINURIA DUE TO CYSTATHIONINE SYNTHASE DEFICIENCY: CLINICAL-ROENTGENOLOGIC CORRELATIONS*

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HOMOCYSTINURIA comprises a group of inborn errors of methionine metabolism. There are 3 different enzymatic deficiencies, at 3 gene loci, identified to date: cystathionine synthase; methyltetrahydrofolatehomocysteine methyltransferase; and methylenetetrahydrofolate reductase[4,17] (Fig. 1). The most common and best studied is the deficiency of cystathionine synthase, which is inherited on an autosomal recessive basis. There is evidence that even at this locus, there are multiple alleles resulting in at least 2 genetically heterogeneous diseases; *i.e.*, pyridoxine-responsive and pyridoxine-resistant cystathionine synthase deficiencies. Clinical and biochemical aspects have been thoroughly reviewed.[1,3–5,9,11,14]

Most clinical and roentgen information is available concerning cystathionine synthase deficiency, but, even within this enzymatic type, the wide variation in both clinical and roentgen manifestations is striking. The main clinical features of homocystinuria due to cystathionine synthase deficiency include: mental deficiency; ectopia lentis; thromboembolic phenomena; and skeletal abnormalities. It has been found that vitamin B_6 administered orally in massive doses results in a reduction in the amount of methionine and homocystine in the plasma and urine and an increase in the amount of cystine. This is associated with an increase in the activity of cystathionine synthase in some patients.

Roentgen features of homocystinuria have been described by Morreels *et al.*,[10] Smith,[16] and Schedewie and colleagues.[13] Their patients were diagnosed by demonstrating the presence of homocystine in the urine and only in some instances was a specific enzymatic diagnosis made from liver or skin biopsies.

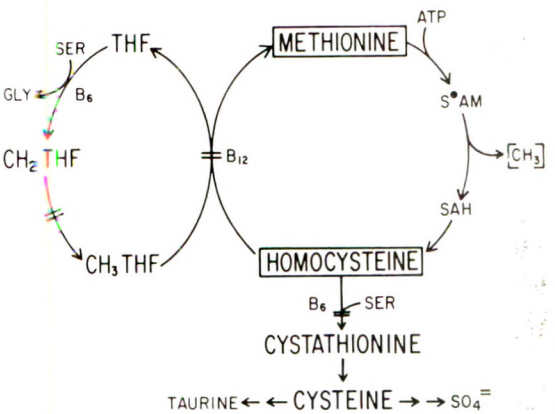


FIG. 1. Metabolism of methionine showing: block in conversion of homocysteine and serine (ser) to cystathionine (cystathionine synthase); block in transmethylation of ^{5}N-methyltetrahydrofolate (CH_3THF) with homocysteine to form methionine (methyltetrahydrofolate-homocysteine methyltransferase); block in reduction of ^{5,10}N-methylenetetrahydrofolate (CH_2THF) to CH_3THF (methylenetetrahydrofolate reductase). Abbreviations: $S^{(+)}AM$ = S-adenosylmethionine; SAH = S-adenosylhomocysteine; B_6 = pyridoxal phosphate; B_{12} = methylcobalamin; gly = glycine.

We have studied the roentgen features of 10 patients with homocystinuria, in each of whom cystathionine synthase deficiency was established. Correlation was sought between the roentgen, enzymatic, and clinical severity of the disease.

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TABLE I

SUMMARY OF THE CLINICAL FINDINGS IN 10 CASES OF CYSTATHIONINE SYNTHASE DEFICIENCY

	Adults	Children
Mental deficiency	2/4	3/6
Psychiatric or behavior disorder	2/4	2/6
Seizures	1/4	1/6
Ectopia lentis	3/4	4/6
Thromboembolic disease	4/4	0/6
Marfan-like appearance	1/4	0/6
Genu valgum	2/4	4/6
B_6-responsive	2/4	2/6

MATERIAL AND METHOD

This study was comprised of 10 patients (6 children under 10 years of age and 4 adults) with homocystinuria, all of whom were found to have cystathionine synthase deficiency on the basis of enzymatic analysis of liver obtained by percutaneous puncture.[6,7] They were referred from various sources and were not identified on the basis of screening from any 1 source.

Roentgenographic evaluation included the following in all patients: chest, skull, complete survey of the appendicular and axial skeleton, metacarpal index, bone age and excretory urography (with accompanying superior venacavagraphy in 8 patients).

Because it is known that this is a progressive disease,[3,9] apparently with no distinguishing roentgen features at birth, the children and adults were considered separately. Specific attention was directed to identification of the roentgen findings occurring early in the life of these patients before the disease was clinically obvious.

RESULTS

Of the 4 adults, 2 were responsive and 2 unresponsive to massive doses of vitamin B_6 (Table 1). There is some evidence to suggest that 1 of the responsive adults is actually a severely affected heterozygote. Two of 4 children were siblings who were completely responsive to vitamin B_6; *i.e.*, on an *ad libitum* diet the plasma methionine decreased to normal concentrations, the homocystine disappeared, and the cystine increased to normal concentrations. Two other children were also siblings and were only partially responsive; *i.e.*, on massive doses of pyridoxine with some limitation of protein intake the methionine and homocystine decreased but the cystine did not appear. The third set of siblings was unresponsive to pyridoxine.

As expected in a progressive disease, the children were less involved clinically and roentgenographically than the adults (Table 11). In general, those patients with

TABLE II

INCIDENCE OF POSITIVE ROENTGENOGRAPHIC FINDINGS IN 10 PATIENTS WITH HOMOCYSTINURIA DUE TO CYSTATHIONINE SYNTHASE DEFICIENCY

	Adults	Children
Spine		
Biconcave vertebral bodies	2/4	2/6
Narrow upper thoracic disks	2/4	0/6
Osteoporosis	1/4	2/6
Scoliosis	3/4	1/6
Chest		
Pectus excavatum	2/4	0/6
Calcification in superior vena cava	1/4	0/6
Skull and Face		
Dural calcification	2/4	0/6
Wide diploic space	1/4	0/6
Prognathism	3/4	0/6
Long Bones		
Prominent growth arrest lines	2/4	5/6
Osteoporosis	1/4	1/6
Humerus varus	2/4	0/6
Bowed radius and ulna	2/4	0/6
Hemiatrophy	1/4	0/6
Hands and Wrists		
Metacarpal index >8.5	1/4	1/6
Metaphyseal spicules	0/4	5/6
Bone age	Adult	Within 1 year of chronologic age
Large capitate and hamate	1/4	1/6
Feet		
Long talus	2/4	4/6
Excretory Urogram		
One kidney not visualized	1/4	0/6
Dense papillary blush	2/4	0/6

TABLE III

SUMMARY OF ENZYMATIC, CLINICAL AND ROENTGEN DATA IN 10 PATIENTS WITH CYSTATHIONINE SYNTHASE DEFICIENCY

Patient	Age	Synthase Level (Per Cent of Normal) Response to Vitamin B_6	Main Clinical Features	Main Roentgen Features
C.H.	21 yr.	Synthase 15% B_6 responsive	Hemiparesis Thrombophlebitis Minor motor seizures Violent behavior	Hemiatrophy Prognathism Left kidney not visualized Scoliosis*
R.N.	21 yr.	Synthase 4% B_6 responsive	Slight mental deficiency Psychiatric disorder "Stroke" Lens dislocation	Biconcave vertebral bodies Narrow upper thoracic disks Humerus varus Bowed radius and ulna Prognathism Long talus IVP: Dense papillary blush
L.M.	25 yr.	Synthase 0% B_6 unresponsive	Mental deficiency Thrombophlebitis Lens dislocation	Scoliosis Narrow upper thoracic disks Prominent growth arrest lines Calcified thrombus in superior vena cava Pectus excavatum Dural calcification Wide diploic space
M.S.	24 yr.	Synthase 0% B_6 unresponsive	Marfan-like appearance Lens dislocation Detached retina Thrombosis (testis) Learning disability	Biconcave vertebral bodies Osteoporosis Scoliosis Prominent growth arrest lines Humerus varus Bowed radius and ulna Pectus excavatum Prognathism Dural calcification Arachnodactyly (metacarpal index = 8.95) Large capitate and hamate Long talus IVP: Dense papillary blush
A.G.	5 yr.	Synthase 5% B_6 responsive	Normal clinically (Sister of J.G.)	Growth arrest lines
J.G.	9 yr.	Synthase 5% B_6 responsive	Mental deficiency Behavior problem Lens dislocation (Brother of A.G.)	Biconcave vertebral bodies Growth arrest lines Arachnodactyly (metacarpal index = 8.85)
R.C.	5 yr.	Synthase 3% B_6 partially responsive	Hyperactive behavior Myopia	Metaphyseal spicules Large capitate and hamate

* This 21 year old male had had a cerebral vascular accident in the neonatal period, resulting in left-sided hemiplegia. The scoliosis may have been secondary to the resultant hemiatrophy.

IVP = intravenous pyelography.

TABLE III—(*Continued*)

Patient	Age	Synthase Level (Per Cent of Normal) Response to Vitamin B_6	Main Clinical Features	Main Roentgen Features
			Treated with vitamin B_6 since age 12 months (Brother of M.C.)	Long talus
M.C.	7 yr.	Synthase 0% B_6 partially responsive	Mental deficiency Hyperactive behavior Seizures Lens dislocation Retinal detachment (Sister of R.C.)	Biconcave vertebral bodies Osteoporosis (spine) Growth arrest lines Metaphyseal spicules Long talus
J.O'R.	7 yr.	Synthase 0% B_6 unresponsive	Mental deficiency Lens dislocation (Brother of A.O'R.)	Growth arrest lines Metaphyseal spicules Long talus
A.O'R.	9 yr.	Synthase 0% B_6 unresponsive	Lens dislocation (Sister of J.O'R.)	Scoliosis Osteoporosis Metaphyseal spicules Growth arrest lines Long talus Metacarpal index = 8.5

the most severe enzymatic deficiency[6,7] had the greatest number of major roentgenographic findings, but the particular type of finding was variable (Table III).

In 2 adults with similar severe biochemical defects (0 synthase, no response to vitamin B_6), widely different roentgen and clinical findings were present (Table III). One (M.S.) showed severe skeletal involvement (Fig. 2, *A–D*) and had had 2 episodes of orchitis, thought to be thromboembolic. The other (L.M.) had a calcified thrombus obstructing the superior vena cava.[12] His skeletal involvement was limited to scoliosis, growth arrest lines in the distal tibia and a wide calvarial diploic space (Fig. 3, *A–E*). A considerable amount of dural calcification was noted on his skull roentgenograms. The 2 adults who were responsive to vitamin B_6 had fewer major roentgen findings than the 2 who were unresponsive. The adult with residual synthase activity of 4 per cent of normal (R.N.) had several minor skeletal changes including biconcave vertebral bodies, humerus varus, prognathism and an elongated talus (Fig. 4, *A–C*). The adult (C.H.) with the mildest disease biochemically (15 per cent residual synthase) had no skeletal changes, except hemiatrophy, which was related to a cerebral vascular accident in early infancy, as well as prognathism.

The metacarpal index[15] was determined in all patients as an objective measure of arachnodactyly. One adult and 1 child had metacarpal indices greater than 8.5, evidence of arachnodactyly.

The most frequently occurring findings in the adults and children together were prominent growth arrest lines in the distal tibia and elongation of the talus (Fig. 2*A*; Table II). In the adults, changes in the spine and prognathism were usually present (Fig. 4, *A* and *C*; Table II). In children, the occurrence of one or more calcific spicules in the cartilaginous growth plate at the distal radius or ulna was noted most frequently. In each case a dense longitudinal streak was seen extending into the shaft just proximal to the calcific spicules (Fig.

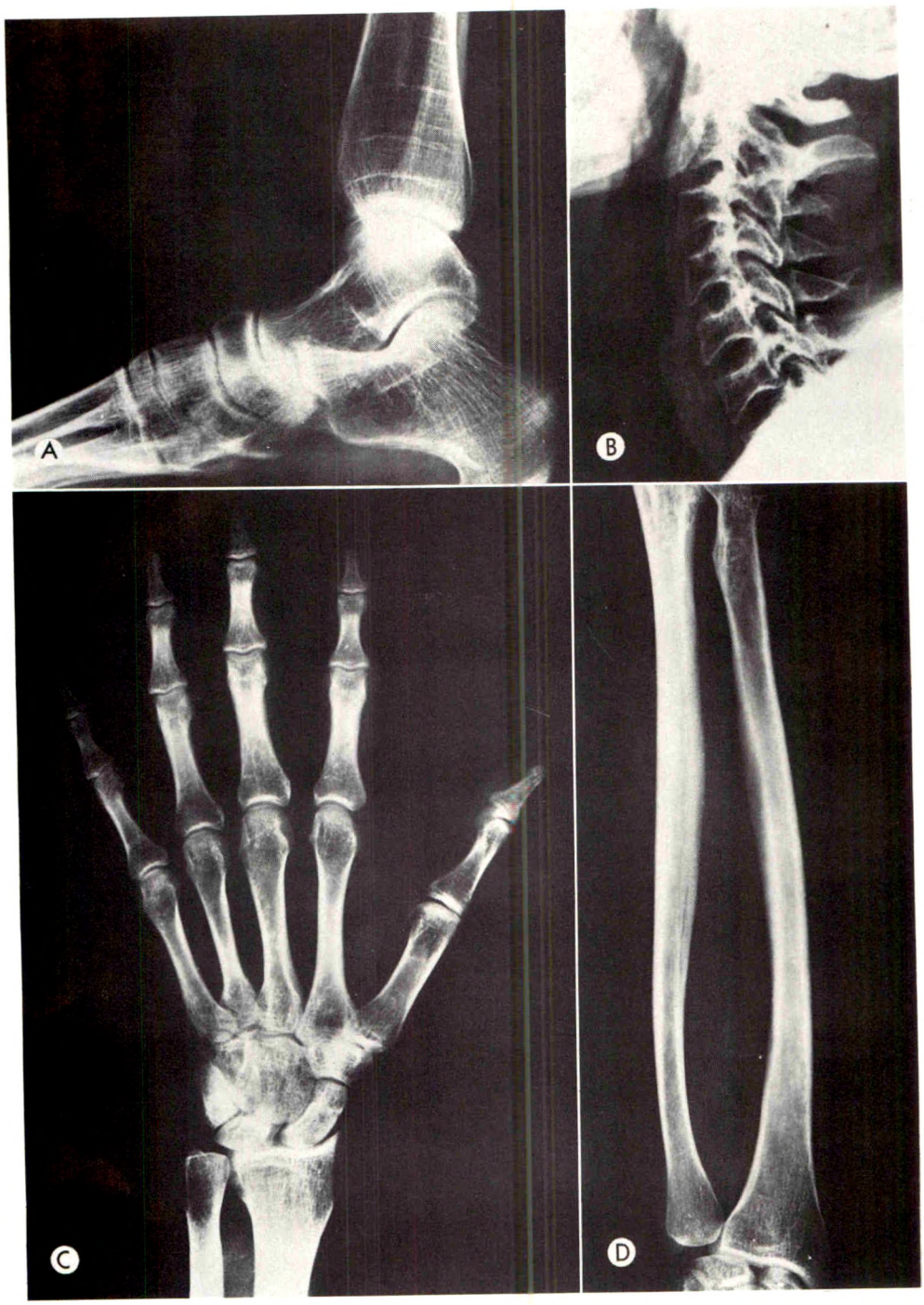


Fig. 2. Roentgenograms of a 24 year old man (M.S.) with severe enzymatic deficiency, unresponsive to pyridoxine, with extensive skeletal involvement.

(*A*) Lateral view of the foot and ankle shows osteoporosis, multiple growth arrest lines, and elongation of the talus. (*B*) Lateral view of the cervical spine shows biconcave vertebral bodies and osteoporosis. (*C*) Osteoporosis, enlargement of the capitate and hamate, and arachnodactyly are noted. (*D*) There is bowing of the distal radius and ulna.

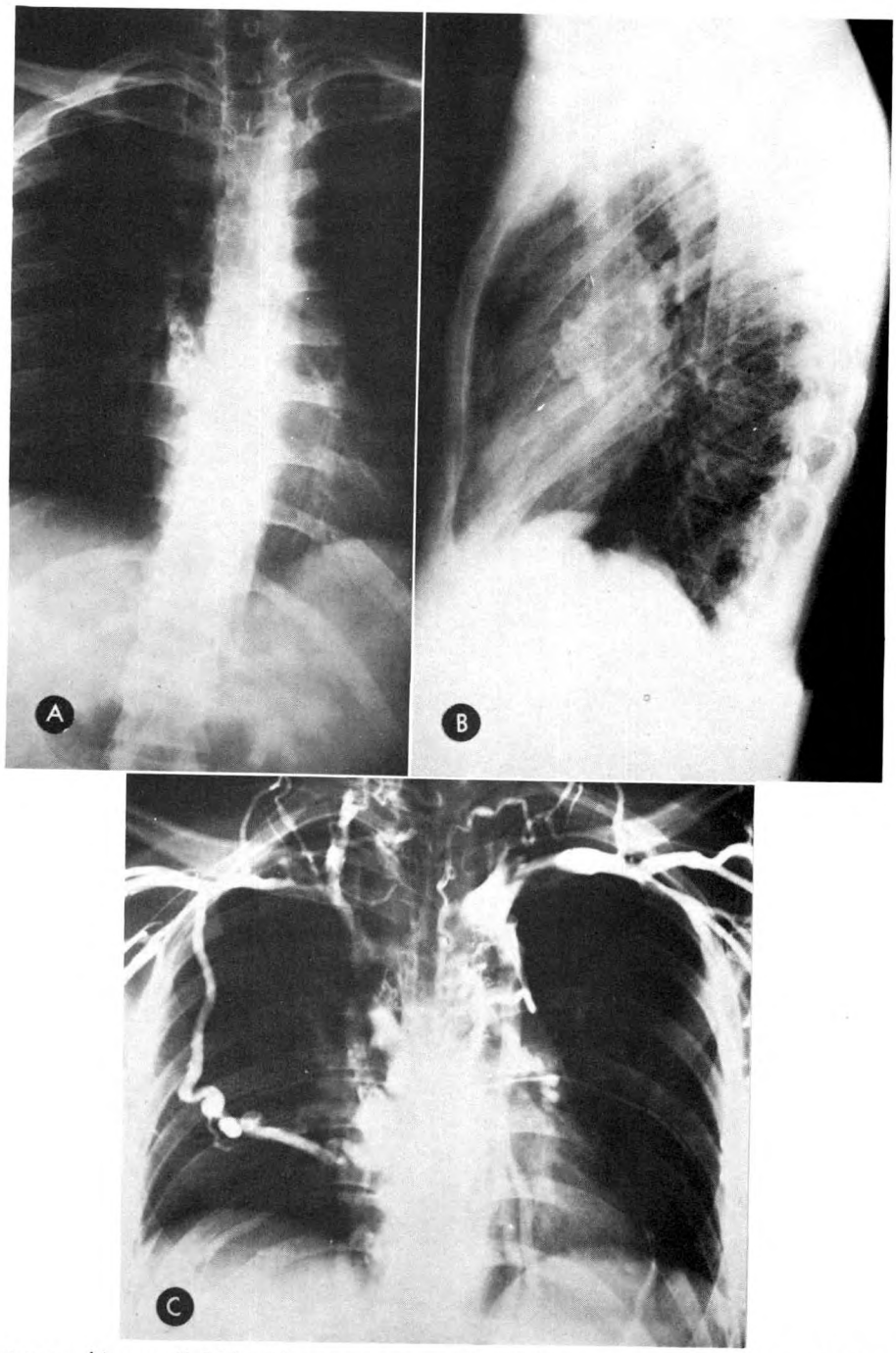


FIG. 3. A 25 year old man (L.M.) with severe enzymatic deficiency, unresponsive to pyridoxine, with extensive thromboembolic disease and limited skeletal involvement.

(*A*) Superior vena cava calcification and scoliosis are seen. (*B*) The extent of the calcified thrombus in the superior vena cava is best appreciated in the lateral view. (*C*) Bilateral injection of antecubital veins with Renografin 76 shows complete obstruction of the superior vena cava by the calcified thrombus. Numerous large collateral veins are demonstrated.

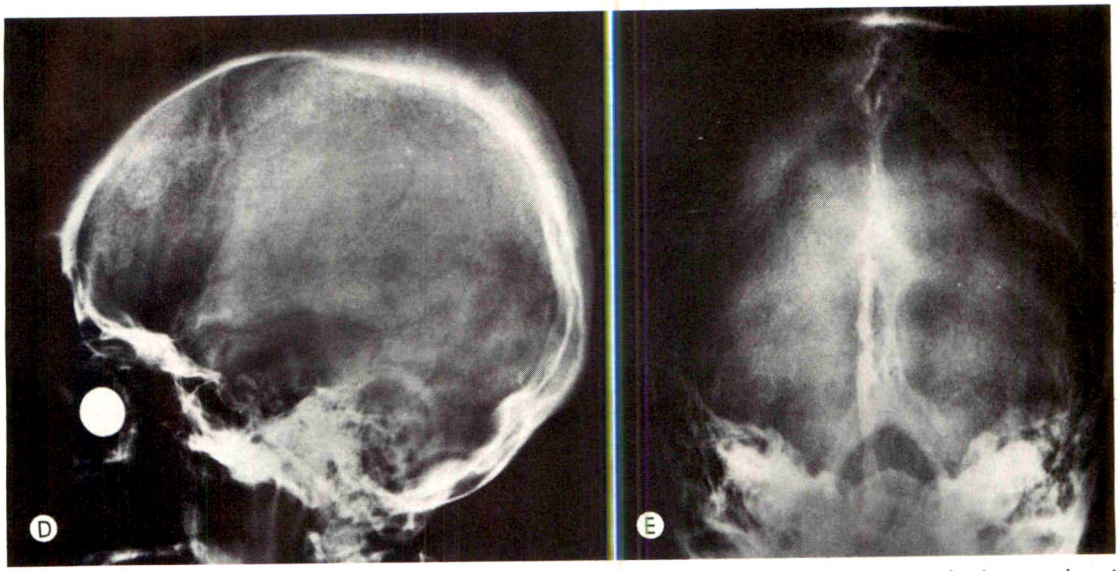


Fig. 3. (*D*) The diploic space is wide. Dural calcification is noted anteriorly. An eye prosthesis was placed following an enucleation performed because of acute glaucoma. (*E*) Dural calcification is best seen in the Towne's view of the skull.

5). A similar finding may be present in phenylketonuria.

On excretory urography, no abnormalities were found in any of the children. The left kidney was not visualized in the adult with the mildest enzymatic deficiency. A dense papillary blush was seen bilaterally in 2 adults, 1 responsive and 1 unrespon the to vitamin B_6 (Fig. 6). In the course ofsive injection for excretory urography, roentgenograms of the chest were obtained in all the adults and 4 of the children. There were no cases of superior vena cava obstruction except for the one suspected clinically and on the basis of calcification noted on chest roentgenograms.

DISCUSSION

The ocular, skeletal, and vascular changes in homocystinuria may simulate Marfan's disease, but thromboembolic phenomena and osteoporosis have been considered to be distinguishing clinical features of homocystinuria.[14] In our patients, however, osteoporosis was not as prominent as described by previous authors.[10,13,16] It is likely that this is related to the young age of our patients (children under 10 years and adults in their 20s), and to the fact that they were referred from a variety of sources without emphasis on any particular clinical feature which might correlate with bone changes.[2,8]

Thromboembolic phenomena were a feature in all of the adults but in none of the children. The adults, however, had probable evidence of thrombotic episodes during childhood.

If vascular injections are necessary in patients with homocystinuria, the use of catheters is to be avoided because of the danger of initiating thrombosis.[10] The fact that thrombi are known to occur in both arteries and veins suggests a hypercoagulable state, but the exact etiology is uncertain.[3]

Gaull *et al.*[6] and Uhlendorf *et al.*[17] have recently demonstrated that patients with cystathionine synthase deficiency who respond to massive doses of vitamin B_6 in the form of pyridoxine have the largest amount of residual cystathionine synthase activity. Virtually no residual activity is present in those who are unresponsive to pyridoxine. In our patients, the severity of the enzymatic deficiency and its response to vitamin B_6 correlated quantitatively, but not qualitatively with roentgenographic in-

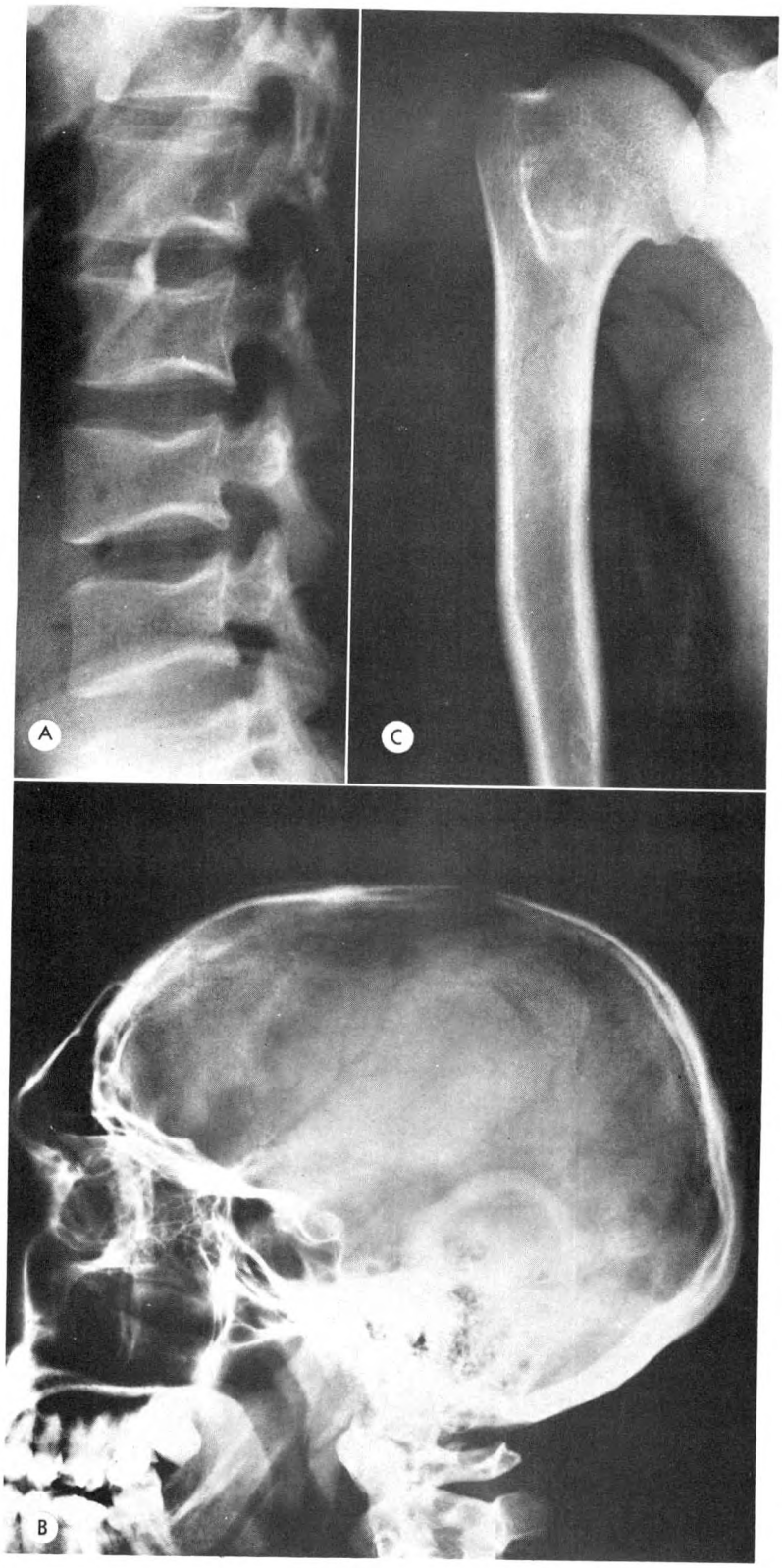

A
C
B



Fig. 5. A dense longitudinal streak extends into the ulnar shaft from a metaphyseal calcific spicule.

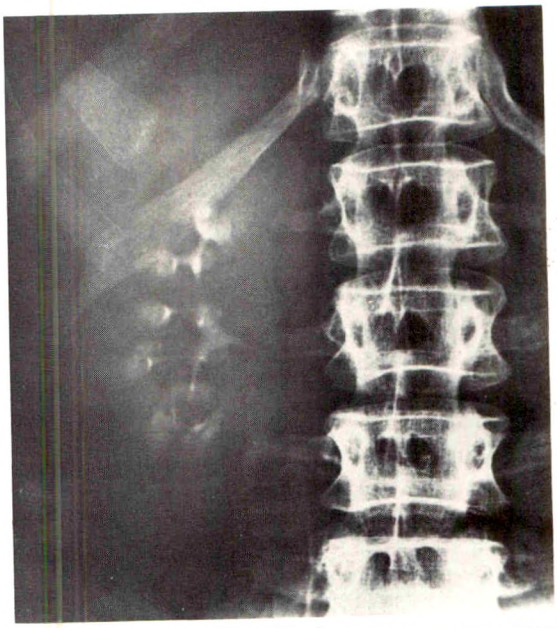


Fig. 6. A dense papillary blush was seen on excretory urography in 2 adults.

volvement. This is not surprising in view of the now recognized genetic heterogeneity of this disease.[6,17] One clinical feature with an interesting relation to the roentgenologic findings was ectopia lentis. The 3 patients (Table III; C.H., A.G. and R.C.) who have normal lenses have very little skeletal involvement. Two of these (A.G. and R.C.) are children, however, and it will be of interest to see whether this status is maintained.

The adult whose skeletal disease is the mildest (C.H.), is suspected on clinical and enzymatic grounds of being a heterozygote.[6] He has no skeletal involvement except for a prognathic jaw and hemiatrophy, the latter attributable to an acute infantile hemiplegia.

One of the children (R.C.) is unusual in that he has been treated with vitamin B_6 since the age of 12 months. He is now 5 years old and is the only one of the 6 children without prominent growth-arrest lines.

A variety of renal abnormalities have been mentioned in homocystinuria.[3,9,10] The dense papillary blush, seen in 2 of our adult patients, may be part of the spectrum of medullary sponge kidney. Morreels *et al.*[10] noted 1 patient with homocystinuria who had medullary sponge kidney.

Fig. 4. A 21 year old man (R.N.) with some residual synthase activity, responsive to pyridoxine, has mild skeletal involvement.

(*A*) This man has biconcave vertebral bodies without osteoporosis. Renal contrast material is noted secondary to an intercurrent excretory urography. (*B*) The jaw is prognathic and the frontal sinuses are large. (*C*) There is a varus deformity of the humeral head. The cortical thickness of the humeral shaft is well maintained.

CONCLUSIONS AND SUMMARY

The roentgenologic findings have been documented in 10 patients with enzymatically diagnosed deficiency of cystathionine synthase. Patients responsive to massive doses of pyridoxine have the largest amount of residual cystathionine synthase activity. Those who are unresponsive show virtually no residual activity. The pyridoxine-responsive patients show the mildest and fewest roentgenologic findings. Similarly, those patients unresponsive to pyridoxine are the most severely affected roentgenologically, although the type of involvement is variable.

Calcific spicules and streaks in the radius or ulna may be diagnostically useful as an early finding in children. In young adults with thromboembolic disease, the presence of one or more of the diverse skeletal changes may suggest the diagnosis of homocystinuria.

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REFERENCES

1. BRENTON, D. P., CUSWORTH, D. C., DENT, C. E., and JONES, E. E. Homocystinuria: clinical and dietary studies. *Quart. J. Med.*, 1966, *35*, 325–346.
2. BRETT, E. M. Homocystinuria with epilepsy. *Proc. Roy. Soc. Med.*, 1966, *59*, 484–487.
3. CARSON, N. A. J., DENT, C. E., FIELD, C. M. B., and GAULL, G. E. Homocystinuria: clinical and pathological review of ten cases. *J. Pediat.*, 1965, *66*, 565–583.
4. CARSON, N. A. J., and RAINE, D. N. Inherited Disorders of Sulphur Metabolism. J. A. Churchill, London, 1971.
5. GAULL, G. E. Homocystinuria, vitamin B_6, and folate: metabolic interrelationships and clinical significance. *J. Pediat.*, 1972, *81*, 1014–1018.
6. GAULL, G. E., STURMAN, J. A., and SHAFFNER, F. Homocystinuria due to cystathionine synthase deficiency: enzymatic and ultrastructural studies. To be published.
7. GAULL, G. E., STURMAN, J. A., and RASSIN, D. K. Enzymatic and metabolic studies of homocystinuria: effects of pyridoxine. *Neuropadiatrie*, 1969, *1*, 199–226.
8. MACCARTHY, J. M. T., and CAREY, M. C. Bone changes in homocystinuria. *Clin. Radiol.*, 1968, *19*, 128–134.
9. MCKUSICK, V. A. Heritable Disorders of Connective Tissue. Fourth edition. C. V. Mosby Company, St. Louis, 1972, pp. 224–281.
10. MORREELS, C. L., FLETCHER, B. D., WEILBAECHER, R. G., and DORST, J. P. Roentgenographic features of homocystinuria. *Radiology*, 1968, *90*, 1150–1158.
11. MORROW, G., and BARNESS, L. A. Combined vitamin responsiveness in homocystinuria. *J. Pediat.*, 1972, *81*, 946–954.
12. OKAY, N. H., and BRYK, D. Collateral pathways in occlusion of superior vena cava and its tributaries. *Radiology*, 1969, *92*, 1493–1498.
13. SCHEDEWIE, H., WILLICH, E., GROBE, H., SCHMIDT, H., and MULLER, K. M. Skeletal findings in homocystinuria: collaborative study. *Pediat. Radiol.*, 1973, *1*, 12–23.
14. SCHIMKE, R. N., MCKUSICK, V. A., HUANG, T., and POLLACK, A. D. Homocystinuria: studies of 20 families with 38 affected members. *J.A.M.A.*, 1965, *193*, 711–719.
15. SINCLAIR, R. J. G., KITCHEN, A. H., and TURNER, R. W. D. Marfan syndrome. *Quart. J. Med.*, 1960, *29*, 19–46.
16. SMITH, W. S. Roentgen findings in homocystinuria. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *100*, 147–154.
17. UHLENDORF, B. W., CONERLY, E. B., and MUDD, S. H. Homocystinuria: studies in tissue culture. *Pediat. Res.*, 1973, *7*, 645–658.

COMPLICATIONS OF TOTAL HIP REPLACEMENT*

A ROENTGEN EVALUATION

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SURGERY for implantation of a total hip prosthesis is becoming more common in the United States following a somewhat earlier lead in Europe. Charnley in the early 1960's introduced the concept of low-friction arthroplasty. "In this procedure, both the acetabulum and the femoral head are simultaneously replaced by a prefabricated joint. The plastic socket is made of a high-density polyethylene and a femoral head of a relatively small diameter is considered essential to the design. Even more essential is the use of self-curing acrylic* cement to bond the implant to the bone of the acetabulum and femur."[7]

Several modifications of Charnley's technique have now come into widespread use. As the use of these prostheses becomes more common in the population, it behooves the radiologist to become more knowledgeable in their radiologic evaluation. This involves no new principles, only familiarity with problems unique to the design and implantation of these devices.

Numerous devices are available, each with its advocates. Arguments for and against make interesting reading for the orthopedically inclined.[5] The radiographic appearance of various devices used in total hip replacement has been succinctly reviewed by Angell and Watts.[2]

At the University of Missouri Medical Center the device implanted is the Bechtol total hip prosthesis, similar in design to that of Charnley and the Müller (Fig. 1).

While various complications have been associated with total hip replacement, not all of these complications lend themselves to roentgen evaluation.



FIG. 1. Normal appearance of the Bechtol prosthesis. Note the radiopaque marker ring on the outer margin of the polyethylene acetabular component. The x-ray beam is centered over the mid-pelvis.

The purpose of this communication is to describe the roentgenographic appearance of the most frequently encountered complications. Since November 1971 the following complications have been recorded at

* The acrylic cement (referred to as methyl methacrylate) is made radiopaque by the incorporation of barium sulfate at the time the prosthesis is implanted.

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this institution: (1) dislocation; (2) disruption of the transplanted greater trochanter; (3) periarticular calcification and ossification or both; (4) protrusion of the acetabulum; (5) infection; (6) penetration of lateral femoral cortex; and (7) suboptimal acetabulum orientation.

DISLOCATION

Dislocation occurs most commonly in the immediate postoperative period. In fact, it is most likely to occur during recovery from anesthesia or within the first few days after surgery.[6] This phenomenon is presumably due to laxity of the periarticular musculature and improper postsurgical positioning allowing excessive external rotation of the leg. If no error in the location of the acetabular prosthesis is present, reduction of the dislocation usually suffices. Late and recurrent dislocation is presumably related to improper fitting of the prosthesis, usually excessive anteversion of the plane of the mouth of the socket or of the axis of the neck of the femoral component.[4] Posterior dislocations are usually associated with trochanteric detachment.[5] Deffner *et al.*[6] have applied the term "Saturn Ring" sign to the appearance of the dislocated Charnley-Müller prosthesis. When in place the radiopaque wire embedded in the cup of the prosthesis gives the appearance of Saturn's rings equidistant from the planet or femoral head. Dislocation of the prosthesis is usually grossly evident (Fig. 2). Charnley[4] has recently reported a low incidence (1.5 per cent) of posterior dislocation which he attributes to avoidance of anteversion of the socket and reattachment of the trochanter in such a way as to shorten capsular structures after reduction of the femoral head.

DISRUPTION OF THE TRANSPLANTED TROCHANTER

Total hip replacement is really a reconstruction of the entire hip joint rather than simple replacement. Because of this it is desirable to rearrange the lever ratios in the hip joint in order to provide a more favorable load on the cement bond to the acetabulum. One facet of rearranging the lever ratios is the reattachment of the trochanter more laterally than normal by means of wire sutures.[4] Radiographic evidence of broken wires (Fig. 3) is not in and of itself indicative of serious problems. Wire breakage is usually a metal fatigue phenomenon from repeated bending and need not be associated with avulsion of the trochanter.[1] While considered undesirable, fibrous union of the transplanted trochanter to the femur still allows abduction of the leg if migration of the transplant posteromedially has not occurred. Avulsion of the trochanter on the other hand disrupts the abduction mechanism and constitutes a serious problem requiring reattachment.

PERIARTICULAR CALCIFICATION AND OSSIFICATION

Periarticular calcification and ossification while included in the list of complica-

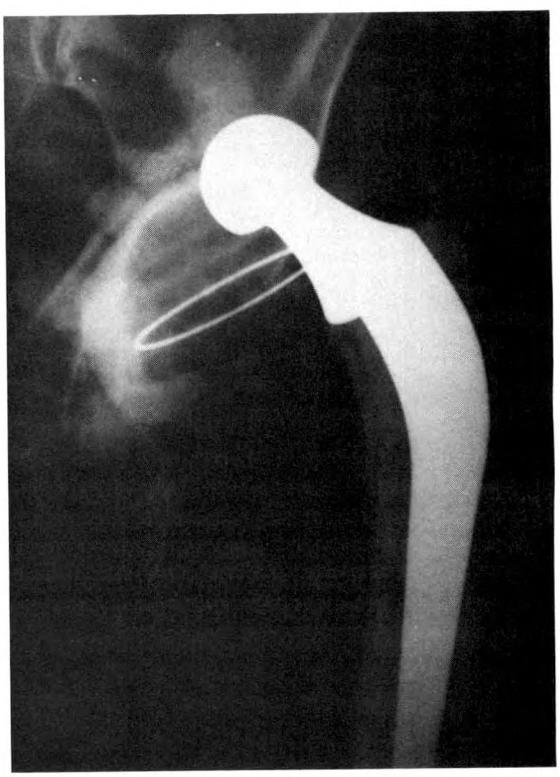


FIG. 2. Posterior dislocation of Bechtol prosthesis. Note relationship of metallic femoral head to radiopaque marker ring.

tions does not prejudice the success of total hip replacement barring excessive compromise of the range of motion of the femur. The cause of ectopic bone formation (Fig. 4) is not known, although there is an increased incidence of its occurrence following dislocation or reoperation.[1]

PROTRUSION OF THE ACETABULUM

Protrusion of the acetabulum is usually grossly evident roentgenographically (Fig. 5). It is more commonly seen in total hip replacement for rheumatoid arthritis and usually follows excessive use of the reamer in the soft rheumatoid bone of the acetabulum.[3]

INFECTION

Next to pulmonary embolism, postoperative infection is regarded by some as the most serious complication of total hip replacement. Even with scrupulous attention to asepsis and good surgical technique, Charnley reports an infection rate of 3.8 per cent.[3] A much higher rate is found in cases wherein multiple procedures have been attempted as compared to the rate in those cases where total hip replacement was performed *de novo*.[7,10] Obesity, rheumatoid arthritis, and steroid therapy have also been identified as predisposing factors which are associated with an increased incidence of postoperative infection.[7] While well-known radiographic signs of infection such as mottling of bone, bone resorption and periosteal reaction may be associated with clinical evidence of infection, these signs may also appear in the absence of clinical infection. In addition, there are documented cases of infection which exhibit no radiographic findings.[9] Infection may also be associated with loosening of the prosthesis.[1,10] Figure 6 illustrates the radiographic findings in a case of early postoperative infection; *i.e.*, clinical signs apparent 1 month after surgery. A thin lucent

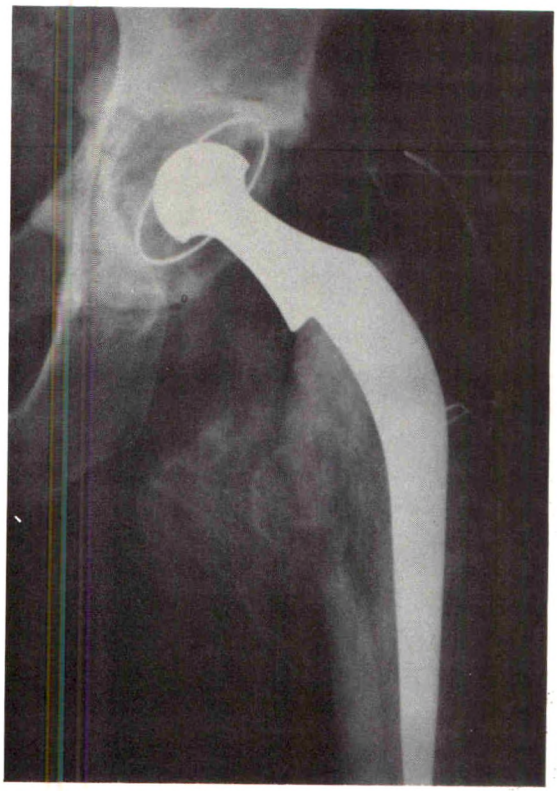


Fig. 4. Extensive periarticular ossification anteromedially. There was no limitation of motion in this case.

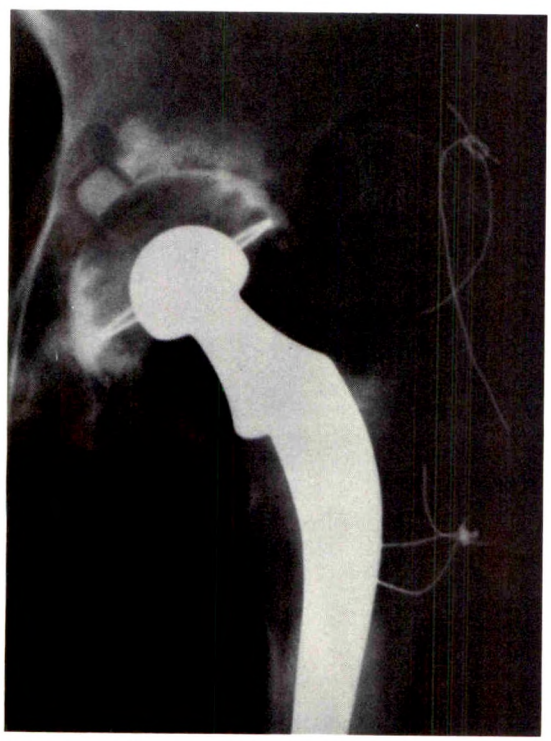


Fig. 3. Avulsion of greater trochanter. Note broken wires. Marked limitation of abduction in this case necessitated reattachment.

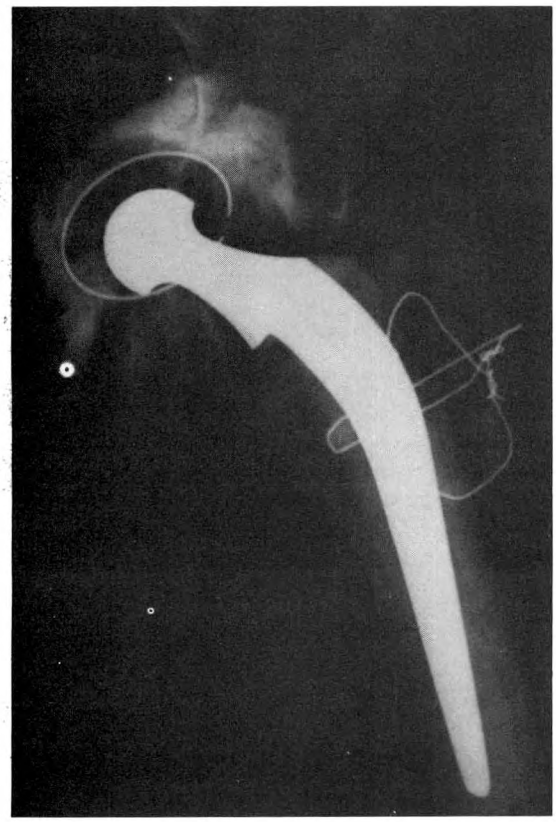


FIG. 5. Protrusion of a cemented acetabular cup into the pelvis in a case of rheumatoid arthritis.

line outlines the acetabular component of the prosthesis separating it from the bony acetabulum. Many authors have noted the indolent nature of infection, some cases reported as late as 4 years after surgery.[3] This suggests to us that radiographic evidence of infection in the absence of clinical signs may deserve more serious consideration.

PENETRATION OF THE LATERAL FEMORAL CORTEX

Technical difficulties in the placement of the femoral implant are occasionally encountered at the time of surgery. Various modifications of surgical technique have been employed in order to reduce this complication, *e.g.*, making a more generous incision proximally, thus allowing the femoral prosthesis to be inserted down the shaft of the femur without having to compete with the abductor muscle bulk.[9] Continued experience in total hip replacement is making this complication (Fig. 7) less common.

SUBOPTIMAL ACETABULAR ORIENTATION

Figure 1 illustrates a normal placement of the prosthesis. The plane of the face of the acetabular component is oriented 45 degrees to the midsagittal plane and minimally anteverted. Anteversion of the prosthesis, as demonstrated in Figure 8, results in "opening" of the circumferential radiopaque marker ring. If measurement of the degree of anteversion is desired, care must be taken to control the radiologic exposure geometry. For example, centering over the mid-pelvis as in Figure 1 results in artifactual "opening" of the plane of the ring due to beam angulation. For proper evalua-

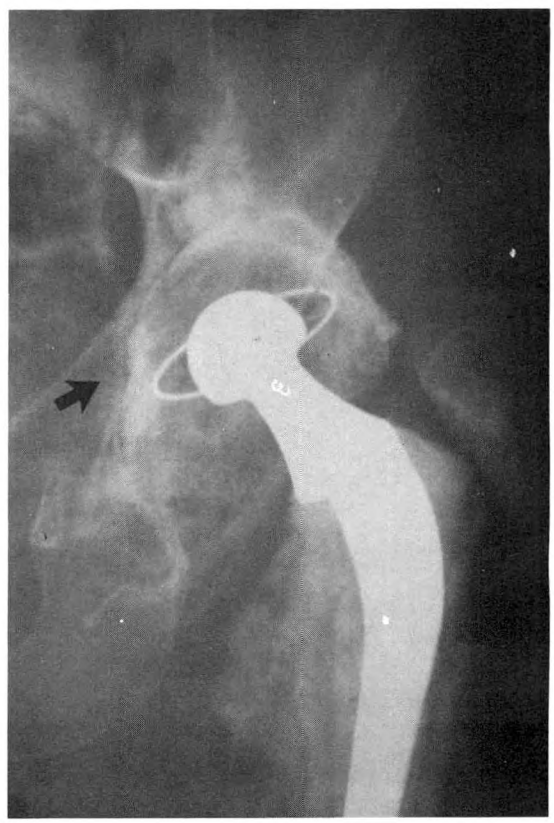


FIG. 6. Postoperative infection. A 72 year old man who developed clinical signs of infection one month after surgery. He was treated with antibiotic irrigation. The arrow indicates lucent line of bone resorption between methyl methacrylate and acetabulum. Periarticular calcification is also evident anteromedially.

tion and to avoid beam angulation, the correct centering position is over the acetabulum itself which results in no artifactual opening of the ring; *i.e.*, both sides of the ring are superimposed, as shown in Figure 9. Müller[8] recommends 10 to 15 degrees of anteversion for his prosthetic device which contains an opaque circumferential ring related equatorially to the cup. He states that this degree of anteversion corresponds to about 10 mm. of radiologic separation or "opening" of the ring. The Charnley prosthesis contains an opaque half circle located in a polar relationship to the cup. While this does not facilitate radiologic assessment of anteversion, it does allow for *in vivo* determination of wear. The Bechtol modification of the Charnley prosthesis contains an equatorial ring similar to that of the Müller modification.

SUMMARY

With increasing use of total hip replacement in orthopedic surgery, it is becoming essential for the radiologist to familiarize himself with the normal as well as the abnormal appearance of prostheses used in this procedure. Several authors have examined the complications of total hip replacement with emphasis on problems which are associated with all aspects of the procedure.[1,3,6,7,9] In a review of cases of total hip replacement using the Bechtol modification of Charnley's low-friction arthroplasty at the University of Missouri since November 1971, only those complications with radiographic findings have been included for discussion.

Radiographically illustrated complications of total hip replacement are: 1. dislocation; 2. disruption of the transplanted greater trochanter; 3. periarticular calcification and ossification or both; 4. protrusion of the acetabulum; 5. infection; 6. penetration of lateral femoral cortex; and 7. suboptimal acetabulum orientation.

It should be noted that not all of these

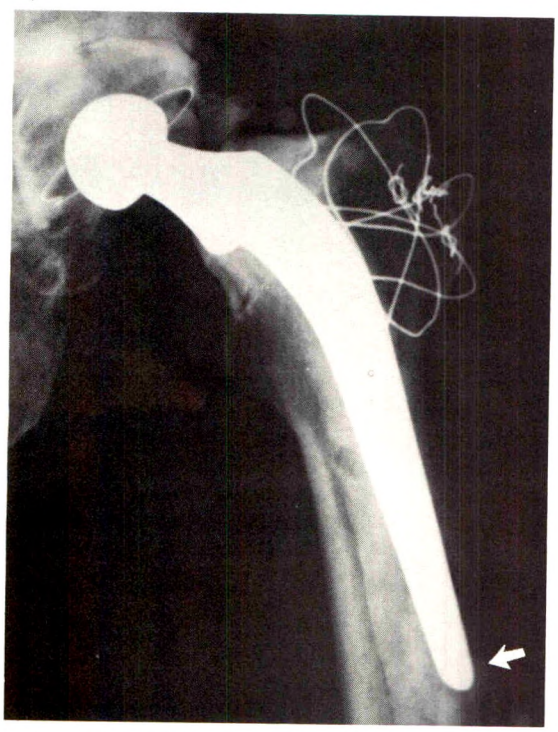


Fig. 7. Tip of femoral prosthesis (arrow) penetrating lateral femoral cortex.

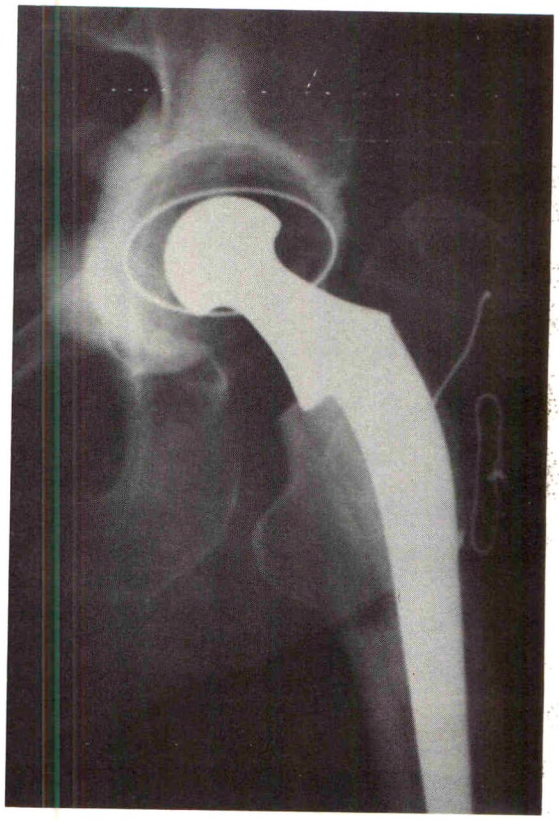


Fig. 8. Marked anteversion of the acetabular cup with the rim oriented 90 degrees to the mid-sagittal plane (compare with Figure 1).

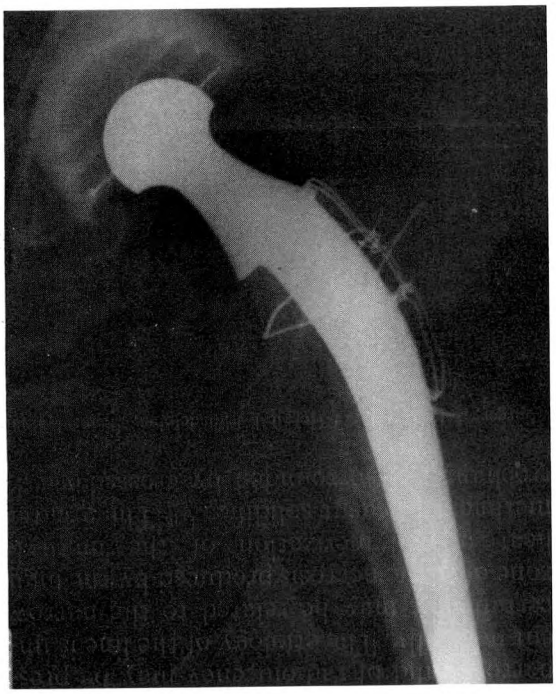


FIG. 9. The x-ray beam is centered correctly over the acetabulum resulting in "closing" of the radiopaque marker ring indicating optimal positioning of the acetabular cup. Note concave channels on acetabular cup which aid in cement penetration and enhance permanent fixation.

complications are associated with clinical or physical disabilities.

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REFERENCES

1. AMSTUTZ, H. C. Complications of total hip replacement. *Clin. Orthopaed. & Related Research*, 1970, *72*, 123–137.
2. ANGELL, F. L., and WATTS, F. B. Total hip replacement: roentgen appearance of current devices. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *110*, 787–792.
3. CHARNLEY, J. Long term results of low-friction arthroplasty of hip performed as primary intervention. *J. Bone & Joint Surg.*, 1972, *54-B*, 61–76.
4. CHARNLEY, J. Total hip replacement by low-friction arthroplasty. *Clin. Orthopaed. & Related Research*, 1970, *72*, 7–21.
5. COLLIS, D. K., and JOHNSTON, R. C. Comparative evaluation of results of cup arthroplasty and total hip replacement. *Clin. Orthopaed. & Related Research*, 1972, *86*, 102–111.
6. DEFFNER, R. H., GEHWEILER, J. A., OLSON, J., WILSON, J. W., and CARDEN, T. S. Dislocation of total hip prosthesis: appearance on frontal roentgenograms. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *115*, 555–560.
7. EFLEKHAR, N. Low-friction arthroplasty: indications, contraindications, and complications. *J.A.M.A.*, 1971, *312*, 705–710.
8. MÜLLER, M. E. Total hip prosthesis. *Clin. Orthopaed. & Related Research*, 1970, *72*, 46–68.
9. STERN, M. B., and GRANT, S. S. Fifty total hip replacements: initial experience. *Clin. Orthopaed. & Related Research*, 1972, *86*, 79–84.
10. TODD, R. C., LIGHTOWLER, C. D. R., and HARRIS, J. Total hip replacement in osteoarthrosis using the Charnley prosthesis. *Brit. M. J.*, 1972, *2*, 752–755.

THE PAINFUL CHARNLEY TOTAL HIP REPLACEMENT*

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TOTAL replacement of the hip by the Charnley technique has gained wide popularity among orthopedic surgeons for its ability to produce a mobile and remarkably painless hip. Pain following a Charnley total hip replacement is unusual. In the Eftekhar[8] series of 120 patients followed for at least 4 years after surgery, only 2 had significant pain at the conclusion of the study. In the patient with a painful total hip replacement, however, this symptom may herald a serious complication.

Careful clinical and roentgenographic examinations are required to determine the etiology of the pain. The radiologist, therefore, must be aware not only of the normal appearance of the prosthetic devices in place, but also of those findings which may indicate a complication.

THE NORMAL CHARNLEY TOTAL HIP REPLACEMENT

The Charnley procedure includes replacement of both the acetabulum and femoral head by prosthetic devices. The acetabular prosthesis is made of high molecular weight, radiolucent polyethylene. It is surrounded superiorly by a wire which aids in localizing the nonopaque prosthesis roentgenographically. The surface is grooved longitudinally and horizontally for better adherence of bone cement to the prosthetic device. When filled with radiopaque bone cement, the grooves may be visible on a roentgenogram. The femoral prosthetic component is a polished, stainless steel structure with a small head designed to fit precisely into the acetabular prosthesis.

Both acetabular and femoral prosthetic components are held in place by methylmethacrylate, an acrylic cement which interdigitates with bone producing a firm surface over which stress is evenly distributed. Methylmethacrylate is radiolucent but is rendered radiopaque by the addition of barium sulfate. To achieve effective anchoring of the acetabular prosthesis, a variable number of "key" holes are drilled in the pelvis about the acetabulum superiorly, medially and inferiorly. When filled with methylmethacrylate, these holes produce a greater surface area over which stress can be distributed. A small, hat-shaped device is placed in the central key hole, where the acetabular bone is thinnest, to prevent cement from entering the pelvis.

The normal roentgenographic positions of the prosthetic components are best visualized on anteroposterior and cross table lateral roentgenograms (Fig. 1, *A* and *B*). A well-placed acetabular component faces laterally at 45 degrees from the horizontal—an angle simulating that of the normal acetabulum. It is medially displaced in the acetabular fossa and usually rotated a few degrees forward. The shaft of the femoral component should be in the center of the medullary canal. Barium impregnated methylmethacrylate should be seen separating the prosthetic device from the bone. At the bone-cement interface, a narrow band of radiolucency may be present normally. The etiology of the line is uncertain but may be related to the narrow zone of tissue necrosis produced by the high heat of polymerization of the methylmethacrylate as it solidifies.[1,13] The greater trochanter is surrounded by crossed verti-

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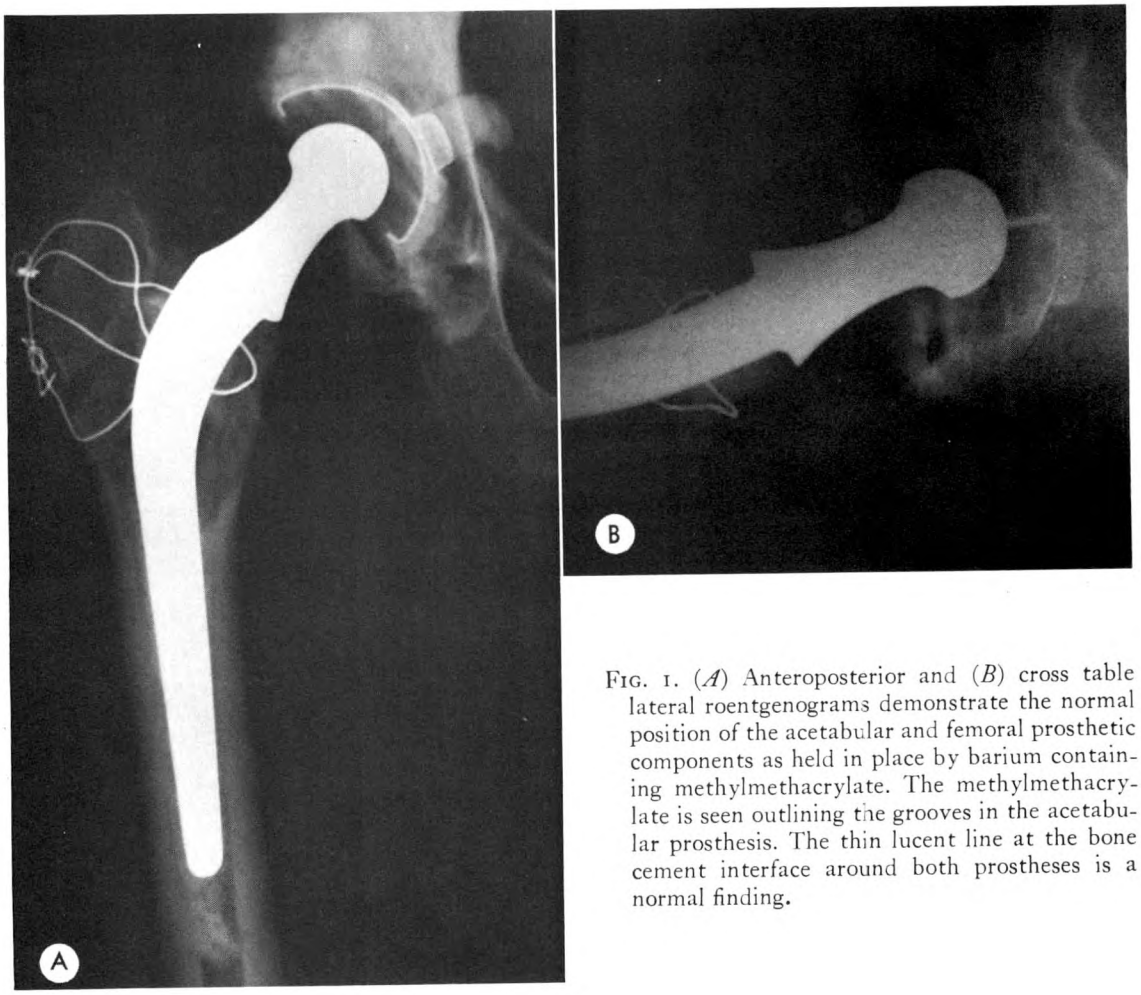


FIG. 1. (*A*) Anteroposterior and (*B*) cross table lateral roentgenograms demonstrate the normal position of the acetabular and femoral prosthetic components as held in place by barium containing methylmethacrylate. The methylmethacrylate is seen outlining the grooves in the acetabular prosthesis. The thin lucent line at the bone cement interface around both prostheses is a normal finding.

cal and horizontal wires which hold the trochanter after it has been osteotomized to permit better exposure of the hip joint during the surgical procedure.

THE ABNORMAL CHARNLEY TOTAL HIP REPLACEMENT

The painful, complicated Charnley total hip replacement may be encountered at any time postoperatively, although some complications are more likely to be found at specific times after surgery.

In the immediate postoperative period, significant pain in the replaced hip which lasts longer or is more severe than is usually encountered after any surgical procedure should be carefully evaluated. Often the diagnosis is clinical, and the pain is due to a wound infection, hematoma or seroma. A rare possibility, to be considered especially in the patient with severely osteoporotic bone, is an unrecognized fracture of the pelvis or proximal femur inadvertently produced during surgery.

After the immediate postoperative period, the most common and most serious causes of pain in a replaced hip are loosening of the prosthetic components and deep infection.

Clinical recognition of a loose prosthesis is usually based on the acute onset of pain following sudden activity or weight bearing stress. The pain may be in the hip, groin, thigh or knee. Pain of a sharp, constricting nature in the mid-thigh frequently indicates loosening of the femoral shaft. A

painful "clunk" elicited on motion is considered a reliable sign of a loose or unstable prosthesis.

Roentgenographic diagnosis of loosening usually depends upon previous roentgenograms for comparison. Progressive widening of the thin, radiolucent line normally seen surrounding the radiopaque methylmethacrylate is an important sign of loosening. Also, the prosthetic components may show migration relative to bony landmarks[15] (Fig. 2, *A* and *B*).

Cine roentgenography has been employed with some success to diagnose loosening, but plain roentgenograms in full adduction and abduction will frequently demonstrate a change in the positions of the prostheses. Occasionally, arthrography may be necessary to conclusively demonstrate loosening. A traction roentgenogram should be included in the routine arthrography examination, since evidence of minimal loosening may be seen only with this maneuver.[12] Extravasation of contrast material around the radiopaque methylmethacrylate is best visualized by subtraction techniques which allow visual separation of the contrast material from the methylmethacrylate which is of approximately the same density.

Loosening alone usually occurs because of improper handling or insertion of the methylmethacrylate during surgery,[15] but more often, the signs of loosening indicate underlying deep infection. Infection surrounding the replaced joint may occur soon after surgery and is suspected when the patient continues to have significant pain in

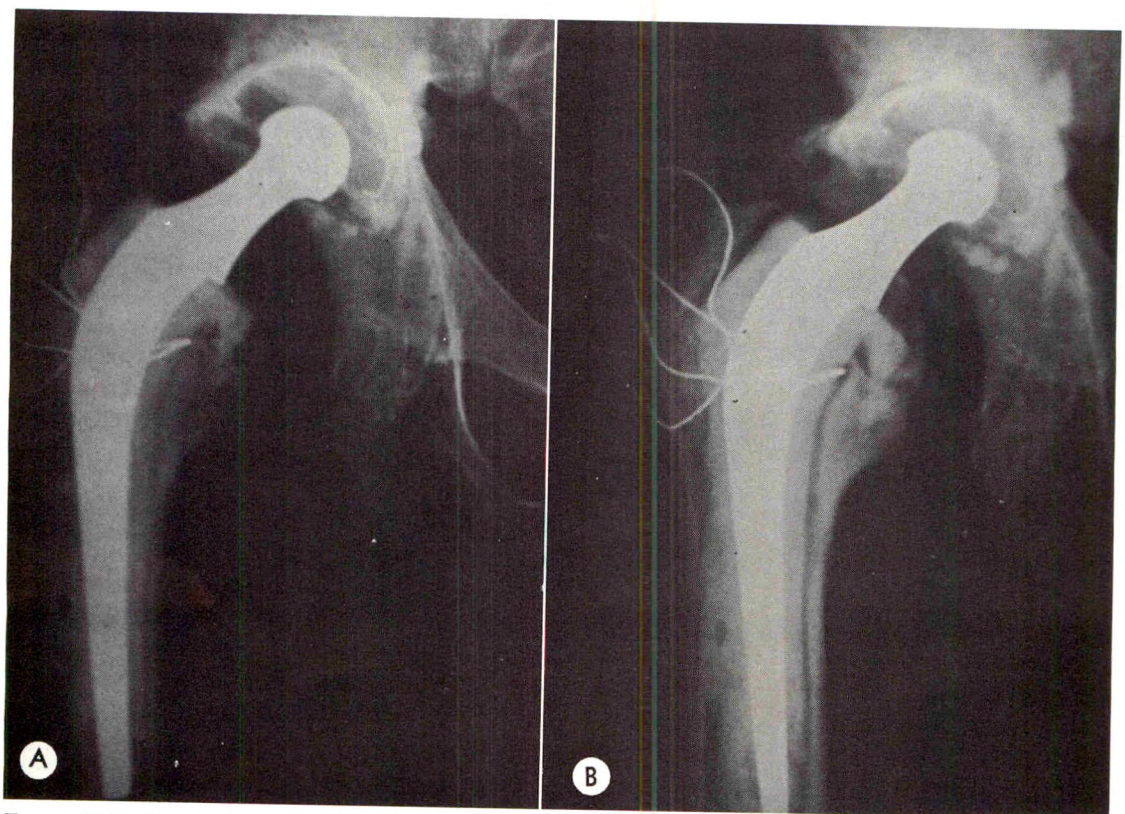


Fig. 2. This 26 year old male had a Charnley total hip replacement after failure of a previous Tronzo total hip replacement.

(*A*) Initial roentgenogram was normal. (*B*) Nine months later, significant bone resorption has occurred about the methylmethacrylate surrounding and anchoring the femoral prosthesis. This finding is consistent with loosening of the prosthesis, either with or without associated deep infection.

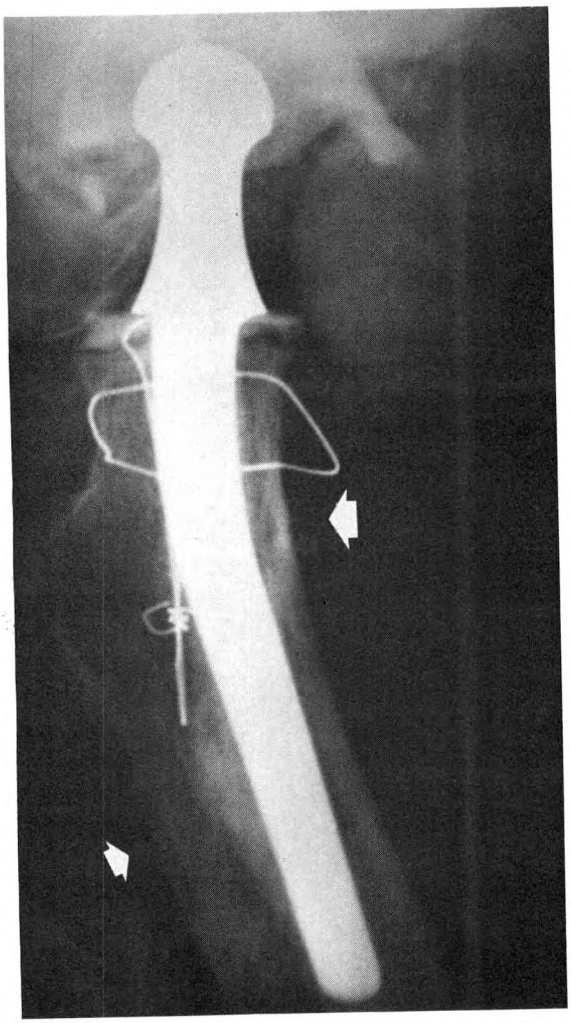


FIG. 3. This 67 year old male had continuous hip pain following his total hip replacement.

The lateral roentgenogram demonstrates periosteal reaction (arrows), the only sign of the infection which was proven to cause the pain.

the early weeks of ambulation. Frequently in total hip replacements, however, the pain of a deep infection may develop only after months of a pain free, uncomplicated course. In Charnley's series of 582 patients followed 4 to 7 years postoperatively, infection was encountered early in 1.6 per cent, but only after several months had passed in 2.2 per cent.[5]

Fever, leukocytosis and other clinical signs of infection are often absent. Even aspiration of the joint may be negative,[7] and pain may be the only clue to the development of a deep infection. Roentgenographically, the appearance of infection may be indistinguishable from that of loosening, with bone resorption due to the infection producing the widening of the lucent line surrounding the cement. Occasionally, the development of a diffuse, ragged osteitis and irregular cortical thickening and sclerosis may aid in recognition of the difference between these 2 entities[9] (Fig. 3).

All patients with a painful total hip replacement should be carefully evaluated for infection but special emphasis should be placed on those who have had previous hip surgery, since statistically they have an increased incidence of infection.[7,10]

Although other modalities have been tried, the only proven therapy for a deep infection is removal of the prosthetic components with conversion to a Girdlestone pseudoarthrodesis.

Severe pain may be caused by factors other than loosening and infection, but they are extremely rare. A prosthetic component may fracture due to fatigue of the material.[11] In the case of the femoral component, the diagnosis is based on the discovery of a lucency in the densely opaque metal. In the case of the nonopaque acetabulum, however, the diagnosis may be more difficult. Unless the fracture is gross and the parts of the prosthesis are separated, the demonstration, during arthrography, of opaque dissecting into the fracture may be the only means of detecting a fracture. The radiologist should be aware of one pitfall that may occur in the evaluation of the acetabular component for fracture. Occasionally, the grooves in the acetabular prosthesis, filled with methylmethacrylate, will be almost superimposed, with only a thin line of lucency between the grooves. The acetabulum may thus appear fractured. Another roentgenogram in a slightly different rotation will, however, show the grooves in a different projection and the widening or further narrowing of the lucent line will prove that the prosthesis is actually intact (Fig. 4, *A* and *B*).

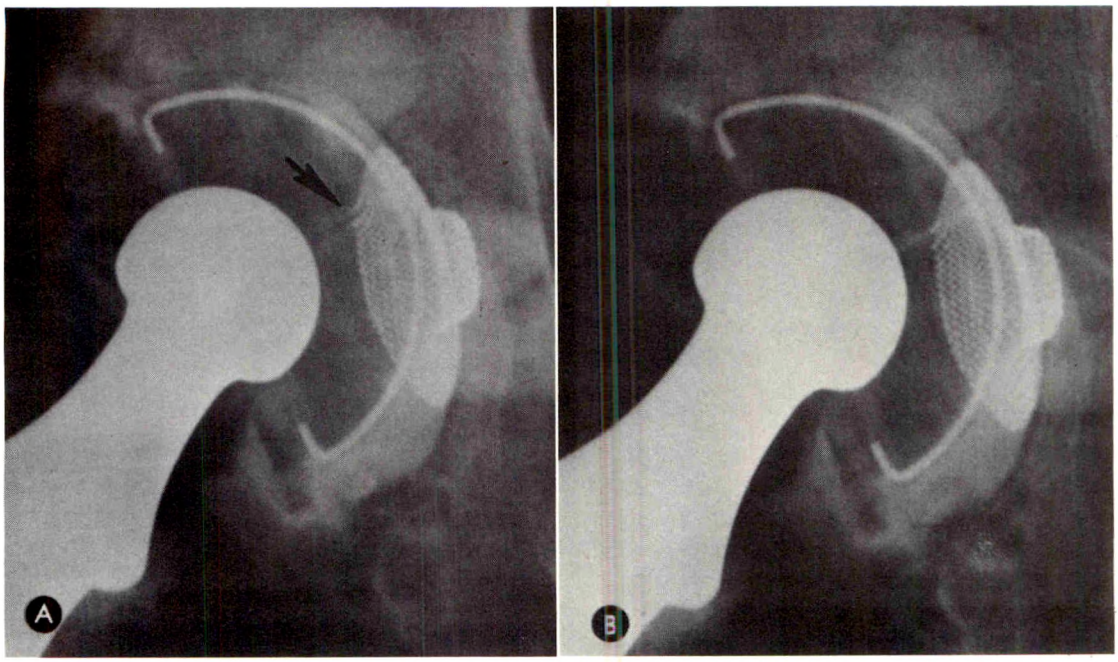


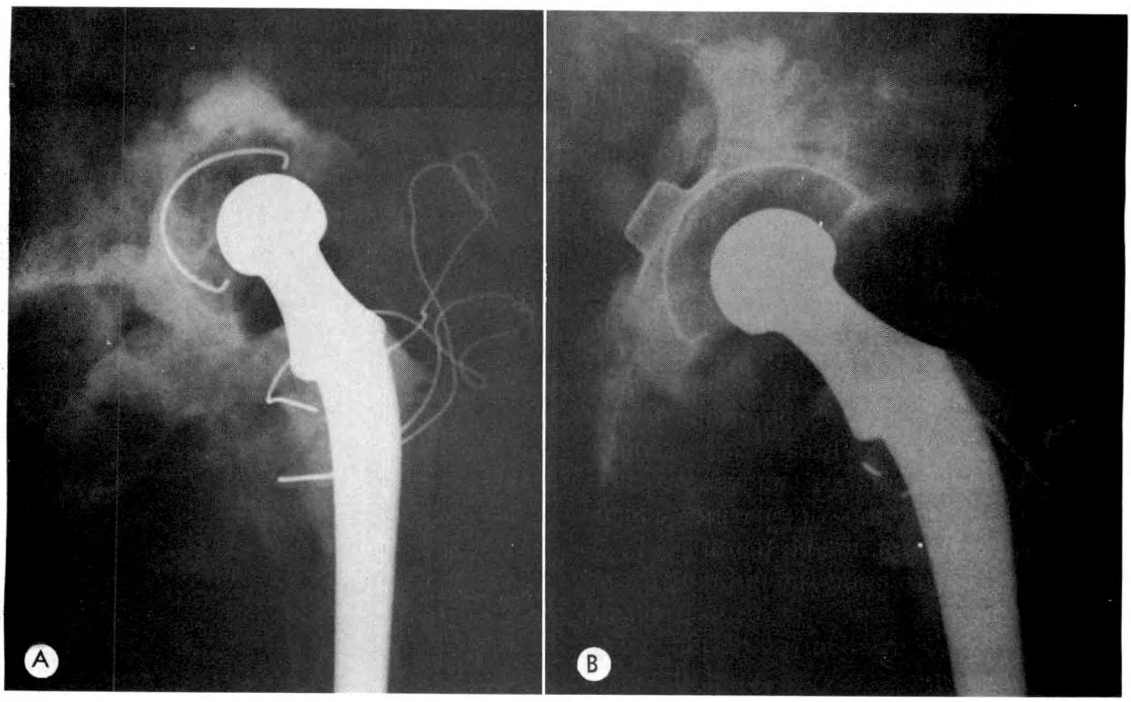
Fig. 4. (*A*) On a routine follow-up roentgenogram of the replaced hip of this 26 year old asymptomatic male there is demonstrated a lucent line (arrow) in the acetabular prosthesis which has the appearance of a fracture. (*B*) In a slightly different rotation, the lucency disappears proving that the prosthesis is intact. The excessively vertical placement of the acetabular component could lead to femoral dislocation.

Most of the other complications of the Charnley total hip replacement do not cause severe pain but may produce a variety of other symptoms. The complication in this category most likely to cause immediate concern is a dislocation. Because of the small femoral head used in the Charnley technique, it was initially thought that dislocation would be common, but it actually occurs in less than 2 per cent of patients.[10] Dislocation is usually seen in the first postoperative month and may result from adduction or ambulation before a solid fibrous capsule has formed around the replaced joint. The roentgenographic diagnosis of a dislocation is based on loss of the normal, almost parallel relationship between the curve of the femoral head and the wire surrounding the acetabular component (Fig. 5).

Dislocation may be the consequence of abnormal angulation of the acetabular component during surgery. Excessively vertical placement of the cup may be conducive to



Fig. 5. This 64 year old, massively obese female had a previous dislocation of an Austin Moore prosthesis. After replacement of this device with a Charnley prosthesis, repeated femoral dislocations occurred, as demonstrated on this anteroposterior roentgenogram.


A
B

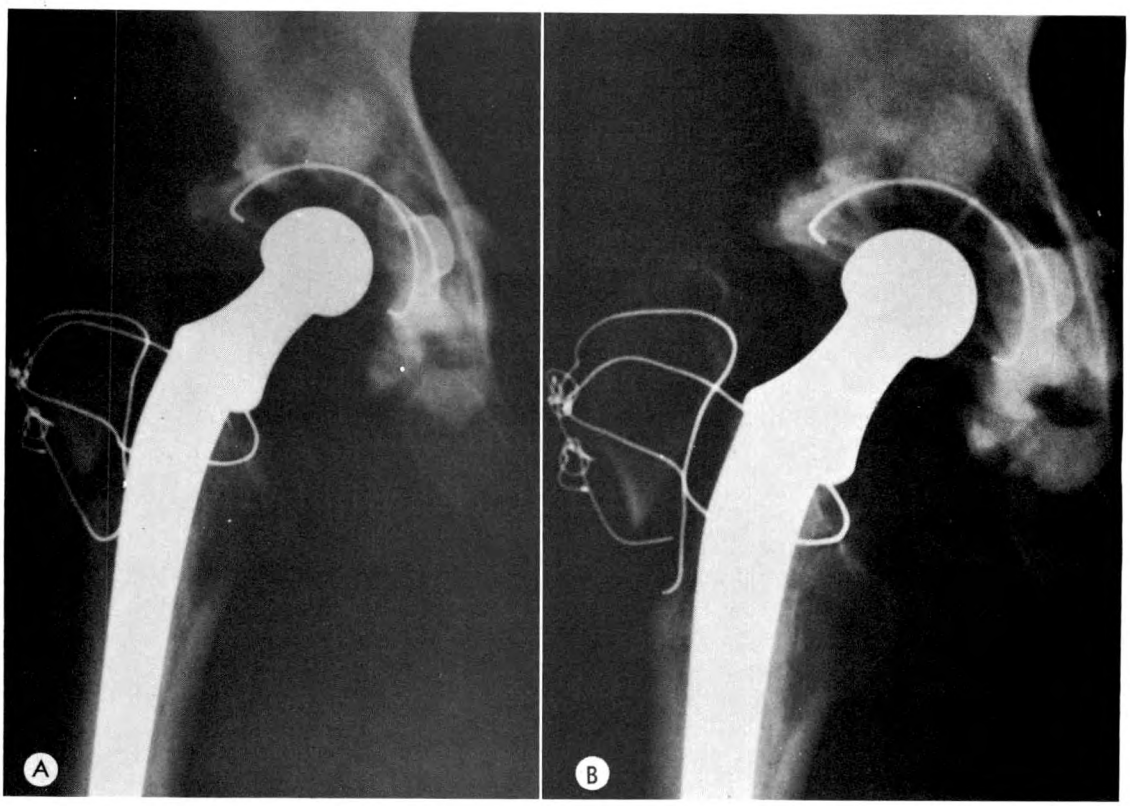

A
B

FIG. 6. (*A*) Anteroposterior roentgenogram of the replaced hip of this 66 year old male illustrates abnormal placement of the acetabular component outside the normal acetabular fossa. (*B*) Another surgical procedure was required to properly position the prosthesis. The trauma of repeated surgery has produced myositis ossificans, demonstrated lateral to the joint.

dislocation on adduction. Dislocation with abduction is a possibility if the cup is too horizontal but such placement usually only limits the range of abduction. The acetabular component may also be placed, during surgery, outside the true acetabular fossa and with stress, either just the femoral head, or even both components, may dislocate (Fig. 6, *A* and *B*).

The most frequently encountered complications of the Charnley procedure, those involving the osteotomized greater trochanter, are also the least likely to produce significant symptoms. In Charnley's series, 7.3 per cent of patients showed breakage of the wires holding the trochanter and 4.2 per cent had nonunion of the replaced trochanter.[5]

Wire breakage is usually seen after the trochanter has united with the femur, either by bony or fibrous union. It does not of itself cause symptoms and is probably due to cyclic stress on the wires with time.[1] When the wires are broken early in the postoperative period, however, the trochanter, which has not yet united with the femur, may migrate, resulting in a nonunion (Fig. 7, *A* and *B*). This complication is also frequently asymptomatic but occasionally has been shown to cause pain, a gluteal limp and an increased tendency to develop an adduction contracture.[10]

Hypertrophic or ectopic bone formation, and myositis ossificans, are occasionally seen, especially in the patient who has had previous hip surgery (Fig. 6*B*). They are infrequent causes of pain and are more likely to be clinically apparent by limiting motion of the joint.

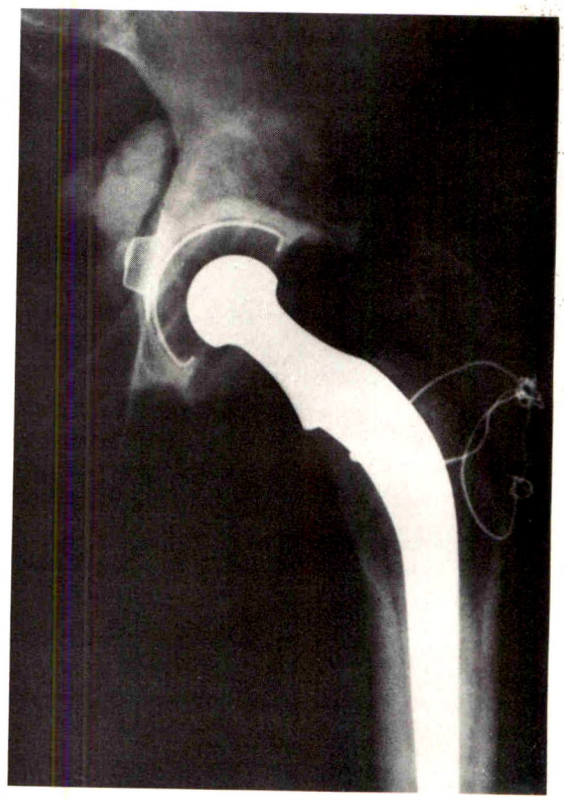


FIG. 8. Extravasation of methylmethacrylate into the pelvis, as demonstrated in this total hip replacement in a 61 year old female, is a common occurrence which rarely produces symptoms.

The extravasation of methylmethacrylate into the pelvis is a very frequent roentgenographic finding in total hip replacements (Fig. 8) and is usually entirely

FIG. 7. (*A*) Roentgenogram taken within 1 month following total hip replacement shows no abnormalities. At this time, the greater trochanter is not yet normally united to the femur. (*B*) Two months later, the wires surrounding the greater trochanter have broken and the trochanter has migrated superiorly. The patient was asymptomatic.

asymptomatic. However, at least 1 case of neurologic impairment of the leg has been reported secondary to sciatic nerve impingement by the intrapelvic cement.[8]

CONCLUSION

In summary, the presence of pain in a hip replaced by the Charnley technique is infrequent, but warrants careful clinical and roentgenographic evaluation for a serious complication.

The most common complications responsible for persistent postoperative pain are loosening of the prosthetic components and deep infection.

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REFERENCES

1. AMSTUTZ, H. C. Complications of total hip replacement. *Clin. Orthop.*, 1970, *72*, 123–137.
2. CAMPBELL, R. E., and ROTHMAN, R. H. Charnley low-friction total hip replacement. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 634–641.
3. CASAGRANDE, P. A., and DANAHY, R. R. Delayed sciatic nerve entrapment following use of self-curing acrylic: case report. *J. Bone & Joint Surg.*, 1971, *43-A*, 167–169.
4. CHARNLEY, J. Total hip replacement by low-friction arthroplasty. *Clin. Orthop.*, 1970, *72*, 7–21.
5. CHARNLEY, J. Low-friction arthroplasty of hip joint: symposium of joint biomechanics and joint replacement. *J. Bone & Joint Surg.*, 1971, *53-B*, 149.
6. CHARNLEY, J., and WELCH, R. B. Low-friction arthroplasty of hip in rheumatoid arthritis and ankylosing spondylitis. *Clin. Orthop.*, 1970, *72*, 22–32.
7. EFTEKHAR, N. Low-friction arthroplasty: indications, contraindications, and complications. *J.A.M.A.*, 1971, *218*, 705–710.
8. EFTEKHAR, N. Charnley "low-friction torque" arthroplasty. *Clin. Orthop.*, 1971, *81*, 93–104.
9. EFTEKHAR, N., and STINCHFIELD, R. E. Total hip replacement by low-friction arthroplasty. *Orthop. Clin.*, 1973, *4*, 483–502.
10. LAZANSKY, M. G. Complications in total hip replacement with Charnley technique. *Clin. Orthop.*, 1970, *72*, 40–45.
11. MÜLLER, M. E. Total hip prostheses. *Clin. Orthop.*, 1970, *72*, 46–68.
12. SALVATI, E. A., FREIBERGER, R. H., and WILSON, P. D. Arthrography for complications of total hip replacement. *J. Bone & Joint Surg.*, 1971, *53-A*, 701–709.
13. STEIN, I., SIMON, W. H., and BELLER, M. L. Role of total hip joint replacement in treatment of hip joint disorders. *Pennsylvania M. J.*, 1972, *75*, 58–62.
14. STERN, M. B., and GRANT, S. S. Fifty total hip replacements: initial experience. *Clin. Orthop.*, 1972, *86*, 79–84.
15. WILSON, J. N., and SCALES, J. T. Loosening of total hip replacements with cement fixation: clinical findings and laboratory studies. *Clin. Orthop.*, 1970, *72*, 145–160.

"CLIPPING INJURY" FRACTURE OF THE EPIPHYSIS IN THE ADOLESCENT FOOTBALL PLAYER: AN OCCULT LESION OF THE KNEE*

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EVEN a passing acquaintance with the Sport pages makes one aware that the knee of an athlete is quite vulnerable to injury, particularly in football. Most discussions center around ligamentous injury and internal derangements in professional and college athletes. One might assume that the high school athlete or adolescent would be susceptible to these same injuries; however, the adolescent knee may react differently to the same stresses. Forces that would result in a ligamentous injury in an adult are likely to result in an epiphyseal injury in a child or adolescent.

The most common cause of a ligamentous injury or epiphyseal separation of the knee is a valgus stress, a force applied on the lateral surface of the knee. This most commonly results from a so-called "clipping" injury in football, when a player is blocked or tackled from the lateral side at the level of the knee with the foot firmly implanted. In the adult this valgus force results in a strain or rupture of the medial collateral ligament.[7,10,12] In the adolescent, the valgus force may be transmitted through the medial collateral ligament onto the distal femoral epiphysis or, less commonly, the proximal tibial epiphysis with a resultant epiphyseal separation.[7,8] Severe valgus stresses are generated in other circumstances and occasionally result in similar injuries.

Most epiphyseal injuries are clinically and roentgenographically apparent. There remains a small number of undisplaced epiphyseal separations wherein the routine roentgenograms are unrevealing, thus suggesting or strengthening the clinical impression of a strain or rupture of the medial collateral ligament. A torn ligament requires immediate surgery, whereas an undisplaced epiphyseal separation is successfully treated by casting alone. A mistaken diagnosis and an unnecessary surgical procedure can be avoided by appropriate roentgenographic evaluation.

MATERIAL AND METHOD

This series consists of 8 undisplaced epiphyseal separations, 7 of the distal femoral and 1 of the proximal tibial epiphysis. All occurred in males between the ages of 14 and 17 years, median age 15.4 years, and were the result of a severe valgus stress. Six occurred while playing football, 4 resulting from a clip-type block and 2 as the result of a tackle. Two were the result of traffic accidents, 1 thrown from an automobile and the other thrown from a motorcycle. The clinical and roentgenographic findings were similar to the "clipping-injury" cases and, therefore, they are included in the present series. The pertinent findings are summarized in Table 1.

The end of a growing bone consists of the epiphysis, growth plate, and metaphysis.[8,9] The growth plate consists of 4 distinct

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TABLE I

FINDINGS IN "CLIPPING INJURY" CASES

Case	Age (yr.)	Side	Mechanism of Injury	Salter-Harris Classification	Roentgenographic Projection Revealing Fracture
			Distal Femoral		
I. GB	17	Left	Clip	Type 3	Stress
II. RG	15	Right	Tackle	Type 3	Oblique
III. RN	17	Right	Clip	Type 3	Stress
IV. LP	16	Left	Auto accident	Type 3	Oblique
V. RAR	14	Right	Tackle	Type 3	Stress
VI. ROR	15	Right	Clip	Type 3	Anteroposterior
VII. MW	15	Left	Clip	Type 3	Anteroposterior (14 days post injury)
			Proximal Tibial		
VIII. CL	14	Left	Motorcycle accident	Type 2	Probable on anteroposterior, confirmed on stress

zones of cartilaginous cells: the resting cells in juxtaposition to the epiphysis; the proliferating cells; the hypertrophied cells; and, lastly, the zone of provisional calcification at the metaphysis. In epiphyseal separations, the line of fracture consistently lies within the zone of hypertrophied cells.

The Salter-Harris classification[8,9] of epiphyseal injuries consists of 5 types. The first 3 are caused by forces parallel to or away from the epiphysis. These are predominantly shearing or avulsive forces. The shearing forces are usually generated directly upon the epiphysis by the force creating the injury, whereas the avulsive forces are transmitted to the epiphysis by ligaments and tendons attached to it. Type 1 is a pure epiphyseal separation, a line of fracture involving the hypertrophied zone of the growth plate without involvement of either the epiphysis or metaphysis. Type 2 consists of a fracture line through the zone of hypertrophied cells which at some point extends into the metaphysis, thus a fragment of the metaphysis accompanies the epiphysis. Type 3 is a fracture extending through the zone of hypertrophied cells which then turns into the epiphysis, thus separating off a portion of the epiphysis from the metaphysis. The 2 remaining types, Types 4 and 5, are the result of a force directed or transmitted towards the epiphysis through the opposing bone. Type 4 is a fracture extending across the epiphyseal growth plate and metaphysis. Type 5 is a pure compression injury of the growth plate. The forces generated by a clipping injury are either shearing or avulsive in nature. All 7 of the distal femoral epiphyseal separations were Type 3. The proximal tibial epiphyseal separation was Type 2. Undisplaced Type 2 injuries of the distal femoral epiphysis have been reported[7,11] as a result of a clipping injury, although none were encountered in this series. Type 2 injuries are the only injuries reported of the proximal tibial epiphysis[7,13] under these exact circumstances.

PHYSICAL EXAMINATION

An undisplaced epiphyseal separation is difficult to diagnose by physical examination alone. Muscle guarding and swelling are present. Pain is elicited by palpation over the medial aspect of the knee. The latter would tend to strengthen the impression of a medial collateral ligamentous sprain or tear. A careful, detailed examination might elicit tenderness and pain completely around the growth plate in the

presence of a Type 2 injury;[7] however, this is unusual. None of the present series were diagnosed on the basis of physical examination alone. Roentgenographic evaluation is required.

ROENTGENOGRAPHIC EVALUATION

Grossly displaced epiphyseal separations are easily recognized.[3] Type 2 injuries are most common and distal femoral separations are more frequent than proximal tibial separations. The metaphyseal fragment, or corner sign, is evident.

Undisplaced epiphyseal separations represent a diagnostic challenge. The opposing margins of an epiphyseal fracture consist of cartilage and are relatively smooth; therefore, the separated epiphysis may glide easily over the opposed metaphysis. This is aided by the pull of tendons and ligaments about the joint (Fig. 1). The epiphyseal separation may then reduce spontaneously. A Type 3 injury of the distal femoral epiphysis involving the medial condyle is the most common undisplaced, spontaneously reduced, epiphyseal injury at the knee. Similarly, undisplaced Type 2 separations of the proximal tibial[7,13] or distal femoral epiphysis[7,11] may occur.

The diagnosis is made by the identification of a widened growth plate and a fracture line in the epiphysis in Type 3 (Fig. 2, *A* and *B*; and 3*C*) or a fracture line in the metaphysis, creating a corner sign, in Type 2 (Fig. 6, *A* and *B*). In a Type 2 injury created by valgus forces, the metaphyseal fracture is situated laterally. The widening of the growth plate is more pronounced medially in either type (Fig. 2*A*; 3*C*; and 6*A*). In Type 3 injuries of the distal femoral epiphysis, the medial femoral condyle is involved. The epiphyseal fracture line lies within the intercondylar notch (Fig. 2*A*; 3*C*; and 5*C*).

The diagnosis was obvious on the standard anteroposterior and lateral roentgenograms in only 1 of the 7 Type 3 fractures of the distal femoral epiphysis (Fig. 2*A*). A vague vertical fracture line may be evident within the notch in the anteroposterior view. In retrospect, 3 cases in the present series demonstrated such lines (Fig. 3*A*;

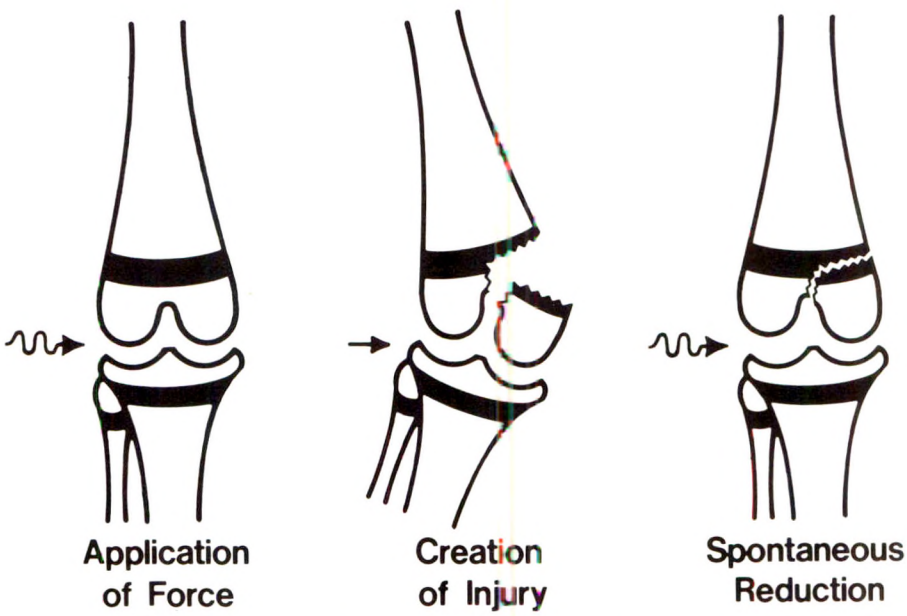


Fig. 1. Diagrammatic representation of mechanism of Salter-Harris Type 3 injury of the distal femoral epiphysis. The spontaneous reduction occurs because of the pull of the ligaments and tendons about the joint aided by the smoothness of the opposing margins of the fractured cartilage within the growth plate.

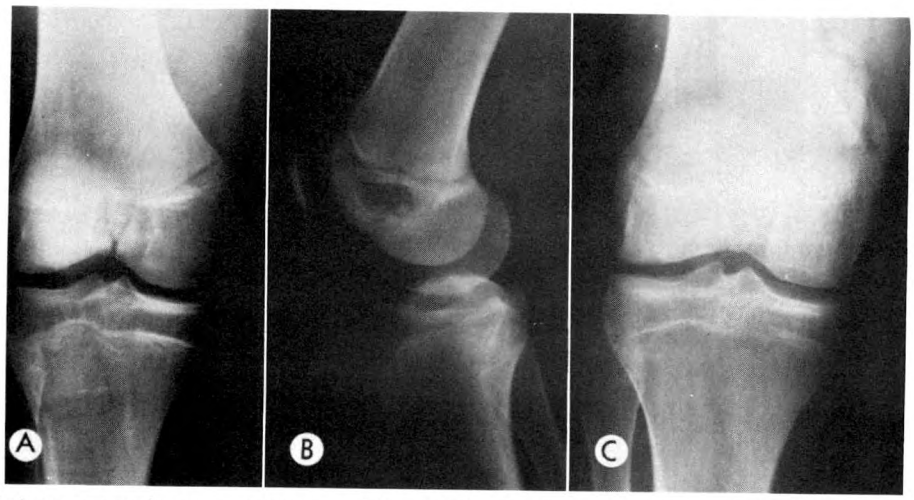


FIG. 2. (*A*) Anteroposterior roentgenogram of the right knee of a 15 year old football player who sustained a clip-type block. Note fracture lines in the intercondylar notch and the medial portion of the distal femoral growth plate. (*B*) The lateral view is unrevealing except for the presence of a joint effusion. (*C*) Three and one-half months post injury. Extensive calcification is present at the superior margin of the medial femoral condyle and the distal femoral shaft, presumably the result of torn periosteum. The fracture has healed.

4*A*; and 5*A*). The proximal tibial Type 2 injury was suggested, but not considered definite, in the standard projections (Fig. 6, *A* and *B*).

Oblique projections have been helpful, particularly when made with the knee in a few degrees of external rotation (Fig. 3*C*). These were obtained in only 2 cases and demonstrated an otherwise inapparent injury in each case. In 1 of these 2 cases, a tunnel or notch view was obtained and also definitely demonstrated the intercondylar fracture.

Anteroposterior roentgenograms obtained during the application of a valgus stress on the knee have been utilized to confirm the presence of a medial collateral ligamentous rupture by the demonstration of a medial

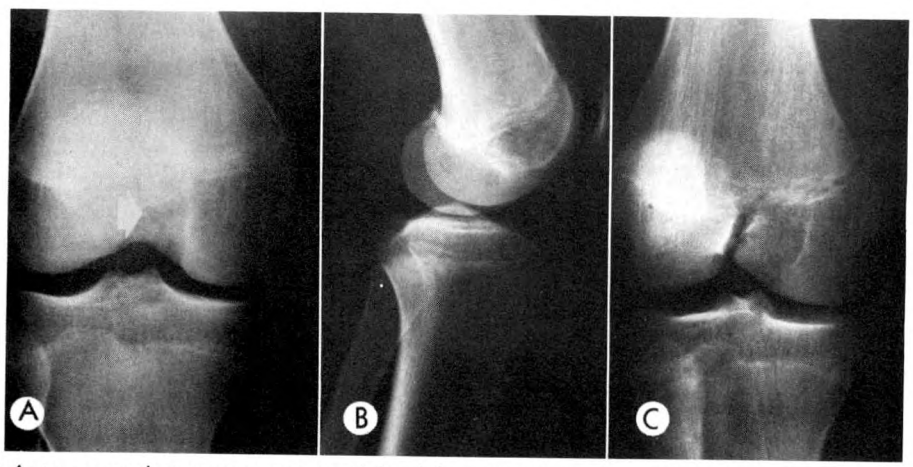


FIG. 3. (*A*) Anteroposterior roentgenogram of the right knee of a 15 year old football player who was tackled from the side at the knee. This view is unremarkable. In retrospect, there is a vague vertical fracture line (arrow) in the intercondylar notch and a slight offset of the femoral epiphysis medially. (*B*) Lateral view demonstrates only a minute metaphyseal fragment arising from the extreme posterior margin of the distal femoral metaphysis. (*C*) Anteroposterior roentgenogram with minimal external rotation reveals a Salter-Harris Type 3 injury of the distal femoral condyle.

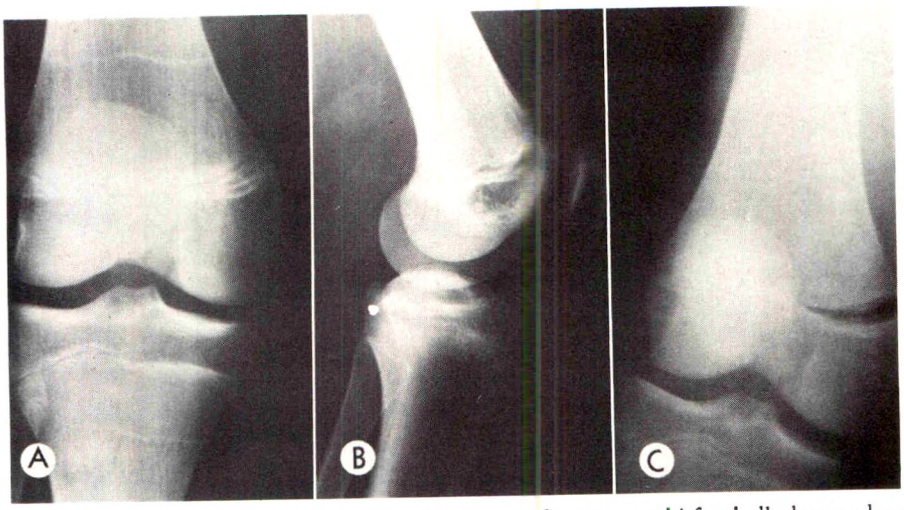


Fig. 4. (*A*) Anteroposterior roentgenogram of the right knee of a 17 year old football player who sustained a clip-type block. Only a vague vertical fracture line is present in the intercondylar notch. (*B*) Lateral view demonstrates only a joint effusion. (*C*) Valgus stress view demonstrates a Salter-Harris Type 3 injury of the medial portion of the distal femoral epiphysis. The vertical component of the fracture line is not well visualized on this particular view. Note "phantom-air" phenomenon in fractured growth plate. (Courtesy of Dr. William Veatch, Olympia, Washington.)

opening or widening of the joint space.[12] The importance of the stress roentgenogram in the adolescent and young adult suspected of medial collateral ligament injury has been emphasized by several authors.[7,11,12] This may demonstrate an otherwise unsuspected, inapparent epiphyseal separation of either the distal femoral[7,11] or proximal tibial epiphysis.[7,12] In the present series, 3 of the 7 Type 3 injuries of the distal femoral epiphysis were disclosed only by this method (Fig. 4*C*; and 5*C*). A stress roentgenogram was also utilized to confirm the Type 2 injury of the proximal tibial epiphysis (Fig. 6*C*) suspected on the basis of standard anteroposterior and lateral roentgenograms. Our experience suggests that the increased use of

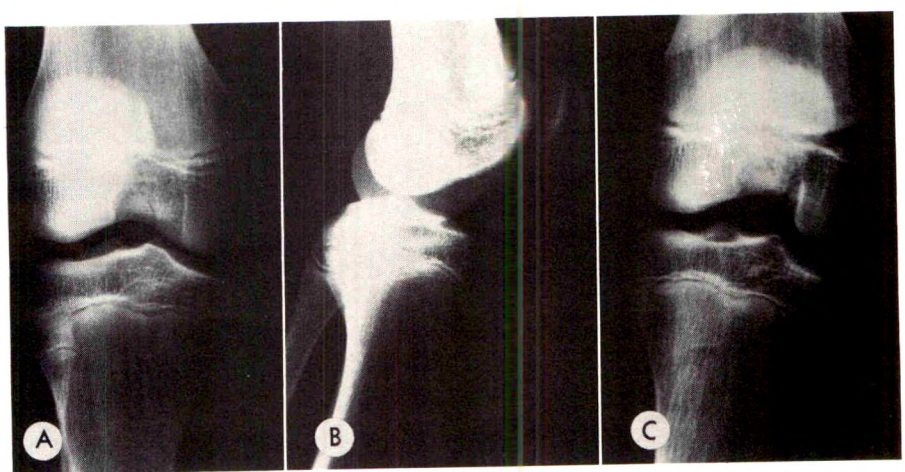


Fig. 5. (*A*) Anteroposterior roentgenogram of the left knee of a 14 year old football player who was tackled from the side. The fracture is not apparent. (*B*) Lateral view. A joint effusion is present. The fracture is not visualized. (*C*) Valgus stress view demonstrates Salter-Harris Type 3 injury of the medial portion of the distal femoral epiphysis.

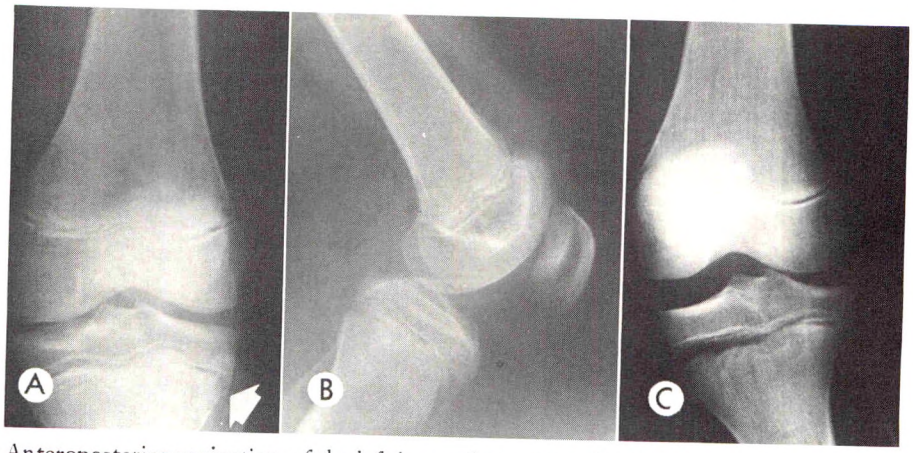


FIG. 6. (*A*) Anteroposterior projection of the left knee of a 14 year old male thrown from a motorcycle. Note questionable widening of the proximal tibial growth plate medially and metaphyseal fracture laterally (arrow). (*B*) The lateral view is unrevealing. (*C*) Valgus stress view demonstrates widening of the proximal tibial growth plate indicating the presence of an epiphyseal injury. This proved to be a Salter-Harris Type 2 injury. The metaphyseal fracture is not visualized in this view. (Courtesy of Dr. Charles E. Baker, Houston, Texas.)

oblique projections might reduce the necessity of obtaining stress roentgenograms to disclose the fracture. In this series, prior oblique projections were not obtained in any of the 4 cases in which a stress roentgenogram was utilized. In all 4 cases, the epiphyseal fracture was successfully demonstrated by this stress roentgenogram.

In 1 case the correct diagnosis was established on the basis of roentgenographic findings in standard anteroposterior and lateral projections made 2 weeks after the injury. These disclosed the fracture line and callus formation. The injury was not apparent on the original roentgenograms. The clinical impression was a ruptured medial collateral ligament. The second roentgenograms were obtained as a preliminary evaluation prior to knee arthrography. The arthrography was cancelled upon review of the preliminary roentgenograms.

TREATMENT AND PROGNOSIS

The importance of making a distinction between ligamentous injury and epiphyseal separation is that undisplaced epiphyseal injuries of the distal femoral[3] and proximal tibial[1] epiphysis may be treated by closed reduction and casting. In contradistinction, ruptures of the medial collateral ligament require an immediate operative procedure and reapproximation of the torn margins.[7]

The prognosis of these particular epiphyseal injuries is excellent. Residual significant deformities, growth disturbances, or limitations of motion have not occurred. Calcification has appeared within the soft tissues attached to the superior margin of the medial femoral condyle on the metaphysis (Fig. 2*C*). This has appeared in all 6 cases of undisplaced Type 3 distal femoral epiphyseal separations in whom post-injury roentgenograms are available for review. It appears between 3 and 8 weeks following injury. This calcification is thought to be related to the torn periosteum or possibly torn adductor magnus insertion. It does not appear to be within the medial collateral ligament and cannot, therefore, be accurately described as Pellegrini-Stieda's disease.[10] In any event, it is not associated with clinical symptoms or signs.

DISCUSSION

Anatomic dissection of a young human cadaver, an immature monkey, and rabbit leg specimens have confirmed that both the medial and lateral collateral ligaments originate proximally on the respective

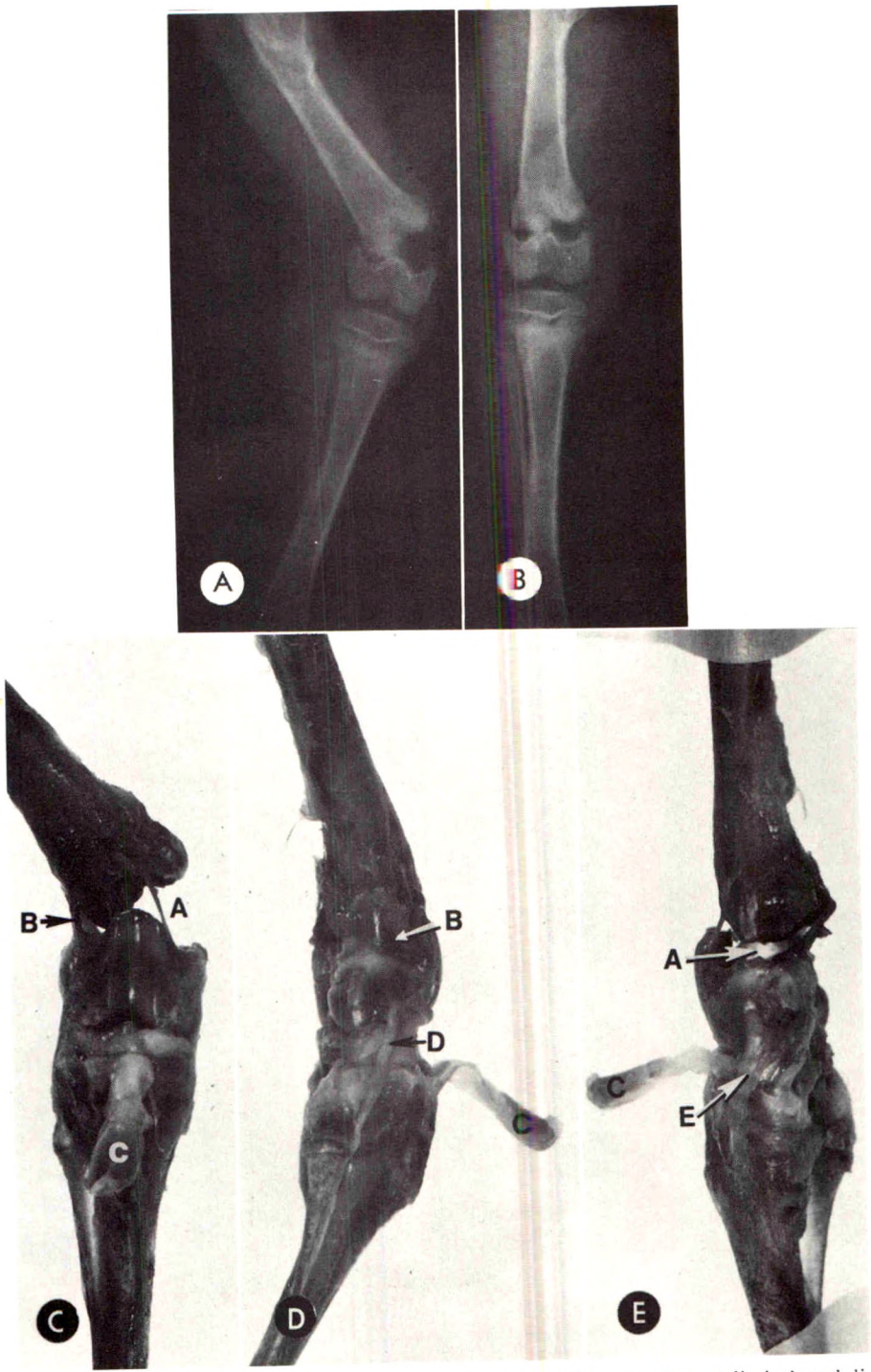


Fig. 7. (*A*) Anteroposterior and (*B*) valgus stress roentgenograms of an experimentally induced distal femoral separation in a young rabbit. A valgus force was applied. (*C*) Anatomic specimen, frontal view. Note fractured distal femoral growth plate (A) and remaining shreds of intact periosteum (B). The patellar tendon is reflected anteriorly (C). (*D*) Anatomic specimen, lateral view. The patellar tendon (C) is reflected anteriorly. The remaining intact periosteum (B) obscures the fractured growth plate. The lateral collateral ligament is intact (D). (*E*) Anatomic specimen, medial view. The patellar tendon (C) is reflected anteriorly. The fractured distal femoral growth plate (A) is evident. The medial collateral ligament (E) remains intact. Note that this ligament originates on the femoral epiphysis distal to the growth plate.

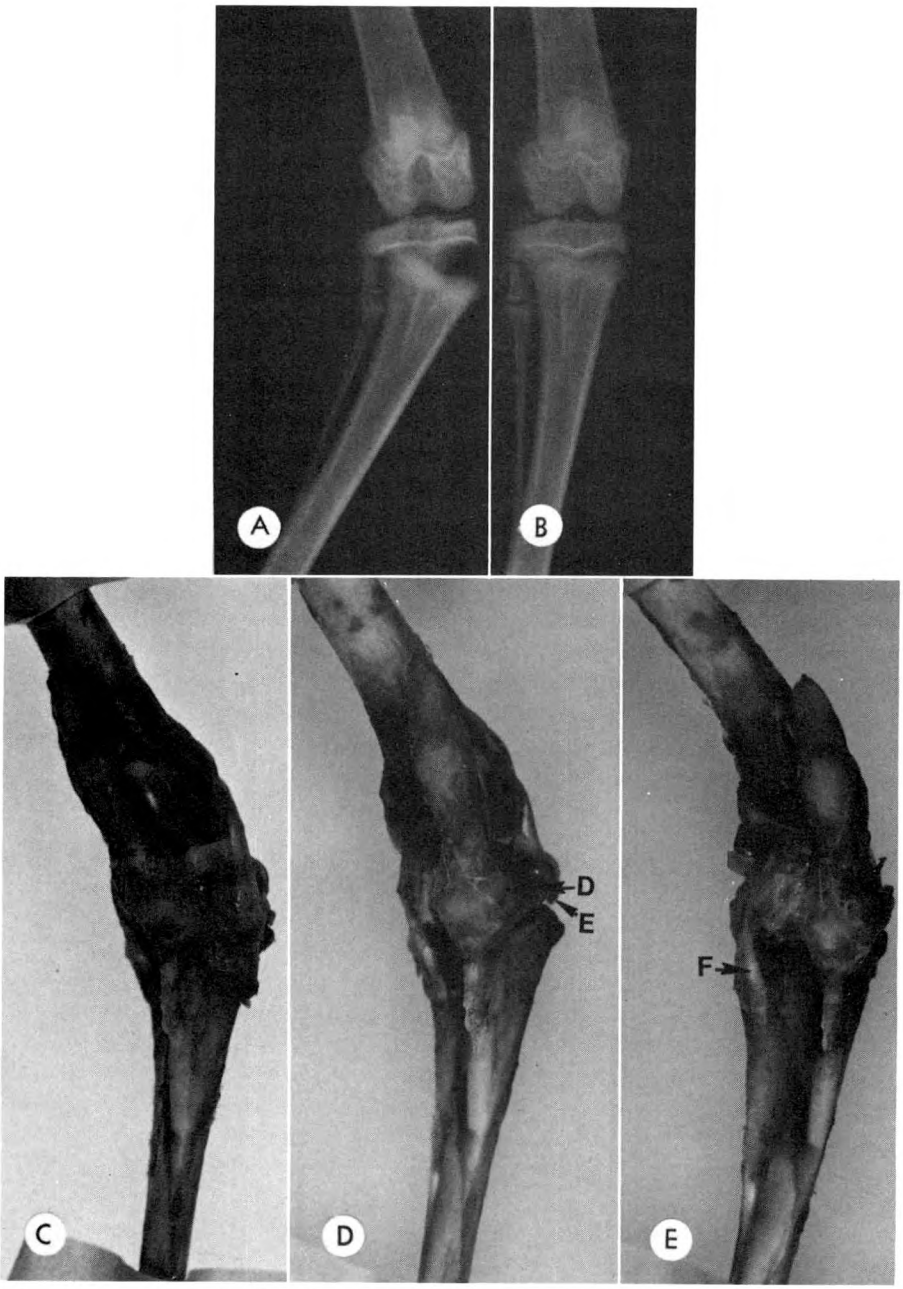


FIG. 8. (*A*) Anteroposterior and (*B*) valgus stress roentgenograms of an experimentally induced proximal tibial epiphyseal separation in a young rabbit. A valgus force was applied. (*C*) Anatomic specimen, frontal view, with slight external rotation. The fracture is not evident. (*D*) Anatomic specimen, frontal view. The periosteal insertion of the medial collateral ligament (D) is reflected superiorly, revealing the fracture of the proximal tibial epiphysis (E). (*E*) Anatomic specimen, frontal view, with slight internal rotation. The lateral collateral ligament (F) is intact.

medial and lateral surfaces of the distal femoral epiphysis. Note that this is distal to the growth plate. The distal site of attachment of the medial collateral ligament is on the medial metaphyseal surface of the tibia, *i.e.*, distal to the proximal tibial growth plate and epiphysis. Similarly, the lateral collateral ligament inserts on the lateral metaphyseal surface of the fibula distal to the proximal fibular epiphysis and growth plate.

Experimental injuries were created in 5 young rabbit rear leg specimens. These were subjected to valgus stresses by holding the thigh in one hand, the lower leg in the other, and pressing against the lateral aspect of the knee with the thumb. In each case an epiphyseal separation, Type 1 or 2, was produced, 2 of the distal femoral and 3 of the proximal tibial epiphysis. These injuries were confirmed by roentgenograms (Fig. 7, *A* and *B*; and 8, *A* and *B*) and anatomic dissection (Fig. 7, *C–E*; and 8, *C–E*). The collateral and cruciate ligaments remained intact in every case. The origin of the medial collateral ligament was distal to the fracture line (Fig. 7*E*) in both cases of separation of the distal femoral epiphysis.

The proximal tibial epiphysis is much less frequently fractured than its counterpart, the distal femoral epiphysis. This is believed to be due to its freedom from collateral ligament attachments.[1] The lateral collateral ligament inserts on the head of the fibula and has no tibial attachment (Fig. 7*D*; and 8*E*). The medial collateral ligament is attached only to a minor extent to the proximal tibial epiphysis but is broadly attached to the tibial metaphysis distal to the growth plate (Fig. 8, *C* and *D*).[2] In the experimentally produced separations of the proximal tibial epiphysis, dissection disclosed that this broad metaphyseal insertion of the medial collateral ligament was stripped free up to the fracture line in the growth plate, but was otherwise intact (Fig. 8, *C* and *D*). This same description was given by Welch and Wynne[13] in reporting a case of proximal tibial epiphyseal separation in a 16 year old boy who had sustained a clipping injury. In this case a medial collateral ligament rupture was suspected and the patient was subjected to operation only to find the ligament intact and the presence of the epiphyseal separation.

The ligaments of the knee, therefore, are generally stronger than the growth plates of the distal femoral and proximal tibial epiphyses.[4,8] This statement was made as early as 1894 by Jonathan Hutchinson,[5] an English surgeon. Our own clinical and experimental observations certainly support this thesis.

SUMMARY

The physician should be alert to the possibility of an underlying epiphyseal injury in an adolescent when a ligamentous injury of the knee is suspected clinically. This is especially true when a valgus force was sustained, as in a clipping injury in football, and a rupture of the medial collateral ligament is suspected. Under these circumstances, if the standard anteroposterior and lateral projections of the knee are unrevealing, oblique and notch views should be obtained. If these are also unrevealing, a valgus stress examination should be performed.

This report is based upon 8 cases, 7 involving the distal femoral and 1 the proximal tibial epiphyses.

Anatomic dissections were performed and experimental injuries were produced in rabbit specimens to determine the anatomic relationships between the growth plate, ligaments, and the epiphyseal fractures.

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REFERENCES

1. AITKEN, A. P. Fractures of proximal tibial epiphyseal cartilage. *Clin. Orthop.*, 1965, *41*, 92–97.
2. BRANTIGAN, O. C., and VOSHELL, A. F. Tibial collateral ligament: its function, its bursae, and its relation to medial meniscus. *J. Bone & Joint Surg.*, 1943, *25*, 121–131.
3. CASSEBAUM, W. H., and PATTERSON, A. H. Fractures of distal femoral epiphysis. *Clin. Orthop.*, 1965, *41*, 79–91.
4. HARSHA, W. N. Effects of trauma upon epiphyses. *Clin. Orthop.*, 1957, *10*, 140–147.
5. HUTCHINSON, J. Lectures on injuries to epiphyses and their results. *Brit. M. J.*, 1894, *1*, 669–673.
6. LARSON, R. L. Epiphyseal injuries in adolescent athlete. *Orthop. Clin. North America*, 1973, *4*, 839–851.
7. O'DONOGHUE, D. H. Treatment of Injuries to Athletes. Second edition. W. B. Saunders Company, Philadelphia, 1970.
8. ROGERS, L. F. Radiography of epiphyseal injuries. *Radiology*, 1970, *96*, 289–299.
9. SALTER, R. B., and HARRIS, W. R. Injuries involving epiphyseal plate. *J. Bone & Joint Surg.*, 1963, *45-A*, 587–622.
10. SMILLIE, I. S. Injuries of the Knee Joint. Fourth edition. E. & S. Livingstone, Ltd., Edinburgh, 1970.
11. SMITH, L. Concealed injury to knee. *J. Bone & Joint Surg.*, 1962, *44-A*, 1659–1660.
12. STEWART, M. J. Unusual Athletic Injuries. A.A.O.S. Instructional Course Lecture, 1960, 377–391.
13. WELCH, P. H., and WYNNE, G. F., JR. Proximal tibial epiphyseal fracture separation. *J. Bone & Joint Surg.*, 1963, *45-A*, 782–784.

ARTHROGRAPHY OF THE LATERAL MENISCUS*

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ARTHROGRAPHY of the knee is a highly accurate diagnostic modality of value in a wide range of clinical disorders of the knee. Its diagnostic accuracy in medial meniscal tears is greater than 90 per cent.[3,6,11,15] Many orthopedic surgeons now depend preoperatively on the arthrographic findings, a situation clinically analogous to myelography.

Lateral meniscal injuries have generally been subordinate to medial meniscal injuries which are 3 to 20 times more common.[17] (The lower figure was derived from athletic injuries, the higher figures are from miners.) In a mixed series the average is 8 to 1.[17] Smillie[18] refers to the "silence" of the lateral compartment of the knee, as large lateral tears may be asymptomatic or refer symptoms to the medial joint space. Compounding the problem, lateral tears often accompany medial meniscal injuries with double lesions occurring in 6 to 12 per cent[3,12] of cases.

A brief review of the roentgen anatomy and pathology of the lateral meniscus visualized by double contrast arthrography will be discussed. All the data and illustrations are derived from over 700 knee arthrographies performed at Thomas Jefferson University Hospital during the past 2 years.

METHOD

Our technique[4] is very similar to the fluoroscopic method described in detail by Butt and McIntyre in 1969.[3] After thorough preparation and evacuation of all joint fluid, 20 cc. of air, 5 cc. of renografin 60 per cent and 30 to 60 cc. of medical grade carbon dioxide are injected into the knee joint through a lateral approach. The joint distention gives excellent visualization and the rapid carbon dioxide resorption decreases post arthrographic pain.[1,3,4]

Our examinations are all performed on a Siemens Siregraphic remote controlled unit and hence 2 people are required; 1 operates the fluoroscopic control panel and changes films, while the other stresses the knee under television control. We have designed a canvas restraining device to facilitate stressing of the knee (Fig. 1, *A* and *B*). This device is more rigid than cloth, easily made and inexpensive. Velcro strips are sewn to an 8 inch strip of No. 128 Barco-Dex white canvas which is attached to a flange that fits over the fluoroscopic table. At least 8 spot film roentgenograms of the lateral meniscus are taken (Fig. 2, *A–H*). The average factors are: 60 kv., 200 ma. for 0.25 seconds with a 0.6 mm. focal spot. Various authors recommend a focal spot size of 0.3 mm.[3] to 1 mm. but we have found the 0.6 mm. focal spot satisfactory.

ANATOMY

The lateral meniscus is nearly circular in configuration and covers a larger portion of the tibial surface than the medial meniscus. Its surfaces are slightly concave and it is triangular in cross section, changing only slightly in size from the anterior to the posterior horn (Fig. 2, *A–H*). Its average roentgenographic width is 10 mm.[8] A large narrow recess is frequently present at the undersurface of its anterior horn and midportion (Fig. 2, *A–D*).

The anterior horn of the lateral meniscus is attached to the anterior intercondylar area of the tibia, behind and lateral to the anterior cruciate ligament. The posterior horn inserts into the intercondylar fossa,

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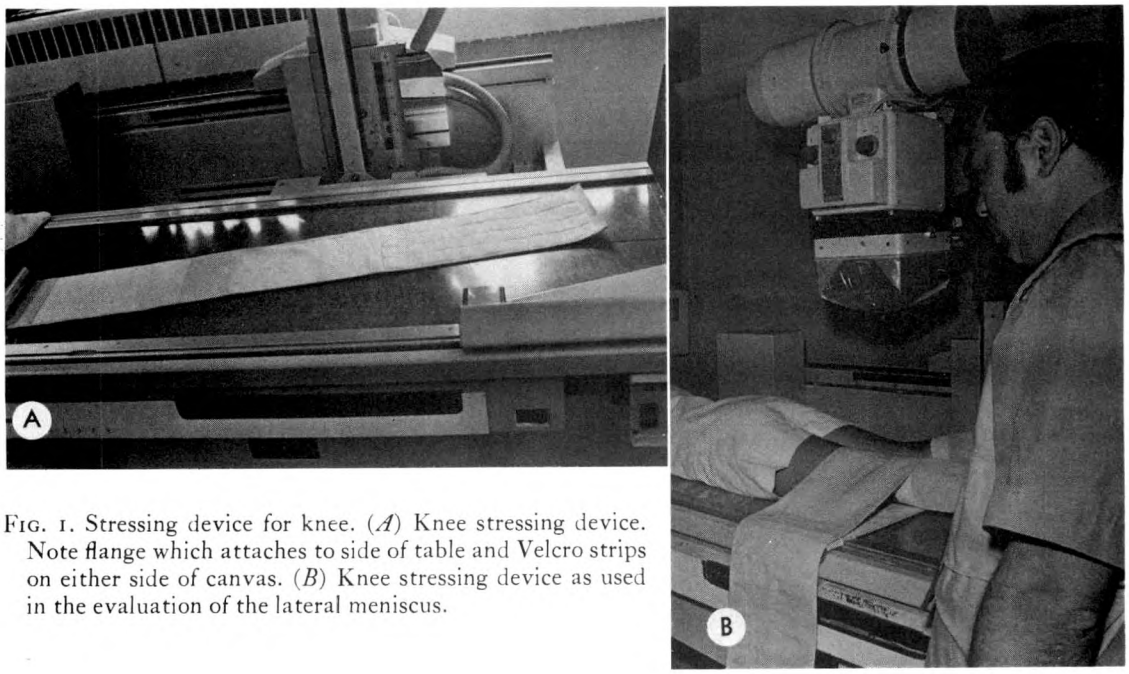


FIG. 1. Stressing device for knee. (*A*) Knee stressing device. Note flange which attaches to side of table and Velcro strips on either side of canvas. (*B*) Knee stressing device as used in the evaluation of the lateral meniscus.

anterior to the medial meniscus. The posterior horn lacks an attachment where the tendon sheath of the popliteus muscle communicates with the joint space posteriorly. Two synovial struts attach the meniscus to the capsule, one above and the other below the muscle (Fig. 2, *G* and *H*).[13] The meniscal femoral ligaments run from the posterior horn of the meniscus to the medial femoral condyle. The fibers are anterior (ligament of Humphry) and posterior (ligament of Wrisberg) to the attachment of the posterior cruciate ligament. The loose attachments posteriorly are responsible for the lateral meniscal mobility.

RESULTS

In the last 2 years we have performed over 700 double contrast knee arthrographies at Thomas Jefferson University Hospital. There were 148 confirmed medial and 53 lateral meniscal tears in 183 patients, 18 of whom had bilateral tears.

Forty-eight of the lateral meniscal tears were diagnosed arthrographically—an accuracy of greater than 90 per cent. There were 7 peripheral tears (Fig. 3) and 7 tears associated with degenerative menisci (Fig. 4) in this group. There were 2 torn discoid menisci (Fig. 5), 1 of which was also cystic, and an additional cystic meniscus (Fig. 6). Four patients had previously undergone medial menisectomy. Of the 5 false negatives, 4 could not be seen on retrospect; 3 of these were sharp horizontal tears and the other was associated with a large popliteal cyst. One patient had a small anterior horn tear which was identified on review of the roentgenograms.

DISCUSSION

The anatomy of the lateral meniscus is more complex than the medial meniscus. Its arthrographic appearance, with particular attention to its posterior horn, was described in detail by McIntyre in 1972.[13] Some authors have had difficulty in diagnosing lateral meniscal tears *via* arthrography.[9,20] Our accuracy was greater than 90 per cent and there were no false positives. The diagnosis of internal derangement of the knee on the basis of history and physical examination is often difficult, particularly with respect to the lateral joint compartment; in 1 orthopedic series it was 54 per cent.[18]

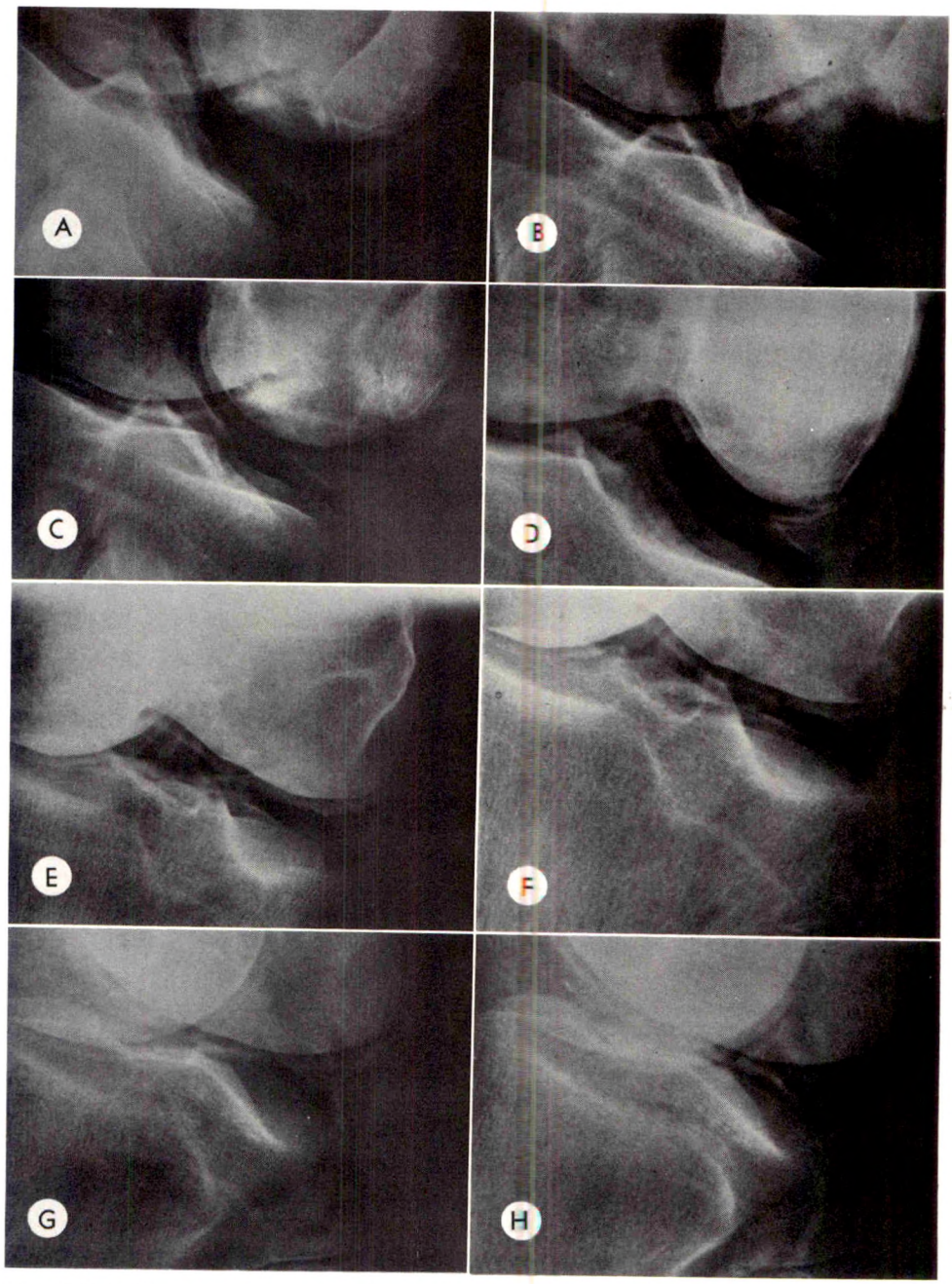


Fig. 2. (*A–H*) Spot film roentgenograms of normal lateral meniscus from anterior to posterior horn. Note that the meniscus size is approximately constant from anterior to posterior horn. A large recess is seen anteroinferiorly (*A–D*). Synovial attachment struts about the popliteus tendon are identified posteriorly (*E* and *F*).



FIG. 3. Peripheral detachment, posterior horn lateral meniscus. Note absence of superior attachment strut and lack of mobility which is inferred by the failure of upward and central movement with stress. (Compare to Figure 2, *E* and *F*.)



FIG. 5. Torn discoid meniscus. The lateral meniscus is large, extending almost to the intercondylar notch. A tear is evident.

In a total of 53 lateral tears there were 18 patients who had concomitant medial tears and 4 additional patients who had undergone previous medial menisectomies. There were double tears in 18 of 183 patients. Peripheral lateral tears were very frequent in McIntyre's series and accounted for all of his lateral tears, excluding cystic and discoid menisci.[13] In his series lateral meniscal pathology was as frequent as medial meniscal tears. We have had a number of arthrographically diagnosed peripheral tears that became pain free on conservative therapy. These patients were excluded from our series as they were not surgically confirmed. Only 7 lateral tears were truly peripheral tears: often the peripheral attachments were torn, but the tear was extensive involving the entire meniscus.[4]

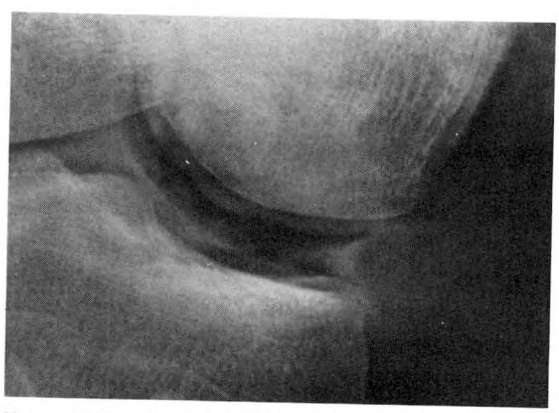


FIG. 4. Torn degenerated lateral meniscus. The undersurface of the meniscus is irregular and unsharp. It appears more opaque than normal secondary to contrast medium within the small horizontal tears. The femoral articular cartilage is irregular.



FIG. 6. Torn degenerated meniscus with cyst formation. The lateral meniscus has an irregular shape with increased opacity secondary to extensive degeneration with horizontal tears. The arrow points to a communicating meniscal cyst.

Lateral tears are occasionally confined to the anterior horn and may present with a locked knee (Fig. 7, *A* and *B*). On occasion, tears are confined to the posterior horn and do not involve its peripheral attachments. Rarely, bucket-handle tears are seen in the lateral meniscus (Fig. 8).

We have had 2 cystic lateral menisci, both with tears. One patient's cystic meniscus was also discoid and the second patient's cystic meniscus was associated with degenerative meniscal changes (Fig. 6). Meniscal cysts are not true cysts as they are lined by synovial cells.[18] They are multilocular and mucinous and are much more frequent laterally[2,16,18] than medially, occurring in 22 per cent of Smillie's lateral menisectomies.[18] They are usually small and peripheral, may communicate with the joint and vary in size on flexion and extension.[2] Their origin is conjectural with developmental,[2] traumatic[18] and degenerative proponents.[16] In our experience they are rarely recognized at arthrography. They are frequently found in association with abnormal menisci and their small size and inconsistent communication with the synovium are probably responsible for the difficulty in arthrographic diagnosis.[2,4]

Two patients had torn discoid menisci and an additional patient, who is not included in the series, had an intact discoid meniscus (Fig. 9). This patient was only 2 years old and is being followed clinically.

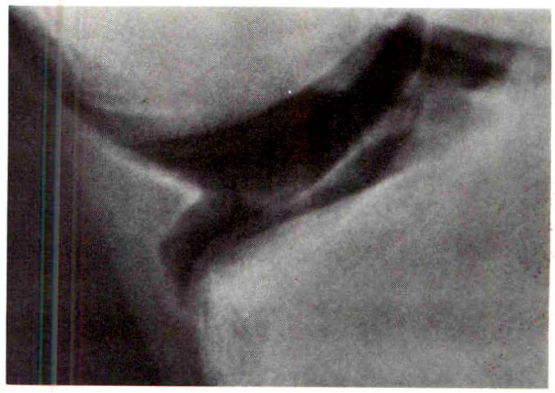


Fig. 8. "Bucket-handle" tear of lateral meniscus.

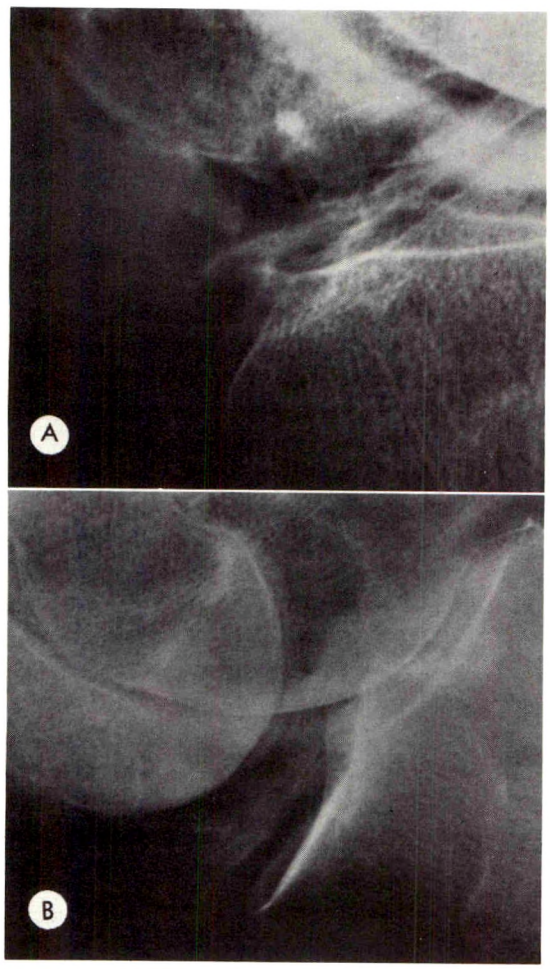


Fig. 7. (*A*) Torn anterior horn, lateral meniscus. Patient presented with "locked knee." (*B*) Linear tear of anterior horn, lateral meniscus, without displacement.

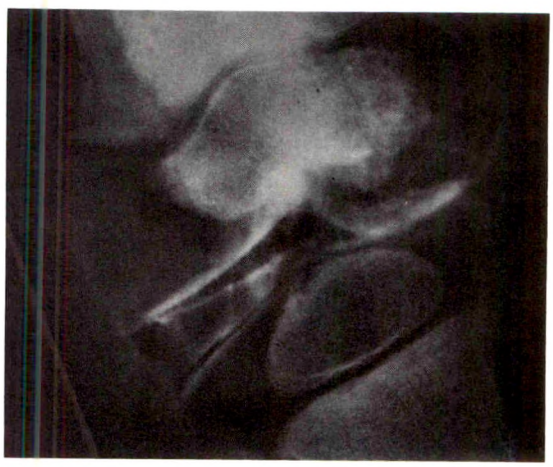


Fig. 9. Intact discoid lateral meniscus in 2 year old patient.

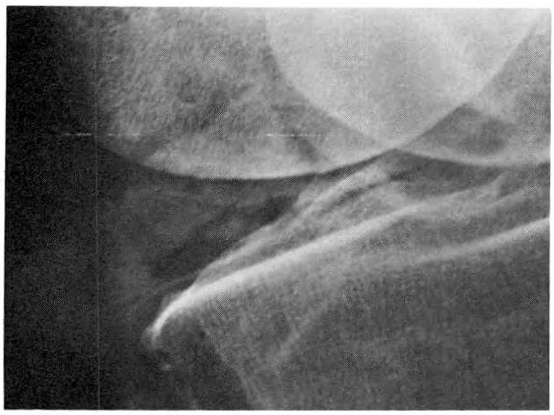


FIG. 10. Large lateral meniscus with irregular configuration secondary to extensive tears.

Discoid menisci are abnormally broad disk-shaped cartilages which almost always occur laterally. They are thought to be related to abnormal posterior attachments of the lateral meniscus.[10,13] Strong meniscal femoral attachments do not permit complete forward movement during extension and pull the meniscus laterally during flexion, causing the characteristic click.[10] This is a developmental rather than congenital abnormality. These thick menisci with impaired mobility are prone to tear and are more common in pediatric series.[14,19] Bilateral[14] and familial[5] cases have been reported. The surgical approach is difficult due to the strong meniscal femoral ligaments and large inferior lateral geniculate arteries.[10] The strong ligaments make removal through an anterior approach difficult and the large arteries predispose to postoperative hemorrhage.[10] The arthrographic diagnosis can be made when the meniscus extends into the intercondylar notch and the body is parallel rather than triangular in shape.[7,13,14,19] As previously stated, we have seen only 2 torn discoid menisci but we have seen a number of cases of large lateral menisci with tears (Fig. 10). The differences between a large lateral meniscus and a discoid meniscus may be subject to interpretation; this, and the small number of children in our series may account for the relatively low incidence of discoid menisci in our material.

We have also seen patients who present with "degenerative joint disease" confined

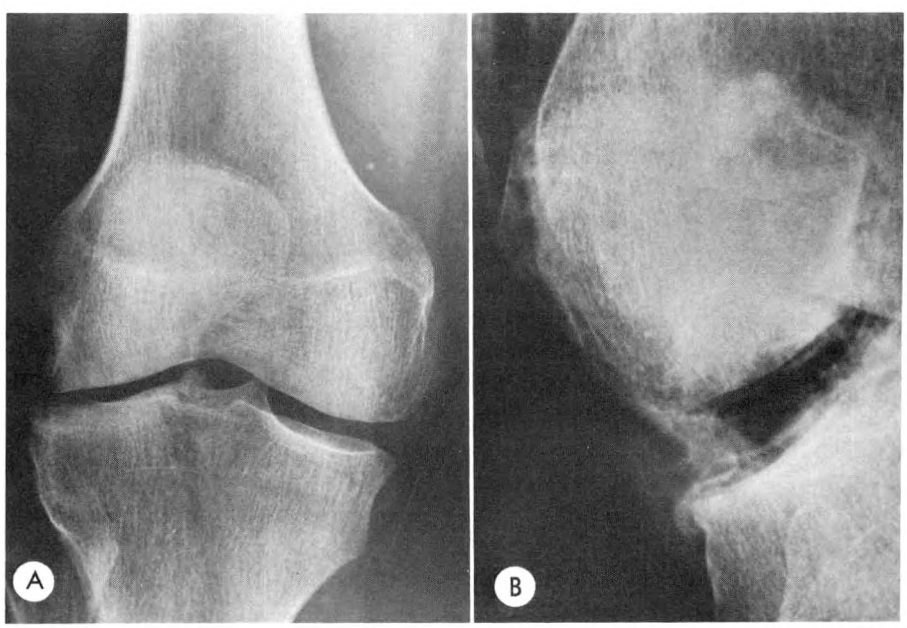


FIG. 11. Lateral meniscal tear presenting as unicompartmental osteoarthritis. (*A*) Posteroanterior roentgenogram of knee revealing narrowing of lateral joint compartment with irregularity of lateral tibial plateau and small marginal osteophytes. (*B*) Arthrogram in same patient revealing abnormal lateral meniscal configuration and thinning of articular cartilage secondary to a torn degenerated meniscus.

to the lateral compartment. On arthrography, a torn degenerative lateral meniscus was seen (Fig. 11, *A* and *B*). We[4] and others[18] believe that an unrecognized meniscal tear may cause unicompartmental osteoarthritis and that arthrography should be performed prior to surgery on these patients.[4] In some cases, menisectomy alone leads to gratifying results.

SUMMARY

Double contrast knee arthrography is very accurate in the diagnosis of lateral meniscal tears.

Forty-eight of our 53 surgically proven lateral meniscal tears were diagnosed arthrographically with no false positives. The frequency of bilateral tears in our series was 10 per cent, but of those with lateral meniscal tears, 18 of 53 had medial tears. Four additional patients had prior medial menisectomies. The high incidence of double tears is significant and for this reason we believe that arthrography is indicated in all patients with presumed "internal derangement of the knee."

It is our belief that routine preoperative arthrography will aid in the diagnosis of the silent lateral tears and perhaps prevent the occurrence of unicompartmental osteoarthritis in these patients.

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REFERENCES

1. Angell, F. L. Fluoroscopic technique of double contrast arthrography of knee. *Radiol. Clin. North America*, 1971, *9*, 85–98.
2. Burgan, D. W. Arthrographic findings in meniscal cysts. *Radiology*, 1971, *101*, 579–581.
3. Butt, W. P., and McIntyre, J. L. Double contrast arthrography of knee. *Radiology*, 1969, *92*, 487–489.
4. Dalinka, M. K., Coren, G., and Wershba, M. Knee arthrography. *Crit. Rev. Rad.*, 1973, *4*, 1–59.
5. Dashefsky, J. H. Discoid lateral meniscus in three members of family. *J. Bone & Joint Surg.*, 1971, *53-A*, 1208–1210.
6. Freiberger, R. H., Killoran, P. J., and Cardona, G. Arthrography of knee by double contrast method. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *97*, 736–747.
7. Haverson, S. B., and Rein, B. J. Lateral discoid meniscus of knee: arthrographic diagnosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 581–586.
8. Heiser, S., La Briola, J. H., and Meyers, M. H. Arthrography of knee. *Radiology*, 1962, *79*, 822–828.
9. Jelaso, D. V. Positive contrast arthrography of knee. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 669–673.
10. Kaplan, E. B. Discoid lateral meniscus of knee joint—nature, mechanism and operative treatment. *J. Bone & Joint Surg.*, 1957, *39-A*, 77–87.
11. Lindblom, K. Arthrography of knee: roentgenographic and anatomic study. *Acta radiol.*, 1948, Suppl. 74, pp. 1–112.
12. McBeath, A. A., and Wirts, H. W. Positive contrast arthrography of knee—report of experience with 311 arthrograms. *Clin. Orthop.*, 1972, *88*, 70–75.
13. McIntyre, J. L. Arthrography of lateral meniscus. *Radiology*, 1972, *105*, 531–536.
14. Moes, C. A., and Munn, J. D. Value of knee arthrography in children. *J. Canad. A. Radiologists*, 1965, *16*, 226–233.
15. Nicholas, J. A., Freiberger, R. H., and Killoran, P. J. Double contrast arthrography of knee: its value in management of 225 knee derangements. *J. Bone & Joint Surg.*, 1970, *52-A*, 203–220.
16. Raine, G. E. T., and Gonet, L. C. L. Cysts of menisci of knee. *Postgrad. M. J.*, 1972, *48*, 49–51.
17. Ricklin, P., Ruttimann, A., and Del Buono, M. S. American Translation, K. Muller, Meniscal Lesions—Practical Problems of Clinical Diagnosis, Arthrography and Therapy. Grune & Stratton, Inc., New York, 1971.
18. Smillie, I. S. Injuries of the Knee Joint. Fourth edition. J. A. Churchill, Ltd., London, E. & S. Livingstone, Edinburgh, and Williams & Wilkins Company, Baltimore, 1971.
19. Stenstrom, R. Arthrography of knee joint in children: roentgenologic anatomy, diagnosis and use of multiple discriminant analysis. *Acta radiol.*, 1968, Suppl. 281.
20. Turner, V. C., and Wurtz, F. B. Arthrography in diagnosis of meniscal injuries of knee: correlation of roentgenographic, clinical and operative findings. *J. Bone & Joint Surg.*, 1959, *41-A*, 1213–1220.

SYNOVIAL RECESSES IN KNEE ARTHROGRAPHY

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PREOPERATIVE assessment of knee joint anatomy and pathology is being increasingly sought by the orthopedic surgeon. Knee arthrography, primarily as a fluoroscopic procedure, has afforded the radiologist with improved technical features in the study. Most pathology is recognized by image intensification fluoroscopy and recorded by fluoroscopic spot filming. The ability of the fluoroscopist to move the tibial plateau perpendicular to the screen at will appears to be the greatest single advantage over the "blind" non-fluoroscopic methods.

Knee arthrographic anatomy and pathology have been well documented.[3,4] Normal synovial recesses are well known and described on occasion in arthrographic reports. The normal recesses have produced some confusing situations in arthrographic interpretation. The recess appearance of traumatic partial separation of the medial meniscus from the joint capsule has also been noted.

The recesses in 100 consecutive examinations performed in a 14 month period of 1971–1972 in a private radiology office were analyzed. These examinations closely

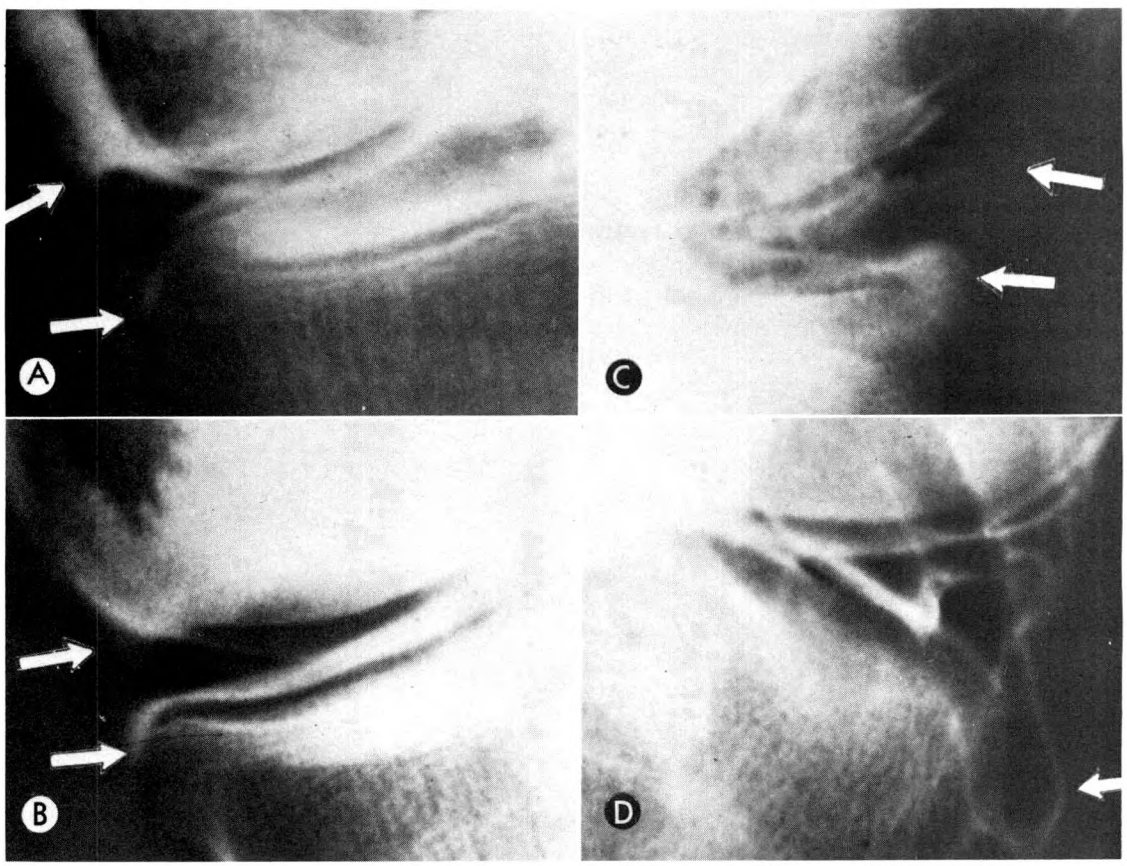


FIG. 1. Normal synovial recesses. Occurrence in this study in parentheses. (*A*) Medial anterior, superior (11 per cent), inferior (91 per cent). (*B*) Medial posterior, superior (32 per cent), inferior (10 per cent). (*C*) Lateral anterior, superior (4 per cent), inferior (98 per cent). (*D*) Lateral posterior, inferior (98 per cent).

followed the technique described by Angell,[1,2] but utilized both prone and supine positions.

NORMAL RECESSES

The normal synovial recesses have the arthrographic appearance in any one projection of a pouch, projecting beyond the adjacent femoral or tibial articular margin. The recess or pouch is better described as a trough which extends a variable distance about the periphery of the medial or lateral portion of the joint.

There is considerable individual variation with no age or sex correlation in size, shape, presence or absence of the synovial recesses (Fig. 1, *A–D*). The tibial or inferior recesses are more frequent. The inferior recess on the lateral side was seen most uniformly (98 per cent), both anteriorly and posteriorly. The anterior component of the inferior lateral recess is often more mobile so that a bilobular appearance is obtained when well distended, giving both cephalad

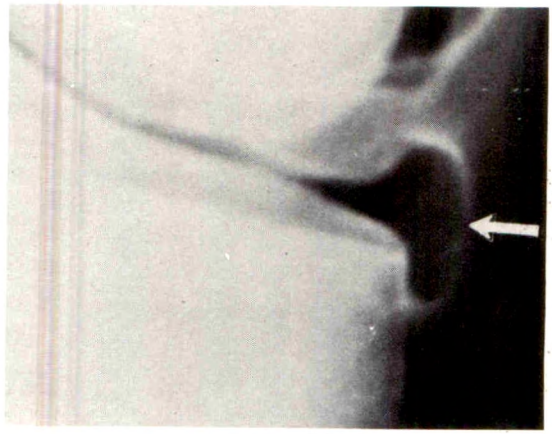


Fig. 2. Normal synovial recess. Half of the lateral anterior inferior recesses in this study assumed this bilobular configuration.

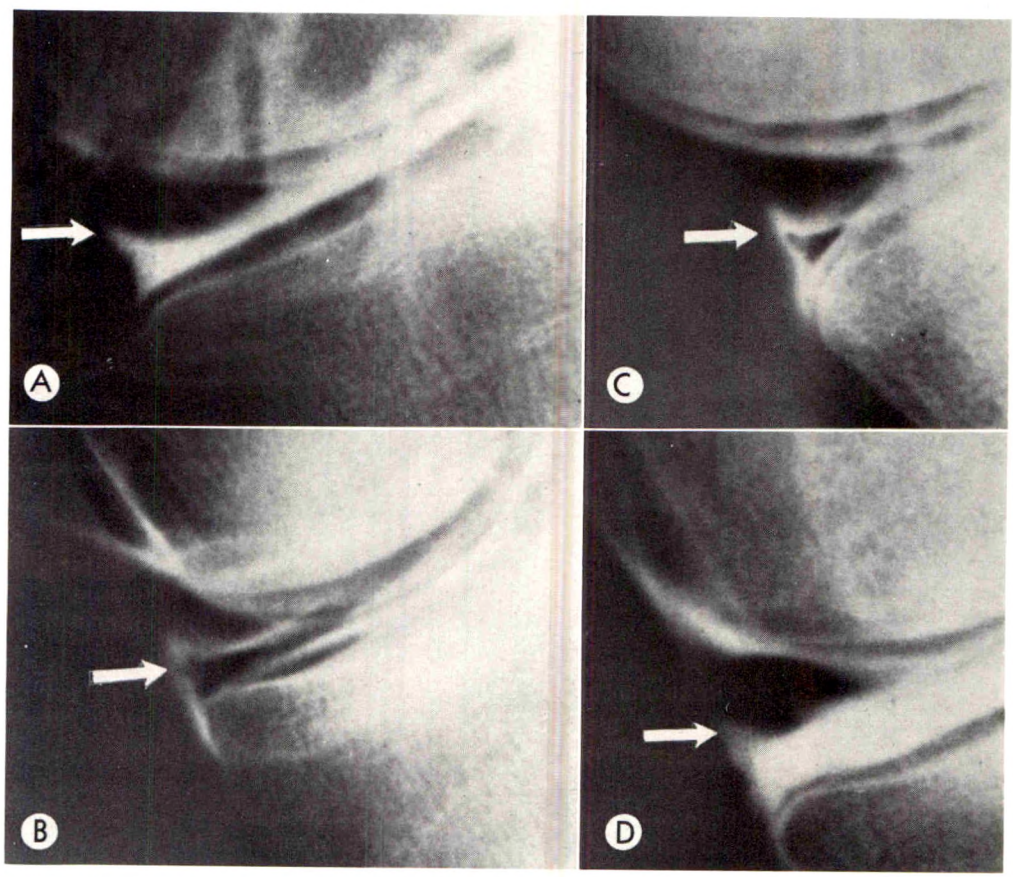


Fig. 3. Abnormal synovial recesses. (*A–D*) Each of these 4 medial posterior pseudorecesses were shown at surgery to be of traumatic nature. The meniscus tear is evident in *D*.

and caudad directions to the distended recess (Fig. 2). The inferior recess on the medial side is near constant anteriorly (91 per cent), but was seen posteriorly in only 10 per cent.

Superior recesses are more infrequent. The medial superior recesses, although appearing to extend to a site between the upper convex meniscus margin and joint capsule, were not correlated with traumatic pathology as is the abnormal medial cephalic directed inferior recess described below.

ABNORMAL RECESSES

Traumatic separation of the convex margin of the medial meniscus from the joint capsule produces a collection of contrast medium having the appearance of a cephalic directed inferior recess in many cases. This inferior capsule separation seen adjacent to the posterior horn of the medial meniscus was present in some degree in 30 per cent of the cases in this study, its frequency leading to this configuration being called a pseudorecess in our practice. Nearly one-half of these separations appear to have healed with conservative management. Of 18 operated patients having a posterior medial pseudorecess, 14 had associated meniscus tears.

In the acute stage the posterior medial pseudorecess undoubtedly represents an abnormal cavity in the loosened fibrous union of the capsule and meniscus, but with chronicity the possibility of synovial proliferation into the traumatically produced space can be considered. Anatomy texts describe an inconsistent bursa between the medial collateral ligament and medial meniscus, but presence of such has been considered by arthrographers to be pathologic for many years.[5] Representative traumatic abnormal inferior posterior medial recesses are shown in Figure 3, *A–D*.

SUMMARY

The occurrence of normal synovial recesses and the frequency of abnormal traumatic recesses in a small arthrographic series are recorded.

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REFERENCES

1. ANGELL, F. L. Fluoroscopic technique of double contrast arthrography of knee. *Radiol. Clin. North America*, 1971, *9*, 85–98.
2. ANGELL, F. L. Restraint device for arthrography of knee. *Radiology*, 1971, *98*, 186–187.
3. BUTT, W. P., and MCINTYRE, J. L. Double contrast arthrography of knee. *Radiology*, 1969, *92*, 487–499.
4. FREIBERGER, R. H., KILLORAN, P. J., and CARDONA, G. Arthrography of knee by double contrast method. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 736–747.
5. OLSON, R. W. Knee arthrography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *101*, 897–914.

ARTHROGRAPHY IN RUPTURE OF THE SUPRAPATELLAR BURSA WITH PSEUDOCYST FORMATION*

By ADAH M. DUNCAN, M.D.
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ARTHROGRAPHY of the knee joint has been used to demonstrate meniscal and ligamental tears,[5,7,11,12] meniscal cysts,[2] Baker's cysts,[8,9] tears of the cruciate ligaments,[10] and masses within the joint space, including loose bodies, pigmented villi, nodular synovitis,[15] lipoma arborescens,[1] synovial chondromatosis,[3] as well as changes due to osteoarthritis of hemophilia.[13]

This paper presents 3 cases of rupture of the suprapatellar bursa (one causing a cyst-like mass in the thigh) proven by arthrography with discussion of the possible etiology.

The technique of knee arthrography used is based on work by Turner and Budin.[14] It is as follows: 20 cc. renografin 60 is instilled into the knee joint employing the lateral suprapatellar approach. Knee joint effusion, if present, is removed and the opaque medium is then injected under fluoroscopic control. The knee is wrapped in an elastic bandage, and flexion and extension exercise is carried out 25 times. Following this, 9 spot roentgenograms of each meniscal horn are obtained in varying degrees of obliquity. Overhead roentgenograms in the postero-anterior, lateral and oblique positions in flexion are taken. A normal lateral view of the knee is shown in Figure 1.

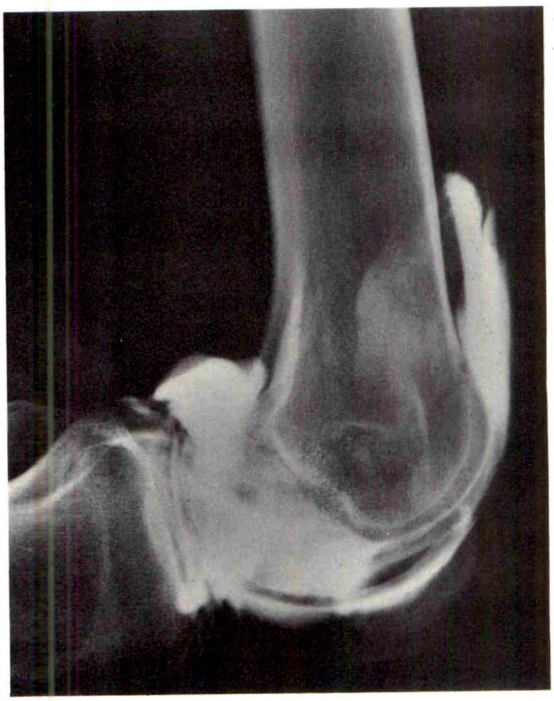


Fig. 1. Normal lateral view of the knee in flexion.

REPORT OF CASES

Case 1. A 49 year old physician, a tennis player, presented with painful swelling of the right thigh of 3 days' duration, located approximately 10 cm. above the patella. He had experienced pain on exercise of the right knee intermittently for 2 years. On physical examination, with the knee extended, a soft, ill defined swelling was identified along the medial distal aspect of the quadriceps muscle approximately 10 cm. above the patella. Arthrography was carried out demonstrating a large tear of the posterior horn of the medial meniscus (Fig. 2). In addition, a tract measuring 3.5 cm. was seen to extend from the top of the suprapatellar bursa to a 5 by 2 cm. "cyst-like space" filled with contrast medium and communicating with the knee joint (Fig. 3; and 4). Anterior to the cystic space, contrast material extended into the muscular plains of the thigh (Fig. 5). Twenty cubic centimeters of serous fluid was removed, the subsequent laboratory analysis demonstrating aseptic fluid. The patient was operated on and a torn medial meniscus removed. The suprapatellar bursa was explored and an area of inflammation and hemosiderin deposit was identified within the vastus medialis and vastus lateralis. The postoperative

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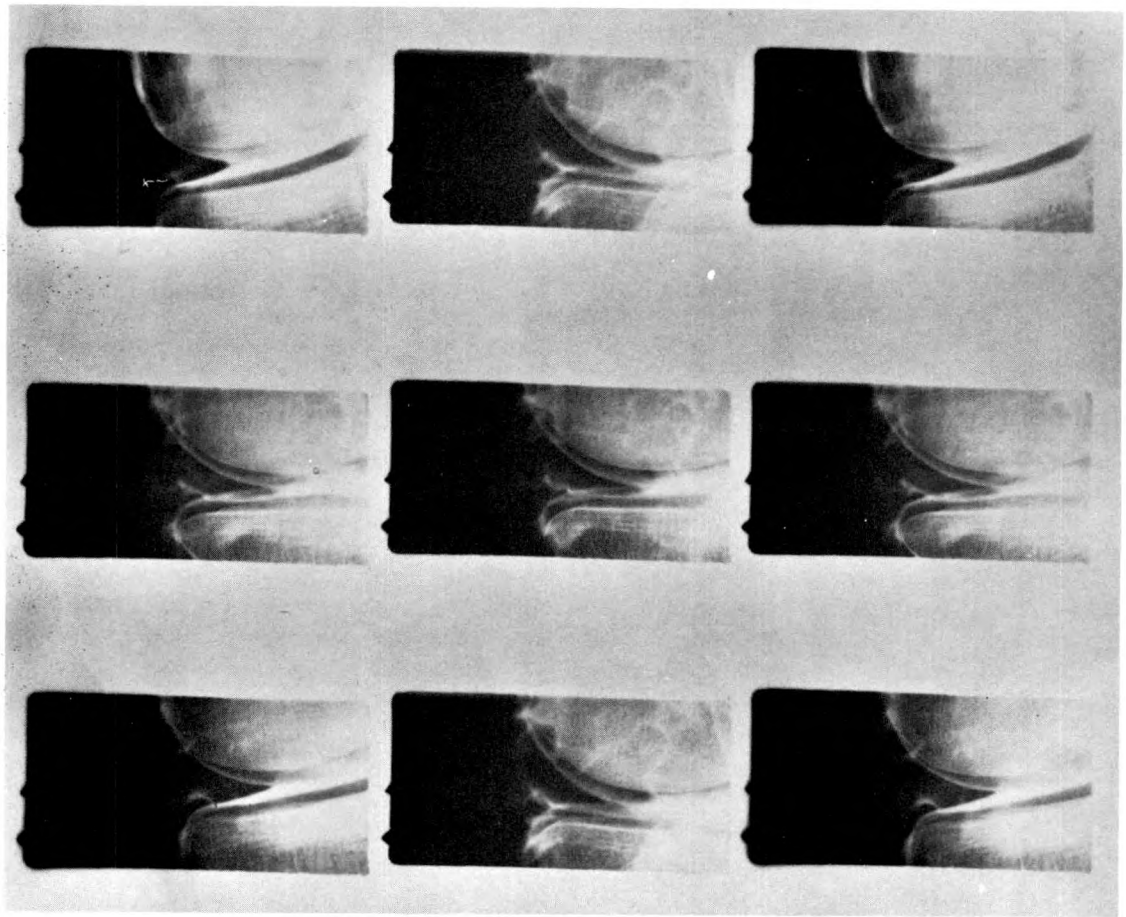


Fig. 2. Case 1. Tear of the medial meniscus, posterior horn.

course was complicated by a hematoma of the right thigh, which was surgically evacuated, and the patient has since done well. Pathologic examination of the tissue taken from the cyst-like area demonstrates acute and chronic inflammation.

Case II. A 25 year old secretary with no previous history of injury, 1 week prior to the arthrography struck her knee on a desk. There was moderate swelling and pain in the left knee and thigh. An arthrogram was obtained demonstrating contrast medium extending above the suprapatellar bursa (Fig. 6). No cyst-like space was seen. No operation was performed.

Case III. A 65 year old woman had a previous cerebral vascular accident and poliomyelitis, which limited the use of the left leg, and produced equinous deformity of the left foot. Ten days prior to the examination there were 2 falls with pain, instability and swelling of the left knee joint. Arthrography was carried out demonstrating contrast material communicating with the suprapatellar bursa and located in the distal thigh (Fig. 7).

DISCUSSION

Suprapatellar effusion[6] and popliteal cysts[8] (Fig. 8) are common findings in internal derangement of the knee. Less frequently seen is the calf cyst of rheumatoid arthritis[4] (Fig. 9). Rupture of the suprapatellar bursa with or without pseudocyst formation may occur secondary to increased intracapsular pressure. The increased intracapsular pressure may be due to chronic knee joint effusion with or with-

out wrapping of the knee with an ace bandage and exercise, or acute increased intracapsular pressure due to injection of 20 cc. of contrast material, wrapping the knee joint with an ace bandage and exercise. In this regard the weakest point of the knee joint has been shown by manometric studies to be the apex of the suprapatellar pouch.[4] Following repair of the internal derangement of the knee, resolution of the suprapatellar pseudocyst would be expected to occur similar to that which can be seen with Baker's cyst.

SUMMARY

Three cases of rupture of the suprapatellar bursa are described.

In one case a pseudocyst formed and presented as a mass in the thigh. The other two cases represent rupture occurring during arthrography.

In the interpretation of knee arthrograms, special attention to the superior portion of the suprapatellar bursa, with additional roentgenograms of the thigh, if necessary, will no doubt reveal more cases of suprapatellar bursal rupture.

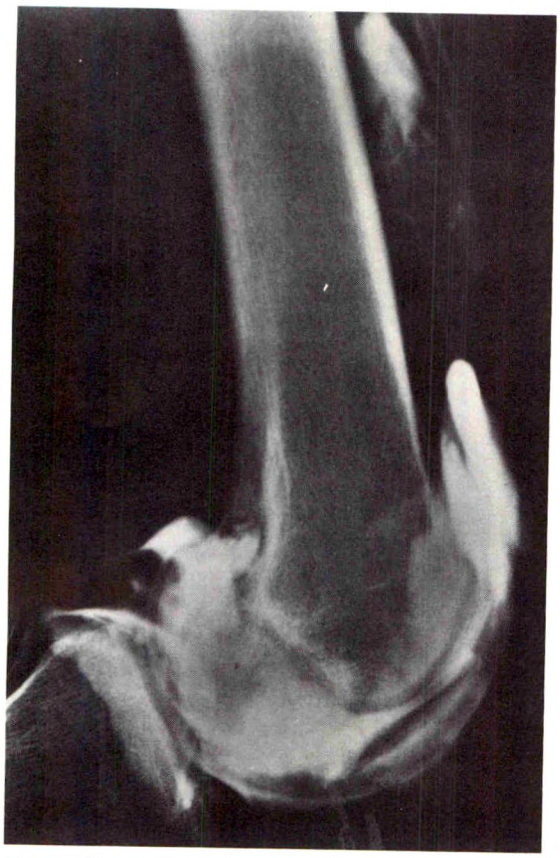


FIG. 3. Case 1. Lateral arthrogram of the knee demonstrating contrast medium filled tract extending from the suprapatellar bursa.

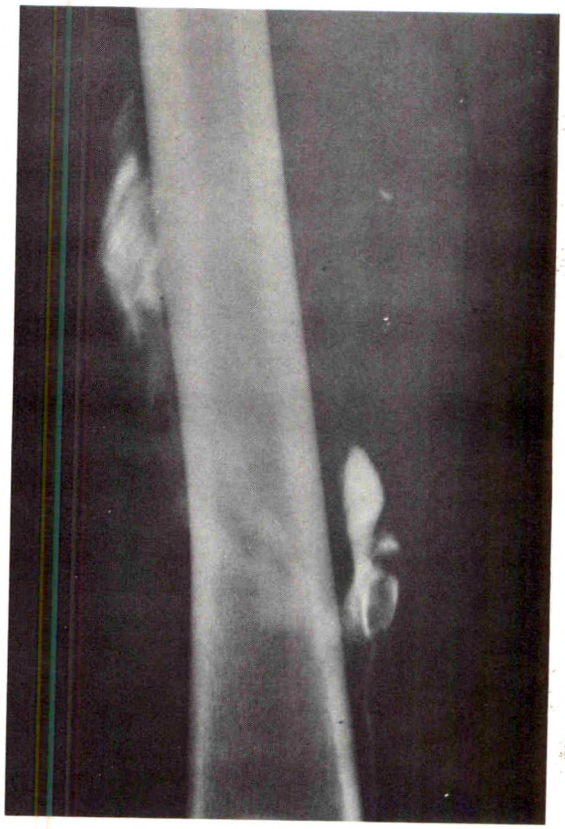


FIG. 4. Case 1. "Cyst-like space" communicating with joint.

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Fig. 6. Case II. Irregular "cyst-like space" arising from the suprapatellar bursa.





Fig. 5. Case I. Contrast material extending from the suprapatellar pseudocyst into the muscular layers of the thigh.

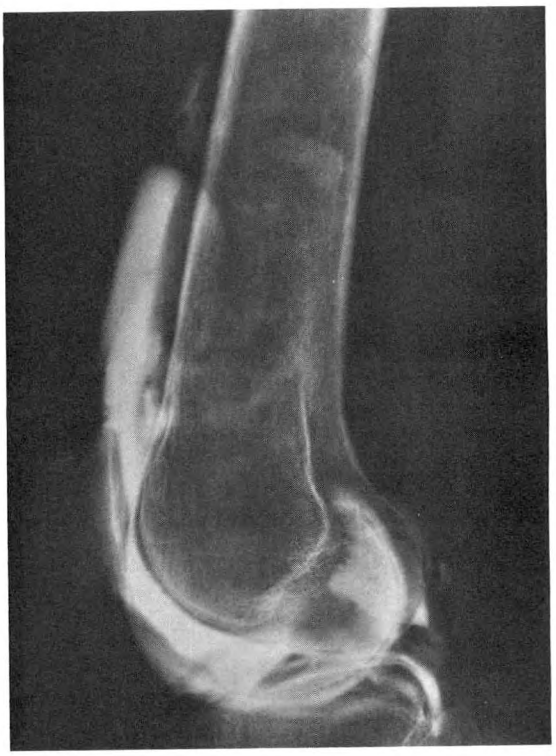


Fig. 7. Case III. Small pseudocyst arising from the top of the suprapatellar bursa.

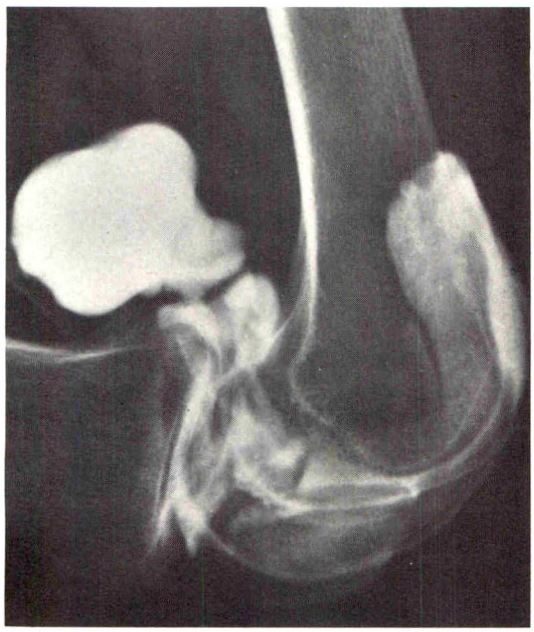


Fig. 8. Lateral view of the knee joint demonstrating popliteal cyst.



Fig. 9. Calf cyst of rheumatoid arthritis.

REFERENCES

1. Burgan, D. W. Lipoma arborescens of knee: another cause of filling defects on knee arthrogram. *Radiology*, 1971, *101*, 583–584.
2. Burgan, D. W. Arthrographic findings in meniscal cysts. *Radiology*, 1971, *101*, 579–581.
3. Crittenden, J. J., Jones, D. M., and Santavelli, A. G. Knee arthrogram in synovial chondromatosis. *Radiology*, 1970, *94*, 133–134.
4. Dixon, A. S., and Grant, C. Acute synovial rupture in rheumatoid arthritis: clinical and experimental observations. *Lancet*, 1964, *1*, 242.
5. Freiberger, R. H., Killoran, P. J., and Cardona, G. Arthrography of knee by double contrast method. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *97*, 736–747.
6. Harris, R. D., and Hecht, H. L. Suprapatellar effusions. *Radiology*, 1970, *97*, 1–4.
7. Jelaso, D. V. Positive contrast arthrography of knee. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 669–673.
8. Lapayowker, M. S., Cliff, M. M., and Tourtellotte, C. D. Arthrography in diagnosis of calf pain. *Radiology*, 1970, *95*, 319–323.
9. Lewin, J. R., and Lawrence, M. M. Lymphatic visualization during contrast arthrography of knee. *Radiology*, 1972, *103*, 577–579.
10. Mitler, S., Freiberger, R. H., and Harrison-Stubbs, M. Method of improving cruciate ligament visualization in double-contrast arthrography. *Radiology*, 1972, *102*, 441–442.
11. Nicholas, J. A., Freiberger, R. H., and Killoran, P. J. Double-contrast arthrography of knee: its value in management of two hundred and twenty-five knee derangements. *J. Bone & Joint Surg.*, 1970, *52-A*, 203–220.
12. Olson, R. W. Knee arthrography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 897–914.
13. Salerno, N. R., Menges, J. F., and Burns, P. F. Arthrograms in hemophilia. *Radiology*, 1972, *102*, 135–138.
14. Turner, A. F., and Budin, E. Arthrography of knee. *Radiology*, 1970, *97*, 505–508.
15. Wolfe, R. D., and Giuliano, V. J. Double-contrast arthrography in diagnosis of pigmented villonodular synovitis of knee. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 793–799.

USE OF XERORADIOGRAPHY IN CRUCIATE LIGAMENT INJURIES*

By HARRY J. GRIFFITHS, M.D., *and* CARL J. D'ORSI, M.D.
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TEARS of the cruciate ligaments constitute about 10 per cent of injuries to the knee. They usually can be diagnosed clinically by the so-called "drawer" sign, but in equivocal cases a simple diagnostic procedure can be of great assistance.[3,6] A lateral xeroradiogram of the knee can demonstrate damage to soft tissues, cruciate ligaments and bones with one exposure and is relatively simple to perform during double-contrast arthrography.

MATERIAL AND METHOD

Double contrast arthrography (using the Butt technique[1]) has been in routine use at the Peter Bent Brigham Hospital in Boston for over 2 years, and nearly 1,000 arthrograms of the knee have been made. Recently a lateral xeroradiogram has been included in routine procedure. Of the 20 patients who have had this additional technique, the cruciate ligaments were normal in 15 (Fig. 1, *A* and *B*) in 3 the cruciate

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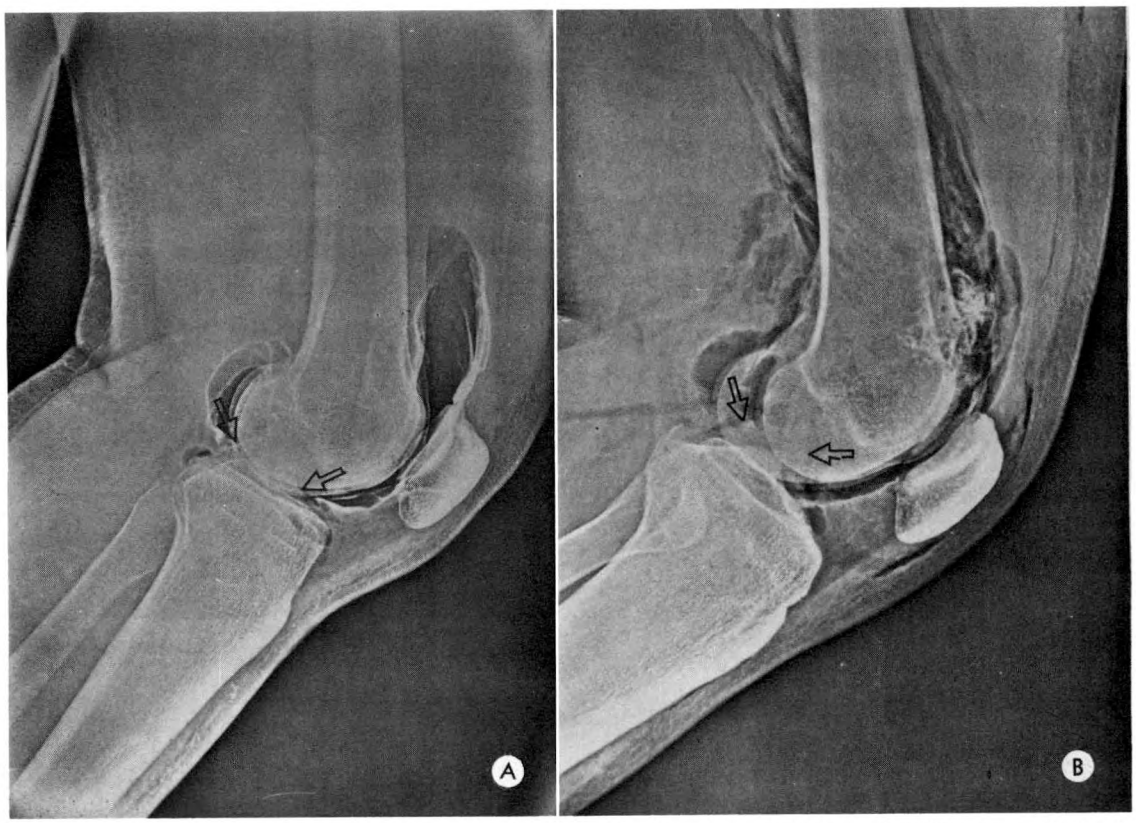


FIG. 1. Normal cruciate ligaments as shown on lateral xeroradiograms of the knee. (*A*) The arrows indicate the synovial reflection overlying both the anterior and posterior cruciate ligaments. (*B*) The arrows indicate the cruciate ligaments. Note the extravasation of both air and contrast medium into the muscle planes.

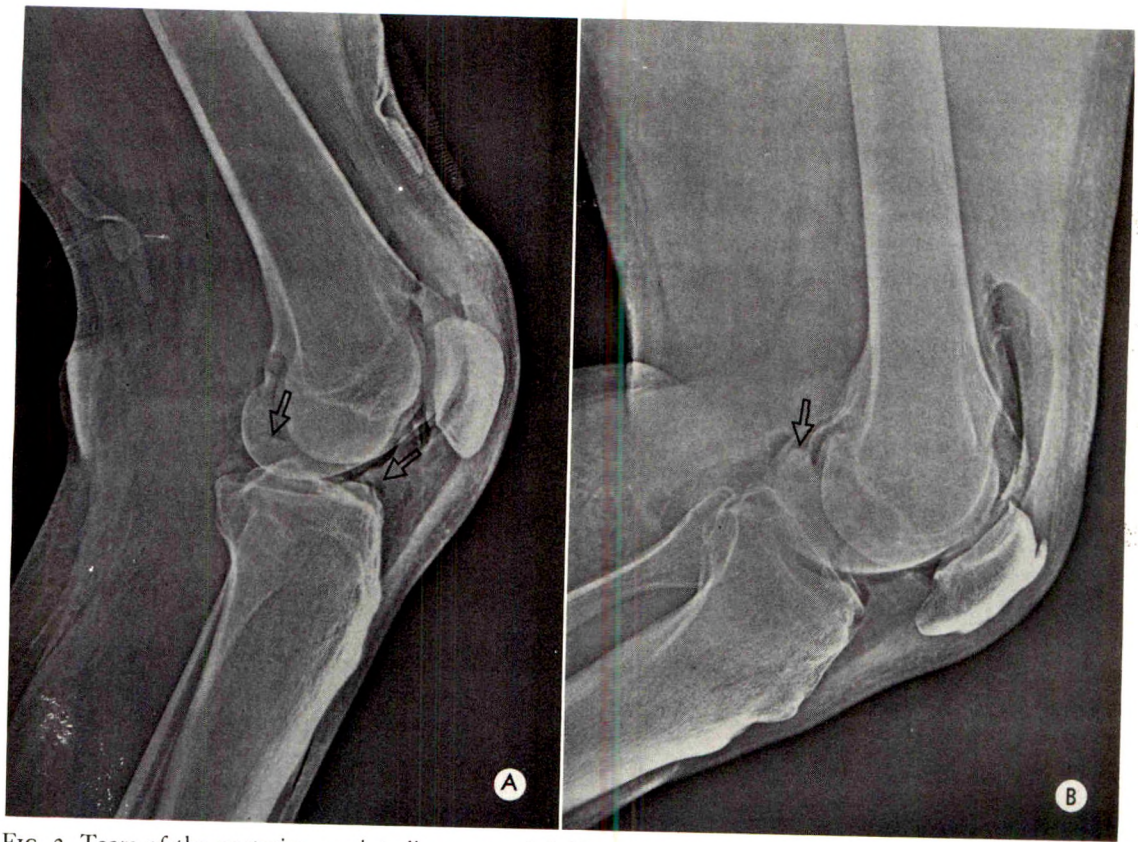


Fig. 2. Tears of the posterior cruciate ligaments. (*A*) Note that the posterior density appears to run horizontally. The patient also has irregularity of the medial meniscus (anterior arrow). Note the ace bandage which is visible around the soft tissues (plus the butterfly clip). (*B*) Tear of the posterior cruciate ligament proven surgically. The normal reflections cannot be seen and a soft tissue mass can be seen lying posteriorly (arrow).

ligaments failed to visualize, and in 2 a tear of the posterior cruciate ligament was demonstrated (Fig. 2, *A* and *B*) (1 of which has been confirmed surgically). Two patients were also found to have Baker's cysts (Fig. 3).

Films of the knee were exposed in anteroposterior and lateral projections using 300 ma. at 1/15 of a second, a 3 mm. focal spot and 120 kvp. Plates were conditioned and processed at "C" setting. The settings (A–D) adjust the unexposed plate to an electrostatic charge appropriate for the area to be examined, and the processor controls the amount of toner powder available for image production.

DISCUSSION

Most authorities use special techniques to demonstrate the cruciate ligaments while performing double-contrast arthrography. Single-contrast arthrography has also been used to demonstrate cruciate tears, but this is not as accurate as double-contrast arthrography in demonstrating meniscal injuries. Since double-contrast arthrography has proven itself for the diagnosis of meniscal tears,[4] it would seem best to employ an additional technique to enhance visualization of the cruciate ligaments at the same time, rather than having to perform 2 arthrographies.

Xeroradiography is a process of image reproduction dependent upon the electrostatic properties of certain groups of substances called semiconductors.[5] High resolution is one of the greatest assets: 600 lines per centimeter can easily be recorded. Its



FIG. 3. Xeroradiogram demonstrating retained contrast medium in the suprapatellar bursa anteriorly and a large Baker's cyst posteriorly (arrow).

edge enhancement is useful in studying structures with sharp borders such as fractures and soft tissue calcification. This effect is somewhat dependent on the kilovoltage, which can be diminished or augmented by manipulating the x-ray technique.[7] It has also been shown that scatter is much lower than with plain-film radiography.[2] These features all contribute to the sharp etched-like quality of xeroradiograms. Furthermore, a wide recording latitude makes it possible for xeroradiograms to demonstrate clearly all radiodensities on a single exposure (*i.e.*, bone through a plaster cast). Because the current technique of xeroradiography utilizes a radiation dose which is lower than was previously necessary, the procedure is now as safe as routine radiography.

Neither single-contrast nor double-contrast arthrography have lived up to their promise in the demonstration of lesions other than to the menisci. Hence, xeroradiography appears to be a promising method of enhancing the radiographic diagnosis of knee injuries.

SUMMARY

The addition of xeroradiography to the radiologist's armamentarium frequently helps in the diagnosis of soft tissue injury.

Twenty patients who had double-contrast arthrography of the knee had xeroradiograms for demonstration of the cruciate ligaments and in 2 patients tears of the posterior cruciate ligaments were shown, one of which was proven surgically.

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REFERENCES

1. BUTT, W. P., and MCINTYRE, J. L. Double contrast arthrography of knee. *Radiology*, 1969, *92*, 487–499.
2. HENNY, G. C. Effect of roentgen-ray quality on response in xeroradiography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *79*, 158.
3. LEE, H. G. Avulsion fracture of tibial attachments of cruciate ligaments. *J. Bone & Joint Surg.*, 1937, *19*, 460–468.
4. MCINTYRE, J. L. Arthrography of lateral meniscus. *Radiology*, 1972, *105*, 531–536.
5. ROACH, J. F. Xeroradiography. *Radiol. Clin. North America*, 1970, *8*, 271–275.
6. TRICKEY, E. L. Rupture of posterior cruciate ligament of knee. *J. Bone & Joint Surg.*, 1968, *50-B*, 334–341.
7. WOLFE, J. N. Xeroradiography of bones, joints and soft tissues. *Radiology*, 1969, *93*, 583–587.

LIPOHEMARTHROSIS*

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TRAUMATIC lipohemarthrosis may occur in any joint in which an intracapsular fracture has occurred, allowing blood and fat to extrude into the joint space. Clinicians have traditionally used the presence of fat in the joint aspirate to distinguish a fracture from simple hemarthrosis. Radiologists have recognized fat blood fluid levels in the knee and the shoulder,[1–6] and have used this as a diagnostic sign of fracture. In the shoulder joint, this finding has always heretofore been associated with a fracture dislocation[6] (Fig. 1, *A* and *B*). The presentation of a fat blood fluid level in a patient with only simple dislocation prompted a review of our experience with shoulder trauma.

MATERIAL

From 1962 to 1971, 80 positive cases of shoulder trauma were seen at the Hospital of the University of Pennsylvania. Of these 80, 44 had roentgenograms available for review. There were 27 fractures, 8 fracture dislocations and 9 simple dislocations of the shoulder joint. Fat blood fluid levels were present in 2 of 27 fractures (8 per cent), 5 of 8 fracture dislocations (63 per cent), and 4 of 9 simple dislocations (45 per cent). In reviewing the roentgenograms, it was evident that many roentgenograms were not made in the erect position, the traditional posture. In severely injured patients, the examination was often made in the supine position, so that the fat blood fluid

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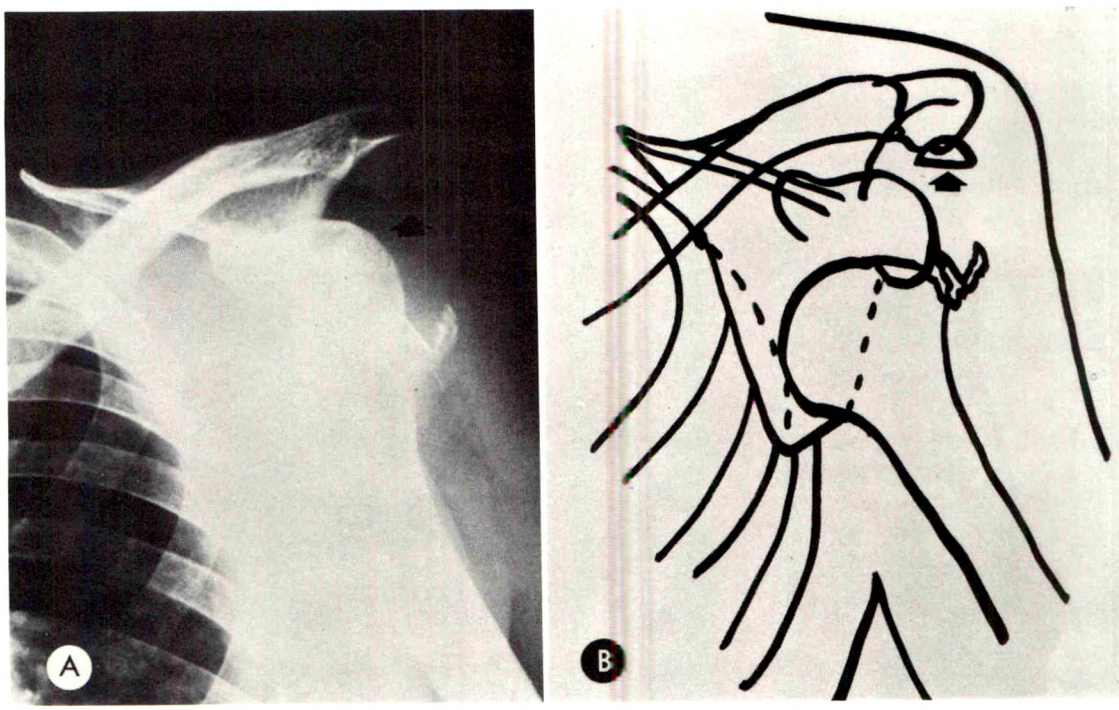


FIG. 1. (*A*) Classic fracture dislocation of shoulder with fat blood fluid level (closed arrow). (*B*) Schematic drawing of *A*.

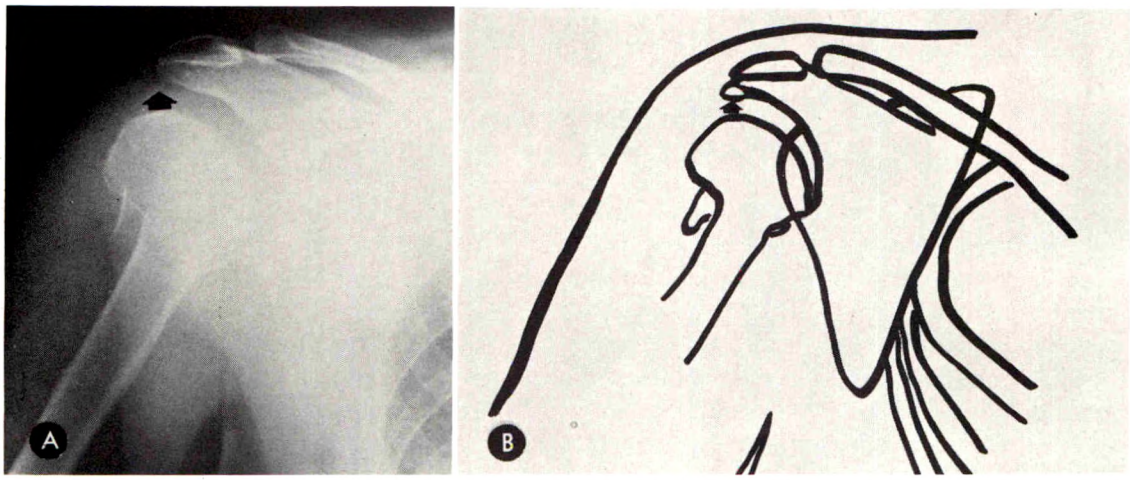


FIG. 2. (*A*) Extracapsular fracture without dislocation showing small fat blood fluid level (closed arrow). (*B*) Schematic drawing of *A*.

level would not be seen. It seems likely that fat blood fluid levels about the shoulder may be seen in even a higher percentage than is indicated by our review.

DISCUSSION

Lipohemarthrosis of the knee following trauma was first described by Kling[3] in 1929. He found fat in the joint aspirate of 6 of 15 patients (40 per cent). Holmagren[2] first reported the roentgenographic demonstration of fat blood fluid levels following knee trauma by making a lateral roentgenogram with a horizontal beam. He found a

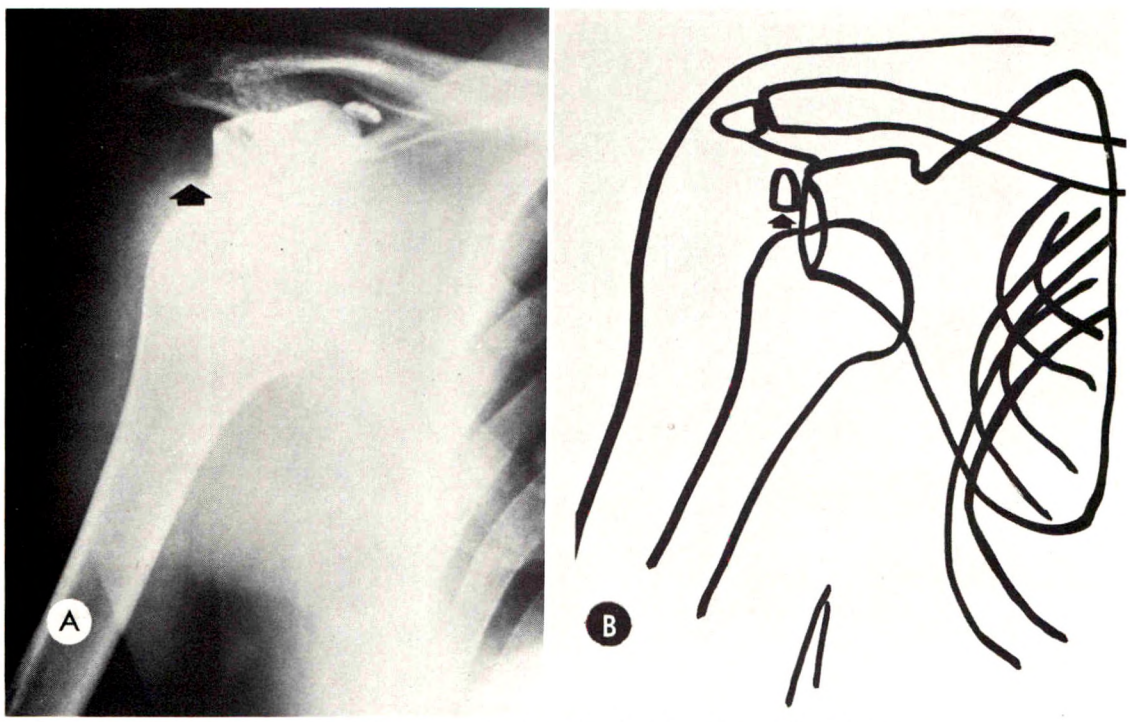


FIG. 3. (*A*) Fat blood fluid level in simple dislocation without fracture (closed arrow). (*B*) Schematic drawing of *A*.

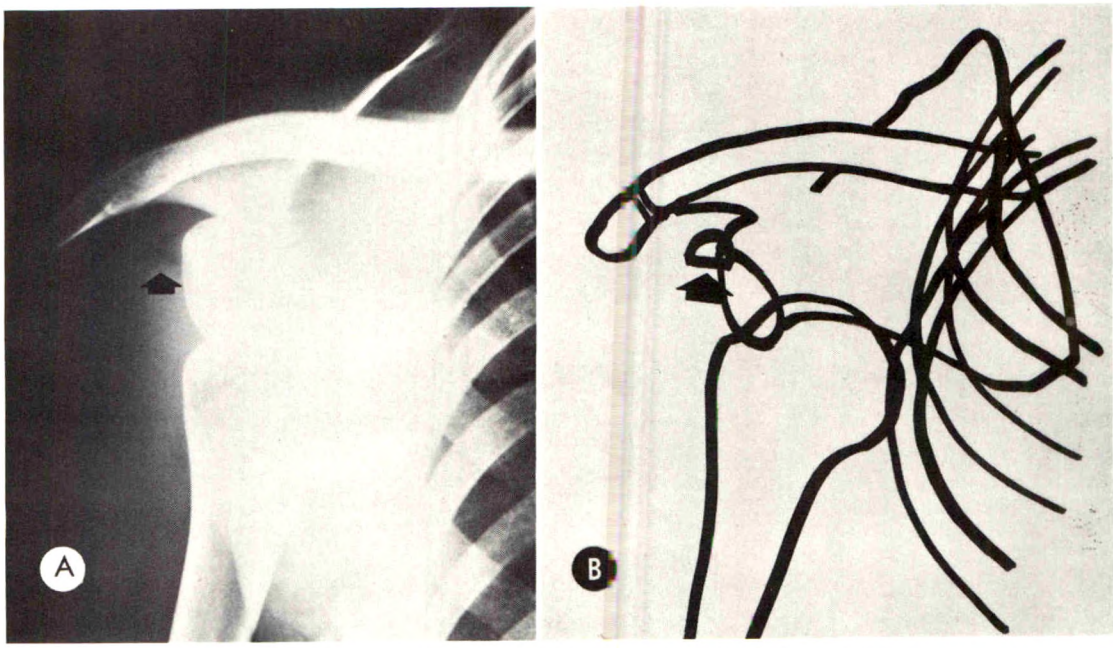


Fig. 4. (*A*) Fat blood fluid level in simple dislocation without fracture (closed arrow). (*B*) Schematic drawing of *A*.

fat blood fluid level in 50 per cent of 65 cases.[2]

Additional authors have reported this in the knee.[1,4,5] In 1962, a fat blood fluid level was reported on shoulder roentgenograms by Saxton[6] in 6 fracture dislocations.

The source of the fat in the joint space is assumed to be the bone marrow. Pierce and Eaglesham[5] have postulated that tearing of a synovial membrane and its associated fat might cause lipohemarthrosis, but they reported no cases without associated fracture.

Saxton[6] stated that 2 prerequisites are necessary for demonstration of a fat blood fluid level. These are: (1) a roentgenogram taken with a horizontal beam; and (2) a distensible joint well filled with blood and fat, preferably with the fat lying clear of the bone. He showed that in the knee joint 10 to 20 ml. of fat and 80 to 100 ml. of blood are necessary to show a fat blood level.

Several of our cases pose interesting problems regarding the origin of the fat in the shoulder joint following trauma. While a fat blood fluid level has not been demonstrated heretofore in a simple fracture of the shoulder without dislocation, its appearance might readily have been predicted. Case II (Fig. 2, *A* and *B*) is a patient with a fracture of the proximal humerus without dislocation. The fracture in this case is not intracapsular, indicating various origins for the fat blood levels.

Cases III and IV (Fig. 3, *A* and *B*; and 4, *A* and *B*) are 2 of 4 simple dislocations in which a fracture could never be demonstrated on either pre- or postreduction roentgenograms.

We can only speculate about the origin of the fat in the shoulder of patients without intra-articular fracture. Presumably, it must come from trauma to the synovium and associated intra-articular or periarticular fat. It is possible that a minute fracture of the humeral head or glenoid was overlooked, but a careful search was made and no fracture could be seen on any roentgenograms. It seems likely that lipohemarthrosis occurs without an associated intra-articular fracture.

SUMMARY

Lipohemarthrosis of the shoulder is discussed.

The association of this entity with simple shoulder fractures and simple dislocations is demonstrated.

Fat blood fluid levels in the shoulder joint in extracapsular fractures and simple dislocations suggest that on occasion the fat may arise from some other source than bone marrow.

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REFERENCES

1. BAVENDAM, F. A., and NEDELMAN, S. H. Some considerations in roentgenology. *Seminars Roentgenol.*, 1966, *1*, 407–436.
2. HOLMAGREN, B. S. Flüssiges Fett in Kniegelenk nach Trauma. *Acta radiol.*, 1942, *23*, 131–137.
3. KLING, D. H. Fat in traumatic effusions of knee joint. *Am. J. Surg.*, 1929, *6*, 71–74.
4. NELSON, S. W. Some important diagnostic and technical fundamentals in radiology of trauma, with particular emphasis on skeletal trauma. *Radiol. Clin. North America*, 1966, *4*, 241–259.
5. PIERCE, C. B., EAGLESHAM, D. C., and DOUGLAS, C. Traumatic lipohemarthrosis of knee. *Radiology*, 1942, *39*, 655–662.
6. SAXTON, H. M. Lipohaemarthrosis. *Brit. J. Radiol.*, 1962, *35*, 122–127.

THE DISTAL ANTERIOR FEMORAL METAPHYSEAL DEFECT: AN ANATOMIC VARIANT THAT MAY SIMULATE DISEASE*

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THE growing skeleton is fraught with hazard for the unwary radiologist. This is particularly true in the distal portion of the femur where one may find a large number of aberrations of growth which simulate pathology. This report is concerned with such a variant in the cortex of the anterior aspect of the distal femoral metaphysis which is innocent in itself, but which has been misinterpreted as a manifestation of incipient disease.

This entity, which I have termed the distal anterior femoral metaphyseal defect, is not uncommon and is probably frequently overlooked. I have seen it repeatedly over the years, but my attention was focused by the referral from an orthopedist of a 15 year old boy, the son of a surgeon, who was being seen with regard to pain in the back of his right knee. The lateral roentgenogram of his knee, made elsewhere, showed the commonly seen irregularity of the cortex of the posterior aspect of the distal femur, and, in addition, a defect in the cortex of the anterior aspect of the distal femoral metaphysis, immediately above the epiphyseal line. The latter finding was of concern to the outside radiologist as well as to the attending orthopedist in that it might be a reflection of a pathologic process, particularly a neoplasm (Fig. 1). A similar appearance was not seen in the opposite knee (Fig. 2). The innocent nature of the finding was pointed out and the boy has done well without treatment. Follow-up roentgenograms showed reversion to normal appearance with continued growth.

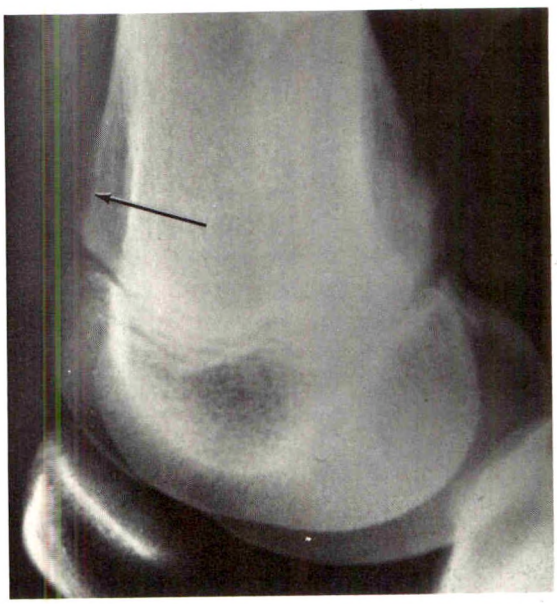


FIG. 1. Anterior cortical femoral defect in the right knee of a 15 year old boy. Note the posterior cortical irregularity (arrow).

DISCUSSION

The anatomic variant described here is seen in adolescents of both sexes, particularly between the ages of 12 and 16 years. Another example is the 13 year old girl shown in Figure 3. This variant of growth is self-limited in that it disappears with advancing age.

The etiology of this cortical irregularity is unknown and is likely to remain an enigma histologically unless it is inadvertently biopsied, as might have occurred in the patient described above if its innocent nature had not been recognized. The portion of the femur involved is intra-articular and it is unlikely that it represents an avulsive cortical irregularity as does the posterior cortical irregularity seen in Figure 1.[2] It is more likely to represent a variation in development similar to that described by Brower *et al.*[1] as a fibrous cortical lesion.

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FIG. 2. The left knee of the same patient seen in Figure 1. The anterior cortical femoral defect is not seen.



FIG. 3. Anterior cortical femoral defect (arrow) in a normal 13 year old girl.

Its importance appears to be solely one of recognition of an insignificant variation of growth and not as a manifestation of a pathologic process.

SUMMARY

A previously undescribed developmental defect in the cortex of the anterior aspect of the distal femoral metaphysis in adolescents is described.

This normal variant may simulate a destructive lesion of bone and its recognition is, therefore, of clinical importance.

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REFERENCES

1. BROWER, A. C., CULVER, J. E., JR., and KEATS, T. E. Histological nature of cortical irregularity of medial posterior distal femoral metaphysis in children. *Radiology*, 1971, *99*, 389–392.
2. BUFKIN, W. J. Avulsive cortical irregularity. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *112*, 487–492.

MULTIFOCAL OSTEOID OSTEOMA*

A CASE REPORT AND REVIEW OF THE LITERATURE

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OSTEOID osteoma is a rather common benign lesion consisting of a small (up to 2 cm.) nidus of highly vascularized osteoid tissue surrounded by sclerotic bone.[4,5,8] In only rare instances may osteoid osteoma be multifocal: 10 such cases have been reported in the world literature.[2,3,7,11,12]

The authors present a case of a multifocal osteoid osteoma with 3 nidi involving the distal tibia.

REPORT OF A CASE

This was a 17 year old male with a history of pain in the left lower leg of 3 months' duration. The pain was promptly relieved by aspirin. Physical examination and laboratory studies revealed no abnormalities. Roentgen examination of the left lower leg (Fig. 1, *A* and *B*) showed an area of sclerosis involving the lateral and anterior aspects of the distal tibia. The margins of the sclerotic focus were poorly defined and there was slight bulging and thickening of the cortex on the lateral aspect. Within the sclerotic area there were 2 fairly well defined radiolucencies seen in both the lateral and frontal projections. Surgical resection of the involved area of the tibia was performed. Pathologic study (Fig. 2) showed 3 sharply delineated oval, reddish, gritty nodules within the spongy marrow measuring 0.2 to 0.5 cm. in diameter which on histologic examination (Fig. 3) showed osteoid tissue with trabeculae of new bone in a vascular stroma. The pathologic findings were consistent with multifocal osteoid osteoma.

DISCUSSION

The characteristic roentgen appearance of osteoid osteoma is that of a small oval or round lucent center (the nidus) surrounded by a zone of radiopacity (the sclerotic bone).[1,10] Occasionally, a densely mineralized central part or ring-like calcified central area can be observed.[11] When the lesion is situated in small bones, the pattern is usually that of a dense center representing nidus ossification separated from the surrounding sclerotic bone by a rim of radiolucency representing the unossified nidus.[1]

Occasional cases of osteoid osteoma may present with atypical manifestations. These are: subperiosteal location (the nidus extends into the soft tissues raising the periosteum);[8,9,11] juxta-articular location;[8,9] accelerated growth;[9] spontaneous regression;[8] occurrence in a very young group of patients;[1,9] occurrence without characteristic pain;[9] or appearance as a double lesion (widely separated lesions in the same bone).[6]

In the previously reported cases of multifocal osteoid osteoma, 5 were true multifocal lesions having the appearance of multiple circumscribed radiolucencies of a size similar to that of a nidus of the usual osteoid osteoma, enclosed in a common block of sclerotic bone. One was medullary in location with 3 foci in the sacral bone.[11] Three cases presented in a peripheral juxtacortical location. One showed 2 foci in the femur,[2] another was in the ethmoid bone (reported as multifocal, but the number of foci is not given),[11] another in the pubic bone (reported as several small foci).[11] The fifth case demonstrated several small foci in the posterior elements of the vertebral body of the second lumbar vertebra involv-

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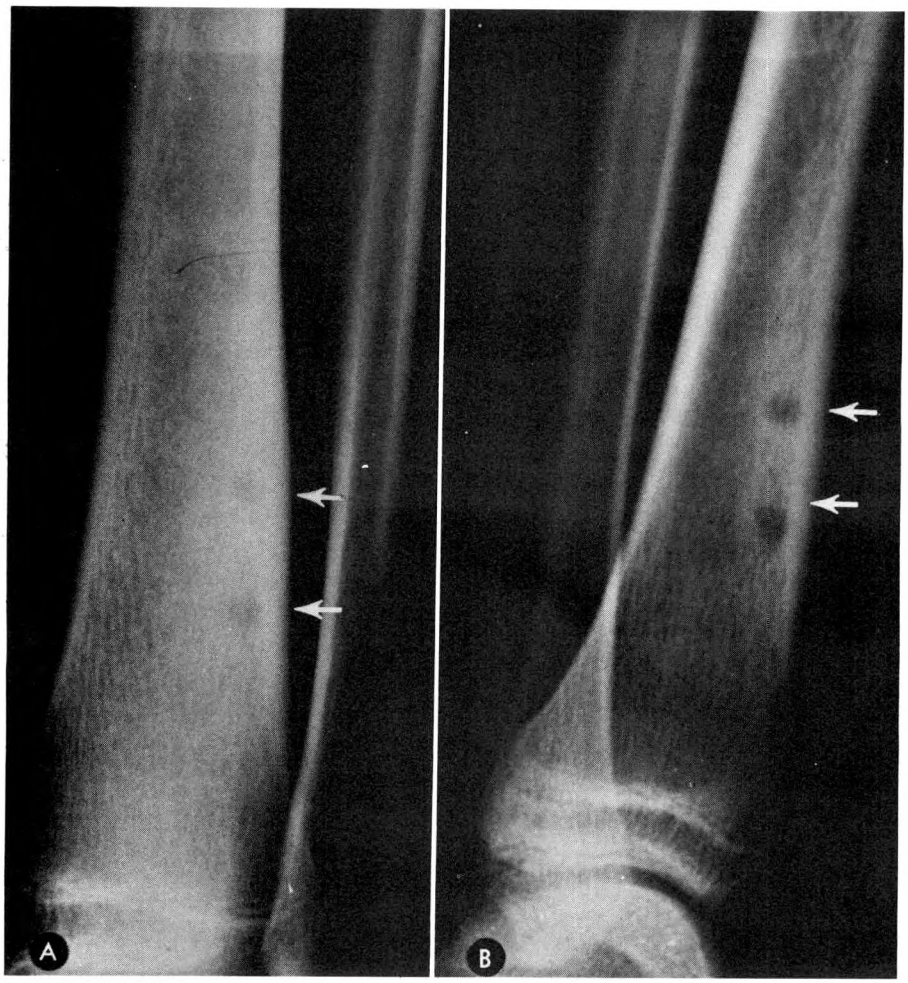


FIG. 1. Roentgenograms of the left distal tibia. (*A*) Frontal and (*B*) lateral views. Note the sclerotic area in the lateral anterior aspect of the distal tibia adjacent to the lateral cortex. There are 2 well defined radiolucencies (arrows) within the sclerotic area. The lower radiolucency is probably due to the lower 2 adjacent nidi seen on the specimen.

ing the arch at its junction with the lamina.[7] Three other reported cases appeared to be unifocal originally, but after apparent surgical resection recurred as multifocal lesions.[2,3,11] A ninth case was treated with radiation therapy and only follow-up roentgenograms and subsequent surgery demonstrated its multifocal architecture.[7] A tenth case showed only one long oval area of decreased density in the anterolateral portion of the epiphysis of the tibia. A specimen, however, showed 3 distinct rounded centers separated from each other by fibrous tissue and normal bone. Each focus had a central portion composed of vascular fibrous tissue with many giant cells and was surrounded by a wide ring of osteoid tissue.[12]

In our case, 2 radiolucent nidi surrounded by sclerotic bone were noted roentgenographically in the distal tibia. The surgical specimen, however, showed 3 nidi with the inferior 2 nidi fairly close to one another but distinctly separated by sclerotic bone. Presumably the lower radiolucency seen roentgenographically was due to the 2 in-



Fig. 2. Low magnification microscopic section of the resected specimen showing 3 separate lesions (arrows) surrounded by sclerotic bone. The lesions measure 0.2, 0.4, and 0.5 cm., respectively, in maximum diameter. Lesions 1 and 2 are partially empty due to artifactual detachment of the nidi.

ferior nidi, which could not be resolved roentgenographically because of their close apposition.

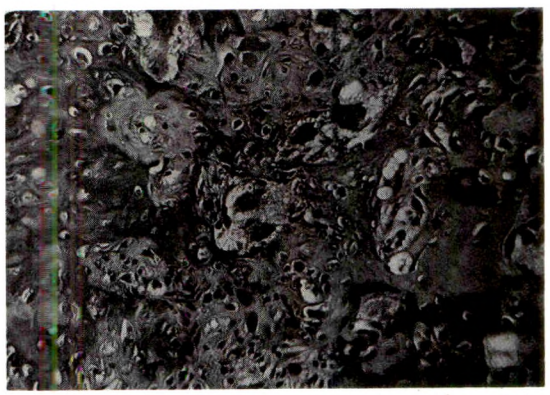


Fig. 3. High power microscopic section of one of the nidi. Note the osteoid tissue with trabeculae of new bone in varying proportions in a vascular stroma, findings consistent with osteoid osteoma.

SUMMARY

A case is presented of a 17 year old male with an osteoid osteoma of the distal tibia which contained 3 nidi.

Multifocal osteoid osteoma is an unusual presentation of this entity with only 10 previously reported cases in the literature.

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REFERENCES

1. Freiberger, R. H., Loitman, B. S., Halpern, M., and Thompson, T. C. Osteoid osteoma: report of 80 cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 194–205.
2. Glynn, J. S., and Lichtenstein, L. Osteoid osteoma with multicentric nidus: report of two cases. *J. Bone & Joint Surg.*, 1973, *55-A.*, 855–858.
3. Goidanich, I. F., and Battaglia, L. Osteoblastoma (fibroma osteogenetica). *Chir. org. Movimento*, 1958, *46*, 353–388.
4. Jaffe, H. L. Osteoid osteoma. *Arch. Surg.*, 1935, *31*, 709–728.
5. Kendrick, J. I., and Evarts, C. M. Osteoid osteoma: critical analysis of 40 tumors. *Clin. Orthop.*, 1967, *54*, 51–59.
6. Lapidus, P. W., and Salem, E. P. Osteoid osteoma: report of case with probable double lesion. *Arch. Surg.*, 1949, *58*, 318–327.
7. Meary, R., D'Aubingne, R. M., and Maza-

BRAUD, A. Osteoblastomes benignes. *Mém. Acad. chir.*, 1965, *91*, 911–925.
8. NORMAN, A. Tumor and tumor-like lesions of bones of foot. *Seminars in Roentgenol.*, 1970, *5*, 407–420.
9. NORMAN, A. Personal communication.
10. PAULSEN, J. O. Osteoid osteoma. *Acta orthop. scandinav.*, 1969, *40*, 198–204.
11. SCHAJOWICZ, F., and LEMOS, C. Osteoid osteoma and osteoblastoma. *Acta orthop. scandinav.*, 1970, *41*, 272–291.
12. SHERMAN, M. S. Osteoid osteoma: review of literature and report of thirty cases. *J. Bone & Joint Surg.*, 1947, *29*, 918–930.

CALCIFICATION IN COOPER'S LIGAMENT*

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CALCIFICATION has been reported in a wide variety of abdominal and pelvic structures,[2] but we are unaware of any accounts of calcification in the pectineal (Cooper's) ligament. We have seen 2 cases of calcification that we believe lie within this structure (Fig. 1, *A* and *B*).

A previous report by Sartor[3] demonstrated almost the same pattern of calcification as seen in our cases, but the location ascribed to it is the "arcus iliopectineus," described as a strip of iliac fascia attached to the inguinal ligament and directed toward the iliopectineal eminence. Radiating fibers from this structure were described as the location of the calcification in Sartor's roentgenograms. This explanation did not corroborate with our concept of the calcification, and prompted us to attempt to correlate the roentgenographic findings with the anatomy of the region.

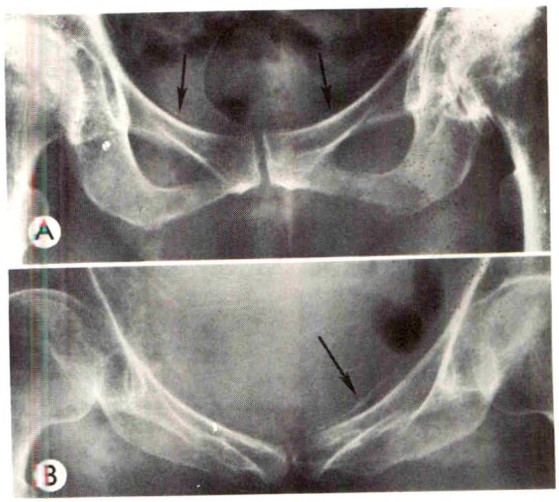


Fig. 1. (*A*) Bilateral symmetrical curvilinear calcification in Cooper's ligament (arrows). (*B*) Unilateral calcification in Cooper's ligament (arrow).

METHOD

A cadaver was dissected in the inguinal area and a metallic marker placed on Cooper's ligament. A roentgenogram of this assembly (Fig. 2) corresponded exactly with the position of the calcification in question. A metallic marker was also placed on the "arcus iliopectineus," described by Sartor as dividing the broad space between the inguinal ligament and the iliac bone into the lateral lacuna musculorum and the medial lacuna vasorum. The marker was noted to be quite distant (Fig. 2) from the calcification described.



Fig. 2. Cadaver roentgenogram. Small arrow indicates marker placed on Cooper's ligament. Large arrow marks location of "arcus iliopectineus."

DISCUSSION

Cooper's ligament is a lateral aponeurotic extension of the lacunar ligament, closely adherent to the pectineal line of the pubis, and extending laterally to the iliopectineal eminence.[1] It is this structure that is the location of the calcification in both the roentgenograms of Sartor and the present study (Fig. 3). The "arcus iliopectineus," which must represent fibers of the iliopectineal fascia, lies considerably lateral to, and is oriented almost perpendicular to the calcification in question, and seems an unlikely explanation.

Calcification in Cooper's ligament should be added to the list of "physiologic" calcifications in the pelvis which appear to be

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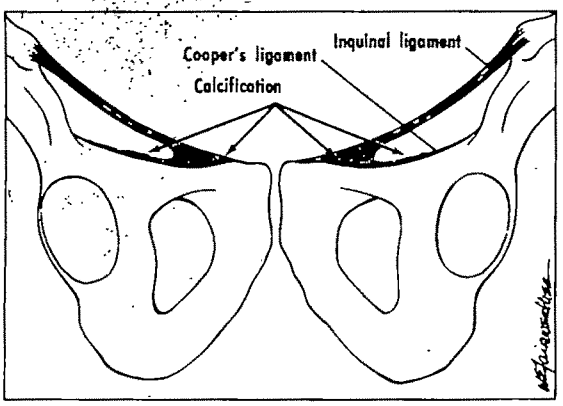


FIG. 3. Diagram illustrating location of Cooper's ligament.

a product of the aging process. Its recognition is important since this rim of calcification might be mistaken as a manifestation of reactive new bone along the iliopectineal line in a patient with pelvic neoplasm.

SUMMARY

Calcification in Cooper's ligament is described and correlated anatomically.

This "physiologic" calcification should not be mistaken for periosteal reaction along the iliopectineal line in patients with pelvic malignancy.

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REFERENCES

1. Gray's Anatomy of the Human Body. Twenty-seventh edition. Edited by G. M. Goss. Lea & Febiger, Philadelphia, 1959, p. 458.
2. McAFEE, J. G., and DONNER, M. W. Differential diagnosis of calcifications encountered in abdominal radiographs. *Am. J. M. Sc.*, 1962, *243*, 609–650.
3. SARTOR, K. Symmetrische Verkalkung des Arcus iliopectinei. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1972, *116*, 120.

EXTRAVASATION OF CALCIUM GLUCONATE AS A CAUSE OF SOFT TISSUE CALCIFICATION IN INFANCY*

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SOFT tissue calcification in the neonate or young infant is an unusual occurrence. In our experience at the C. S. Mott Children's Hospital, the most common cause of roentgenologically demonstrable soft tissue calcification in this age group has been extravasation into the soft tissues during attempted intravenous administration of a solution containing calcium gluconate.

It is our purpose to report 3 such cases of calcium gluconate extravasation. We have also attempted to reproduce this finding in laboratory animals and have obtained histologic specimens for correlation with our roentgenographic findings.

REPORT OF CASES

CASE I. K.P.—I 299 938. A 3 pound 6 ounce Negro male, transferred to the C. S. Mott Children's Hospital at 24 hours of age because of apneic episodes and probable sepsis. The pregnancy was complicated by rupture of membranes 1 week prior to delivery. Delivery was by cesarean section at 33 weeks of gestation because of maternal temperature of 102.6° F. and transverse lie. Cultures of amniotic fluid subsequently grew hemolytic *Streptococcus* and *E. coli*. Apgar at 1 minute was 1 and the baby required intubation and resuscitation. At 6 hours, the baby developed apnea and bradycardia.

On admission the infant was 32 to 33 weeks gestation. There was a Grade 2/6 systolic murmur; the remainder of the examination was otherwise unremarkable. The admission chest roentgenograms were interpreted as possible mild respiratory distress syndrome or pneumonia. The patient was started on penicillin and kanamycin.

At 2 days of age calcium was 5.2 mg. per cent and phosphorus 5.5 mg. per cent. Treatment consisted of an infusion of calcium gluconate into a vein in the dorsum of the hand which subsequently infiltrated into the subcutaneous tissues. Several hours later a purplish discoloration of the dorsum of the hand was noted which was treated by warm soaks and elevation. The following day swelling, erythema, and a large bleb appeared on the dorsum of the hand. Four days later the bleb opened spontaneously and drained, but the erythema was persistent. At 10 days of age, there was persistent swelling and hard deposits between the second and third digits were palpated. At 3 weeks of age a roentgenogram was obtained (Fig. 1) which demonstrated extensive linear and platelike calcification with definite vascular calcification also noted extending up the forearm. At 4 weeks of age a 2 cm. eschar that had formed (Fig. 2) began to slough and was removed, the underlying base appearing necrotic. At 6 weeks the base showed granulation tissue with mild contracture from scar formation causing some limitation of metacarpal-phalangeal flexion of the third and fourth digits. The patient was treated with soaks and mechanical debridement by fine mesh gauze.

The baby was discharged at 10 weeks of age with the lesion on the dorsum of the hand granulating well.

CASE II. S.H.—I 171 867. A 7 pound 3 ounce female was delivered at 41 weeks of gestation by cesarean section because of fetal distress. Apgar was 8 at 2 minute and 10 at 5 minutes. At 2 hours of age the baby developed apnea and cyanosis for approximately 2 minutes which abated on stimulation. Six recurrent episodes of apnea at 30 to 60 minute intervals ensued. Clonic movements of the left arm and leg were noted, associated with the apneic episodes.

Laboratory studies showed a C.S.F. glucose less than 10 mg. per cent, serum glucose less than 10 mg. per cent, and a serum calcium of

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FIG. 1. Case 1. Extensive linear and platelike calcification with vascular calcification extending up the forearm. Three weeks after extravasation of calcium gluconate.

9.1 mg. per cent. Treatment was begun with intravenous and oral glucose.

At 2 days of age the total serum calcium was 6.5 mg. per cent. The patient was treated with 4 cc. intravenous calcium gluconate and calcium gluconate was added to the oral feedings. Three days later the serum calcium was reported as 2.7 mg. per cent. This was treated with 5 cc. intravenous calcium. The following day the serum calcium was 8.3 mg. per cent and the child was doing well and continued to do well on oral feedings with oral calcium supplementation. At 14 days of age an area of necrosis on the right side of the scalp was noted, its etiology unknown. This was described as a central area of black necrosis surrounded by a peripheral area of erythema. Culture of the area showed no growth and 2 needle aspirations demonstrated no fluid or pus. The clinical impression was cellulitis of the scalp and treatment with soaks was begun. The patient was discharged home 3 days later with soaks prescribed for the scalp lesion. Return visit 10 days later showed an indurated "bony" area in the temporal region, posterior and superior to the area of necrosis. The necrotic area was debrided and cultures grew *Staphylococcus aureus*, coagulase positive. Treatment consisted of soaks, topical neosporin, and oral cloxacillin, 125 mg. QID. Ten days later the scalp lesion was felt to be improving and skull roentgenograms were obtained to rule out osteomyelitis (Fig. 3). A calcified plaque was noted in the soft tissues overlying the temporal bone and the possibility of extravasation of calcium gluconate was suggested as the etiology. In retrospect it was noted that an intravenous extravasation had infiltrated in this area 10 days before the area of necrosis and erythema was noted. At 6 weeks of age the scalp was described as erythematous with a white necrotic center surrounded by calcium. Examination 4 days later showed de-



FIG. 2. Case 1. Eschar on dorsum of hand 4 weeks following extravasation of calcium gluconate.

creased induration, good granulation tissue in the necrotic area, and no exudate. At 10 weeks of age the head lesion continued to improve with only minimal cellulitis and small amounts of calcium still present.

At 6 months of age there was complete healing. Only a fibrotic scar was present at the site of previous necrosis.

CASE III. V.B.—1 263 169. One month old infant female, second of twins, was born at 33 weeks gestation with a birth weight of 2 pounds 15 ounces. The infant did well initially, then developed mottling, tachypnea and grunting during the second week. She began having apneic episodes and was transferred to C. S. Mott Children's Hospital at 1 month of age. On admission the baby was severely dehydrated with serum pH 6.8, and blood urea nitrogen of 140 mg. per cent. The initial serum calcium was 4.8 mg. per cent.

The patient was treated for dehydration and acidosis, and the hypocalcemia was treated with intravenous calcium gluconate. The calcium rose to 7.7 mg. per cent. Ten days after administration of calcium, a 2.5 cm. by 2.5 cm. "lump" was noted over the medial condyle of the right knee. This was described as tender, warm, red, circumscribed and nonfluctuant. Consultants from the Departments of Infectious Disease and Orthopedics clinically felt that the differential diagnosis was one of cellulitis *versus* osteomyelitis and the baby was started on intravenous penicillin and methicillin. There was roentgenologic evidence of amorphous calcific density in the region of the right tibial condyle which was believed to be secondary to calcium gluconate infiltration. In spite of the roentgenologic findings, the patient was continued on intravenous antibiotics for 3 weeks without further complication.

The erythema, swelling, and mass diminished and at the time of discharge only a small residual lump remained in that area.

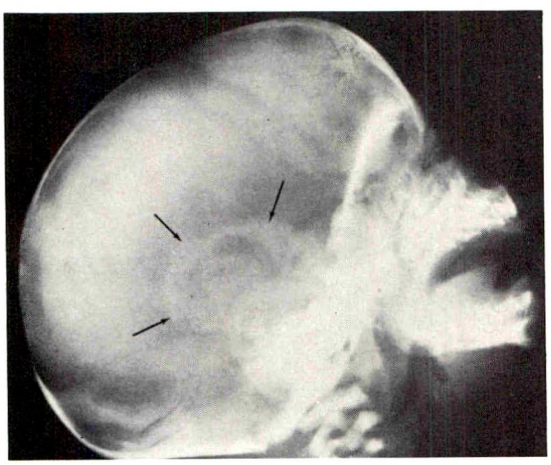


FIG. 3. Case II. Large amorphous calcification (arrows) projecting over temporal region, shown on oblique roentgenograms to be in soft tissues of temporoparietal area. Four weeks following extravasation of calcium gluconate.

REVIEW OF LITERATURE

In 1936 the first 2 reports of complications secondary to intramuscular administration of calcium gluconate appeared in the medical literature. The case of Tumpeer and Denenholz[18] was that of an infant, who on the third day of life developed tetany, and was treated with 1 cc. calcium gluconate intravenously and 9 cc. intramuscularly. An additional 10 cc. of calcium gluconate was given into each buttock on the fourth and fifth days. The infant did well, but on the twentieth day the buttocks were noted to be hard and indurated and on roentgenograms at 5 weeks of age there was diffuse spread of radiopaque material "apparently along the fascial planes of the thighs and buttock, from the hips to the knees." At the sixth week, the overlying skin sloughed and a piece of calcium extruded which was analyzed and proved to be calcium phosphate. On follow-up roentgenograms there was progressive resorption of the calcified material, so that at 4 months of age only a slight amount persisted. Tumpeer and Denenholz[18] speculated that the normally soluble calcium gluconate, "may cause coagulation of the tissue when injected into patients with the disturbed calcium metabolism of tetany." Coagulation "within the blood vessels" was felt to account for the necrosis and sloughing typical of gangrene.

Van Hofe and Jennings[19] reported the case of an infant who had received calcium gluconate on the third, fifth, and sixth days of life because of twitching. Ten cc. of 10 per cent calcium gluconate solution was given into the thigh muscles on 2 occasions

and 10 cc. was also given intramuscularly in the scapular area. At 3 weeks of age, the patient developed induration in all the previously injected areas. There was marked calcification visible roentgenographically throughout the soft tissue about both femora and in the interscapular region. Progressive clearing of the calcific masses occurred at 2 and 3 months of age and at 6 months there was no evidence of calcific deposit.

Shannon[17] reported 2 additional cases, one of which showed roentgenographic visualization of blood vessels of the knee joint, in the area where a mass of calcium also was located secondary to calcium gluconate intramuscular injection. The second case showed the startling finding of a "rather massive deposit of calcium present in the substance of the right lung." The calcific densities eventually disappeared.

Additional cases were reported by Bakwin,[1] Lamm,[6] and McLaren.[10] One of Lamm's 3 cases developed gangrene, sloughing of the buttock area, abscesses, and died.

LABORATORY STUDY

METHOD

Initially 4 cc. of calcium gluconate was injected subcutaneously into disarticulated chicken thighs to determine whether or not the calcium solution itself was radiopaque. Next, 1, 2, or 4 cc. of calcium gluconate was injected subcutaneously into the thighs of living rabbits. These were observed clinically and roentgenologically over the next 6 weeks and the rabbits were sacrificed at various intervals for pathologic examination.

Four additional rabbits were subsequently studied and injected with either normal saline as controls or calcium gluconate as shown in Table 1. These were sacrificed as indicated and histologic specimens obtained. The injection site including

TABLE I

EXPERIMENTAL STUDY: HISTOLOGIC FINDINGS

Time	Agent	Dose	Epidermis and Dermis	Subcutaneum	Soft Tissue Calcification	Vessel Wall Calcification	Muscle Involved	Cellular-Tissue
24 hr.	Saline	2 cc.	o	+	o	o	o	Focal perivascular eosinophilic infiltrate
24 hr.	Calcium gluconate	2 cc.	o	++	+	o	++	Edema, eosinophilia, marked histiocytic reaction
72 hr.	Calcium gluconate	2 cc.	o	++	+	o	+++	Histiocytic infiltrate, occasional foreign body giant cells
7 da.	Saline	2 cc.	o	o	o	o	o	
8 da.	Calcium gluconate	2 cc.	o	+++	++	+	+++	Granulation tissue, fibrous tissue proliferation and foreign body granulomas
15 da.	Calcium gluconate	2 cc.	o	++++	++	+	++++	Same as 8 days, only to a greater extent
30 da.	Calcium gluconate	2 cc.	o	++	+	+	++	Granuloma formation
37 da.	Calcium gluconate	4 cc.	++++	++++	+	+	++	Gross calcified, ulcerated lesion with calcium-filled inclusion cyst formation
45 da.	Calcium gluconate	1 cc.	o	++	+	o	+	Subcutaneum fibrotic, focal calcium in foreign body giant cells in muscle

o = absent; + = present; ++ = mild; +++ = moderate; ++++ = marked.

skin, subcutaneum and underlying muscle was excised and fixed in neutral buffered formalin. Sections were cut at 7 microns and stained with Hematoxylin and Eosin and the Von Kossa stain for calcium. If necessary, the tissue was decalcified prior to sectioning.

EXPERIMENTAL STUDY AND RESULTS

Clinical response following injection of the calcium gluconate was somewhat variable and appeared to be dose related. There was development of some erythema and induration by the second day. By the fifth through seventh days there was more erythema and firm induration. By the tenth day there was a definite hard mass palpable. Erythema and mass were observed with as little as 1 cc. calcium gluconate; however, the degree of associated erythema and the size of the mass appeared dose related. Only 1 rabbit, who received 4 cc. calcium gluconate, developed a necrotic reaction. In the other rabbits there was progressive healing with decrease in erythema, resorption of calcium, and gradual disappearance of the mass. By 6 weeks, the injected site was clinically normal.

The histologic results are summarized in Table 1. Control saline injections produced no reaction except for focal perivascular eosinophilia at 24 hours. The reaction to calcium gluconate at the same time was more eosinophilia, edema and a marked histiocytic reaction with calcification involving muscle fibers in groups and individually. The reaction at 72 hours was a progression of that at 24 hours, with more muscle calcification and the formation of foreign body giant cells, mostly around the calcium salts.

At 8 days, the subcutaneum was markedly thickened with areas of calcification which were also seen in muscle. For the first time, calcium was seen in the walls of arteries and veins. The calcification was focal, involving different layers of the walls in different vessels. No one layer was involved to the exclusion of others. At 15 days the reaction was still further accentuated to its peak (Fig. 4). After 30 days the response was subsiding and there was granuloma formation. At 37 days, however, in the animal which received the largest dose of calcium gluconate, a large ulcerated calcified lesion was present (Fig. 5). This is the only case in which the epidermis was involved and the reaction was present in all layers, including calcification of blood vessel walls below muscle bundles.

The oldest lesion, but the one associated with the smallest calcium gluconate dose, showed primarily subcutaneum thickening with more foreign body giant cells in muscle. The vascular calcification, as well as the muscle and subcutaneum histiocytic infiltrates had resolved.

Roentgenologic Findings. Immediately following injection of calcium gluconate, no evidence of calcification was seen roentgenographically in either the disarticulated chicken legs or the living rabbits. The earliest definite roentgenologic change appeared at 4 days following injection and consisted of minimal linear calcification which appeared vascular. The calcification increased up to a maximum at 2 weeks post injection, it was amorphous with some linearity representing vascular calcification (Fig. 6). On follow-up examination there was a gradual decrease of the calcium and no evidence of calcification was seen 6 weeks post injection.

DISCUSSION

The intravenous use of calcium gluconate should be attended with great care. Calcium gluconate should not be injected intramuscularly even though this use is advocated in Pharmacopoeia,[2] the pharmaceutical reference published by the Council of the Pharmaceutical Society of Great Britain. It states . . . "as it does not cause pain or necrosis it is also given intramuscularly, though in infants it is advisable to employ the oral or intravenous route as cases of abscess formation in the buttocks have been reported."

Calcium gluconate has been shown to be incompatible and form precipitates when

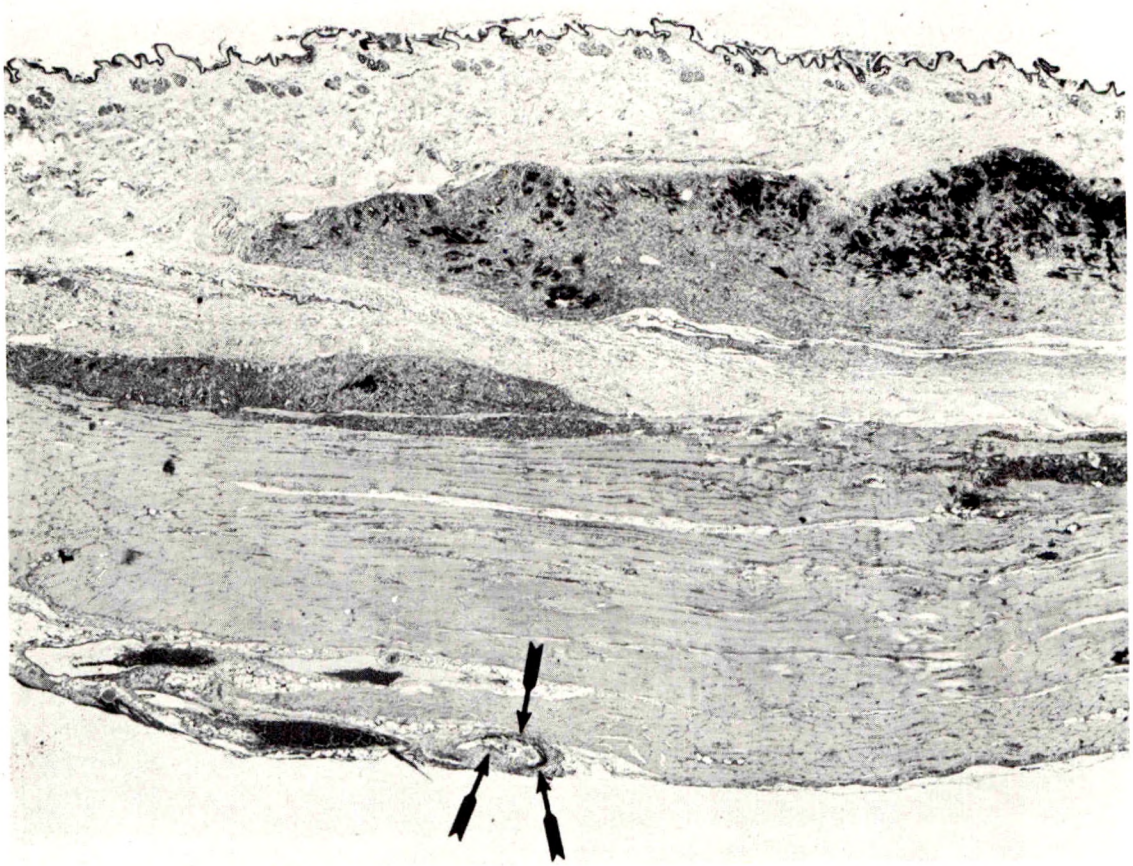


FIG. 4. Cross section of rabbit skin, subcutaneum and muscle (epidermis at top of photograph). Fifteen days post subcutaneous injection of 2 cc. calcium gluconate. Fibrosis, edema and granulation tissue with extensive calcification (black areas). At lower left center (arrows) is vein with calcified intima. (Hematoxylin and Eosin ×9.)

mixed with a variety of pharmaceuticals such as prednisolone sodium phosphate,[11] prochlorperazine maleate, streptomycin sulphate,[13] and amphoteracin.[14] Mizrahi *et al.*[12] stated that calcium gluconate injection should not be added to a continuous intravenous infusion containing sodium bicarbonate, because of the added risk of calcium carbonate precipitation.

Chemical agents other than those containing calcium have been shown experimentally to produce soft tissue calcification. This is dealt with in depth in the monograph by Selye[15] on calciphylaxis. Soft tissue calcification has been produced using lead acetate,[9] vitamin D and ferric chloride.[16]

The histologic changes (Table 1) correlate well with our roentgenographic findings. Thickening of the subcutaneum and calcification there and in the muscle are dose related. Maximum histologic and roentgenographic changes occur at 2 weeks and show a gradual decrease thereafter. By 6 weeks only minimal changes persist histologically and roentgenographic calcification is no longer evident. The vascular calcification seen roentgenographically was also demonstrated histologically and is maximum at 2 weeks post extravasation. This is in keeping with the histologic findings of Leonard *et al.*[7] They injected calcium ATP subcutaneously in rats and produced the formation of white, circum-

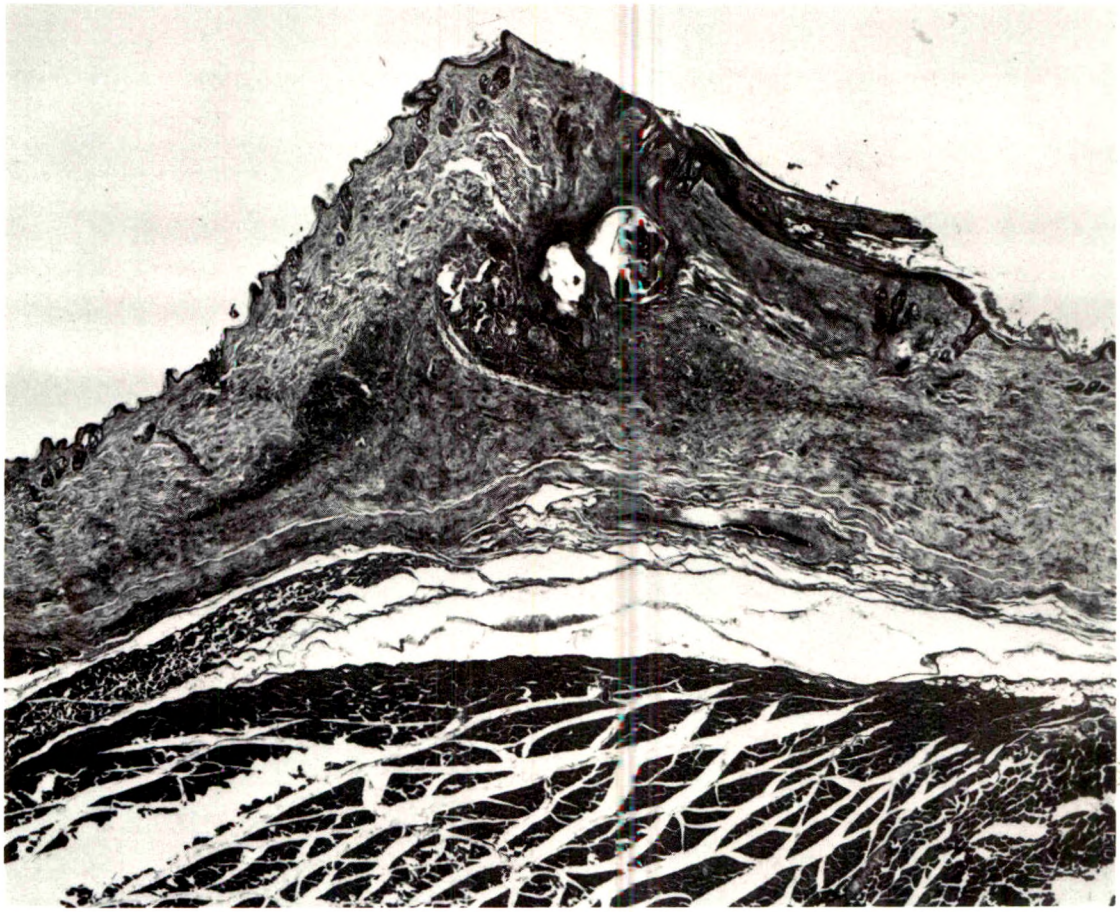


Fig. 5. Cross section of skin, subcutaneum and muscle of rabbit, 37 days post subcutaneous injection of 4 cc. calcium gluconate. Calcium filled inclusion cysts in upper dermis (top middle) with additional calcification in fibrous tissue thickening dermis are seen. (Hematoxylin and Eosin ×9.)

scribed calcific plaques. They demonstrated considerable subcutaneous calcification. On histologic section the mineralization was especially pronounced in and around the blood vessel walls and in the fascia.

The clinical response is variable and may be manifest at 1 to 2 days as erythema and bulla formation, resembling a localized burn as was seen in our Case 1. The lesion may also present as erythema and calcific mass as in Case II. Subsequent eschar formation and sloughing of the skin is common as occurred in 2 of our 3 cases and plastic surgery may be necessary. Secondary infection may occur and there has been 1 reported death as previously described. In one of our cases the clinical diagnosis was osteomyelitis and despite lack of roentgenographic evidence supporting this diagnosis, the infant was still treated with intravenous antibiotics for 3 weeks. With healing of the lesions there is gradual decrease in soft tissue calcification both clinically and roentgenographically and the calcium may completely disappear. This was shown in our Case III and was also demonstrated in our laboratory animals.

Roentgenographic evidence of calcification may be seen within 4 to 5 days post-extravasation. The calcification is amorphous, may be localized or spread through fascial planes. A rather striking finding, that of vascular calcification, was seen in



FIG. 6. Lower extremity of rabbit, popliteal region, 4 weeks post subcutaneous injection of 4 cc. calcium gluconate. Diffuse amorphous calcification with some linearity suggesting vascular calcification.

Case 1. This was also demonstrated clearly in our laboratory animals. One case previously reported by Shannon[17] also showed vascular calcification.

DIFFERENTIAL DIAGNOSIS

As stated at the outset, soft tissue calcification in the neonate and young infant is uncommon. The most commonly reported etiology is subcutaneous fat necrosis of the newborn. This entity has been described associated with hypercalcemia,[8] hypothermia,[3,4] and asphyxia and anoxemia.[5] Calcification of a hematoma in the soft tissues due to birth trauma could conceivably occur. To this differential diagnosis we add extravasation of an intravenous infusion of calcium gluconate. This is an important cause of soft tissue calcification, as our reported cases and those reviewed in the medical literature illustrate.

The roentgenographic findings are of great importance in that they are usually the key to the diagnosis which may be otherwise unsuspected clinically.

SUMMARY

Three cases of soft tissue calcification secondary to subcutaneous extravasation of an intravenous infusion of calcium gluconate are reported.

Histologic changes consist of thickening of the subcutaneum and calcification in the subcutaneum, and the fascial planes. Vascular calcification may also be seen.

Roentgenologic recognition and awareness of this diagnosis is important, as the clinical picture is variable and the etiology of the presenting inflammatory response or mass is often unknown.

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REFERENCES

1. BAKWIN, H. Pathogenesis of tetany of newborn. *Am. J. Dis. Child.*, 1937, *54*, 1211–1226.
2. BLACOW, N. W., and WADE, A. Editors. Martindale, The Extra Pharmacopoeia. Twenty-sixth edition. The Pharmaceutical Press, London, 1972, p. 369.
3. BLAKE, H. A., GOYETTE, E. M., LYTER, C. S., and SWAN, H. Subcutaneous fat necrosis complicating hypothermia. *J. Pediat.*, 1955, *46*, 78–80.
4. DUHN, R., SCHOEN, E. J., and SIU, M. Subcutaneous fat necrosis with extensive calcification after hypothermia in two newborn infants. *Pediatrics*, 1968, *41*, 661–664.
5. HOLZEL, A. Subcutaneous fat necrosis of newborn. *Arch. Dis. Childhood*, 1951, *26*, 89–91.
6. LAMM, S. S. Danger of intramuscular injection of calcium gluconate in infancy. *J.A.M.A.*, 1945, *129*, 347–348.
7. LEONARD, F., BOKE, J. W., RUDERMAN, R. J., and HEGYEL, A. F. Initiation and inhibition of subcutaneous calcification. *Calcified Tissue Research*, 1972, *10*, 269–279.
8. MARTIN, M. M., and STEVEN, E. M. Subcutaneous fat necrosis of newborn with calcification of soft tissues. *Arch. Dis. Childhood*, 1957, *32*, 146–148.
9. MCCLURE, J., and BRIDGES, J. B. Experimental calcifications of soft tissues. *Roy. Soc. Med. Proc.*, 1969, *62*, 240–241.
10. MCLAREN, J. W. Calcium gluconate injection into muscle. *Brit. J. Radiol.*, 1946, *19*, 314–317.
11. MISGEN, R. Compatibilities and incompatibilities of some intravenous solution admixtures. *Am. J. Hosp. Pharm.*, 1965, *22*, 92–94.
12. MIZRAHI, A., LONDON, R. D., and GRIBETZ, D. Neonatal hypocalcemia: its causes and treatment. *New England J. Med.*, 1968, *278*, 1163–1165.

13. Patel, J. A., and Phillips, G. L. Guide to physical compatibility of intravenous drug admixtures. *Am. J. Hosp. Pharm.*, 1966, *23*, 409–411.
14. Riley, B. B. Incompatibilities in intravenous solutions. *Am. J. Hosp. Pharm.*, 1970, *28*, 228–240.
15. Selye, H. Calciphylaxis. University of Chicago Press, Chicago, 1962.
16. Selye, H., Marie, A., and Jean, P. Systemic and topical factors involved in production of experimental cutaneous calcinosis. *J. Invest. Dermat.*, 1961, *37*, 7–12.
17. Shannon, W. R. Tetany syndrome in newborn infants: remote deposit of calcium gluconate. *Am. J. Dis. Child.*, 1938, *56*, 1046–1054.
18. Tumpeer, I. H., and Denenholz, E. J. Calcium deposits following therapeutic injections in tetany of newborn. *Arch. Pediat.*, 1936, *53*, 215–223.
19. Van Hofe, F. H., and Jennings, R. E. Calcium deposition following intramuscular administration of calcium gluconate. *J. Pediat.*, 1936, *8*, 348–351.

PRIMARY LYMPHANGIOMA OF THE CALVARIUM*

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PRIMARY lymphangioma of bone was initially recognized by Bickel and Broders in 1947.[1] Subsequently, additional cases have been reported with multiple bone involvement being usual.[2,8–10]

The case reported here is of interest because of the unusual presentation of a destructive lesion of the skull with absence of other osseous involvement.

REPORT OF A CASE

A man, 27 years old, noted a small bulge in the left frontal region which increased in prominence slightly over 2 years. He had occasional left frontal headaches, but no tenderness over the small lump which appeared to increase in size when he was in the prone position. There was no history of head trauma. Physical examination was unremarkable with the exception of the presence of a nontender, soft tissue prominence over the left orbit. Neurologic examination was normal.

Clinical laboratory data included normal hemogram, urinalysis, blood urea nitrogen, serum electrolytes and serology. The alkaline phosphatase was 19 Iu (normal 9–35 Iu), calcium 4.9 mEq./l. and phosphorus 120 mEq./l. Protein electrophoresis was normal and no Bence-Jones protein was found.

Roentgenologic examination demonstrated a destructive lesion of the left frontal bone just above the orbit with no reactive sclerosis (Fig. 1, *A* and *B*). Anteroposterior and lateral tomograms of the left frontal area demonstrated extension to the roof of the left orbit and cribriform plate. Skeletal survey was normal. A 3.2 mc F^{18} bone scan revealed an area of slightly increased activity in the left frontal bone (Fig. 2). The remainder of the bone scan was normal as was a Tc^{99m} brain scan. Selective left internal and external carotid arteriograms did not show a vascular supply to the osteolytic lesion and were normal. A positive contrast examination of the left orbit did not demonstrate extension of the lesion into the orbit.

Biopsy of the lesion revealed a histologic pattern of folded, branching septa of connective tissue lined by endothelium (Fig. 3). Clear fluid was noted in some of the septa. No hemosiderin deposition, calcification, or clotted blood were seen. The lesion was diagnosed as a lymphangioma and was treated with roentgen therapy (2,000 rads in 5 days). The lesion has regressed in both physical and roentgenologic examination.

DISCUSSION

Lymphangiomas are relatively common tumors of the soft tissues with visceral involvement occurring less frequently. When bone involvement occurs it may be due to an extension of an adjacent lymphangioma, or to origination of the lesion in the bone itself. Flat bones, long bones and the skull are usually involved in this situation. Solitary involvement of the spine and the small bones of the hand has been reported.[4,5] The presentation of an isolated destructive lesion of the skull with no other documented skeletal or visceral involvement has not been previously described.

The destructive lesions of bone apparently result from pressure atrophy secondary to encroachment of the endothelial-lined channels on the osseous architecture. Discovery of the lesion may occur incidentally or may be related to symptoms resulting from effects of the lesion such as pathologic fracture, pain in the area of the lesion or compression and/or invasion of adjacent tissues.

The course of the disease is variable. Patients with generalized skeletal involvement may have progression of the lesions with multiple pathologic fractures resulting, while other patients may be asymptomatic with stabilization or a gradual regression in the size of the bone lesions.

Roentgenographically the bone lesions usually are destructive with a well defined

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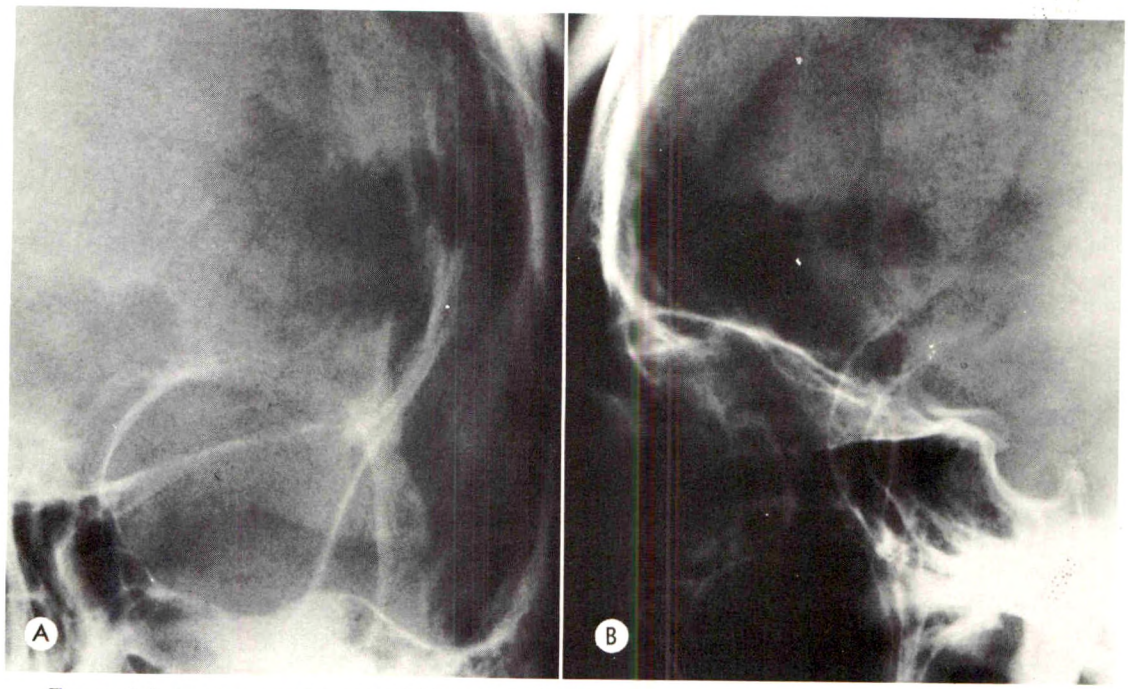


Fig. 1. (*A*) Anteroposterior and (*B*) lateral projections reveal a localized, slightly irregular lesion, lytic in nature, without sclerotic margination.

nonsclerotic margin. Occasionally, the lesions may be ill-defined with thinning and disappearance of bone. Calcification has not been reported. Lymphangiography usually does not enter into the evaluation of destructive lesions of bone. However, Najman *et al.*[6] injected opaque medium directly into a skull lesion and demonstrated a multilocular cystic structure which proved to be a lymphangioma. By means of pedal lymphangiography, Nixon[7] reported demonstration of multiple areas of osseous involvement occurring in a diffuse lymphangioma of a lower extremity. Lerut *et al.*[5] recently presented 3 cases of solitary lymphangioma of bone involving the elbow, femur, and a distal phalanx of the hand, all of which were scan positive. These patients had normal bone surveys and normal scans in the remaining osseous areas.



Fig. 2. An anterior projection of a F^{18} scan demonstrates slight increase in activity in the region of the lesion.

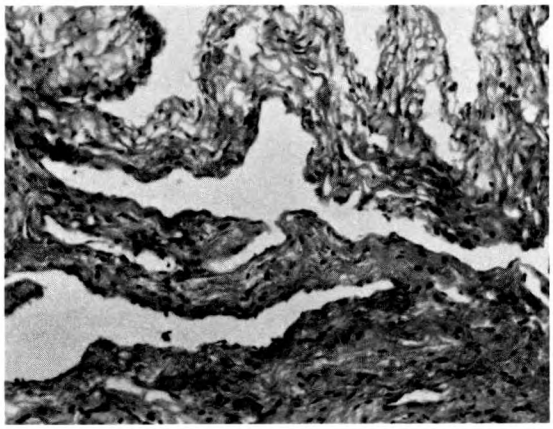


FIG. 3. The histologic pattern is that of multiple fine endothelial-lined channels.

The differential diagnosis of this lesion usually includes metastatic disease, histiocytosis X, Gaucher's disease, fibrous dysplasia, and congenital fibromatosis and neurofibromatosis. If the differential diagnosis is expanded to include lymphangioma, then lymphangiography prior to biopsy might prove helpful, particularly in the lesions of long bones.[8]

As the histology of lymphangioma and hemangioma is very similar, the appearance of the lesion at the time of surgery and the type of fluid it contains becomes important in the final histologic diagnosis.

SUMMARY

Primary lymphangioma, a rare lesion, should be considered in patients with isolated destructive lesions involving the skull.

The diagnosis presently is made by biopsy.

Extent of osseous involvement may be determined by utilizing a bone scan in conjunction with the more routine skeletal survey.

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REFERENCES

1. BICKEL, W. H., and BRODERS, A. C. Primary lymphangioma of ileum: report of case. *J. Bone & Joint Surg.*, 1947, *29*, 517–522.
2. COHEN, J., and CRAIG, J. M. Multiple lymphangiectasis of bone. *J. Bone & Joint Surg.*, 1955, *37-A*, 585–596.
3. FALKMER, S., and TILLING, G. Primary lymphangioma of bone. *Acta orthop. scandinav.*, 1956, *26*, 99–110.
4. GODART, L. Embryological significance of lymphangioma. *Arch. Dis. Childhood*, 1966, *41* 204–206.
5. LERUT, T., WILLEMS, D., and LERUT, H. Lymphangioma of bone. Fourth International Congress of Lymphoma, Tucson, Arizona, March 1973.
6. NAJMAN, E., FABECIC-SABADI, V., and TEMMER, B. Lymphangioma in inguinal region with cystic lymphangiomatosis of bone. *J. Pediat.*, 1961, *71*, 561–566.
7. NIXON, G. W. Lymphangiomatosis of bone demonstrated by lymphangiography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *110*, 582–586.
8. ROSENQUIST, C. J., and WOLFE, D. C. Lymphangioma of bone. *J. Bone & Joint Surg.*, 1968, *50*, 158–162.
9. SHOPFNER, C. E., and ALLEN, R. P. Lymphangioma of bone. *Radiology*, 1961, *76*, 449–453.
10. STEINER, G. M., FARMAN, J., and LAWSON, J. P. Lymphangiomatosis of bone. *Radiology*, 1969, *93*, 1093–1098.

MULTIPLE CALVARIAL "DOUGHNUT LESIONS"*

A CASE REPORT

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THE calvarial "doughnut lesion" previously described in the literature is felt to represent a benign, incidental finding which is histologically (2 biopsy specimens) consistent with reactive osseous fibrosis following local injury. All 21 lesions described by Keats and Holt[1] occurred singly in adults 16 to 72 years of age and were unassociated with other skeletal abnormalities.[2] Their review of the literature disclosed only 1 example of a similar lesion.[3] This was felt to represent an area of infarction in a patient with sickle cell anemia. No biopsy was obtained, however.

The present case demonstrates multiple calvarial lesions, similar in appearance to the aforementioned "doughnut lesion," and associated for the first time with abnormalities in other bones.

REPORT OF A CASE

A healthy 13 year old white boy of Italian-Swedish parentage was referred to Newington Children's Hospital for evaluation of "lumps" on his head.

Four months prior to admission, while combing his hair, the patient felt a firm, nontender bony mass in the posterior frontal region, which seemed to increase in size during the ensuing 3 months. Although he had had occasional nonspecific right periorbital and occipital pain during the preceding year, the patient denied any illness other than intermittent rhinitis.

Past history revealed that the patient had had normal neonatal and childhood development except for fractures of the clavicle, radius, and ulna at the age of 7 years. The patient's parents and 4 siblings were all in excellent health. There was no family history of blood dyscrasia.

Three hard, nontender fixed calvarial masses measuring 1 to 3 cm. were palpable. The patient was neurologically normal, and no organomegaly, ecchymoses, petechiae, or café au lait spots were present.

Laboratory examination. Hemoglobin, hematocrit, and white blood cell count and differential were normal except for 11 per cent eosinophilia. Serum alkaline phosphatase determinations were significantly elevated—14.8 and 10.6 Bodansky units (normal, 2.2 to 6.8). Electroencephalogram, spinal fluid analysis, and all other bacteriological and biochemical studies were normal.

Pathologic findings. Bone marrow aspiration (iliac crest) showed eosinophilic hyperplasia and

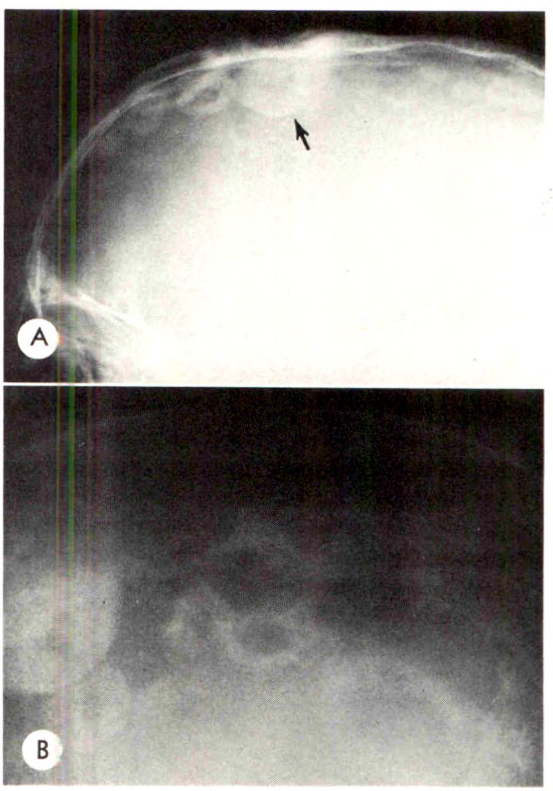


FIG. 1. (*A* and *B*) Numerous lucent foci surrounded by sclerotic margins of variable thickness. Some lesions have a central osseous density. (Arrow indicates subsequently biopsied lesion.)

* From the Department of Radiology, Newington Children's Hospital, Newington, Connecticut.

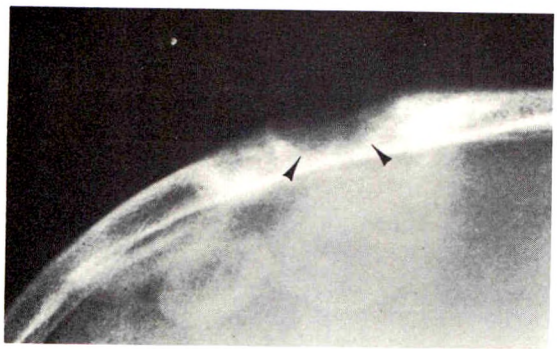


FIG. 2. Elevation and thinning of the external table by one of the 3 palpable lesions (arrows).

mild eosinophilia. Biopsy of the 5th metacarpal (including periosteum, cortex, and marrow) was microscopically normal.

Biopsy of one calvarial lesion demonstrated fibrous tissue with clusters of foam cells or histiocytes with small central nuclei. These were interspersed between osteoid seams and surrounded by sclerotic bone. No eosinophils or inflammatory cells were present. The lesion was felt to be non-neoplastic, but its histologic pattern did not permit definitive classification or diagnosis. Consultation with several bone pathology centers gave quite disparate diagnoses, which included resolving histiocytosis, hyperphosphatasia, fibrous dysplasia, and "unknown non-neoplastic osseous lesion."

Roentgenologic findings. Skull roentgenograms revealed numerous circular calvarial lesions, lucent centrally and surrounded by sclerotic margins of varying thickness. Several lesions seen in profile expanded the outer table without evidence of erosion or associated soft tissue mass, and some had a centrally-located area of bone density (Fig. 1, *A* and *B*; and 2). The bones of the hands and feet were rectangular in profile with absence of normal tubulation (Fig. 3, *A* and *B*). The remaining skeletal and soft tissue structures were unremarkable.

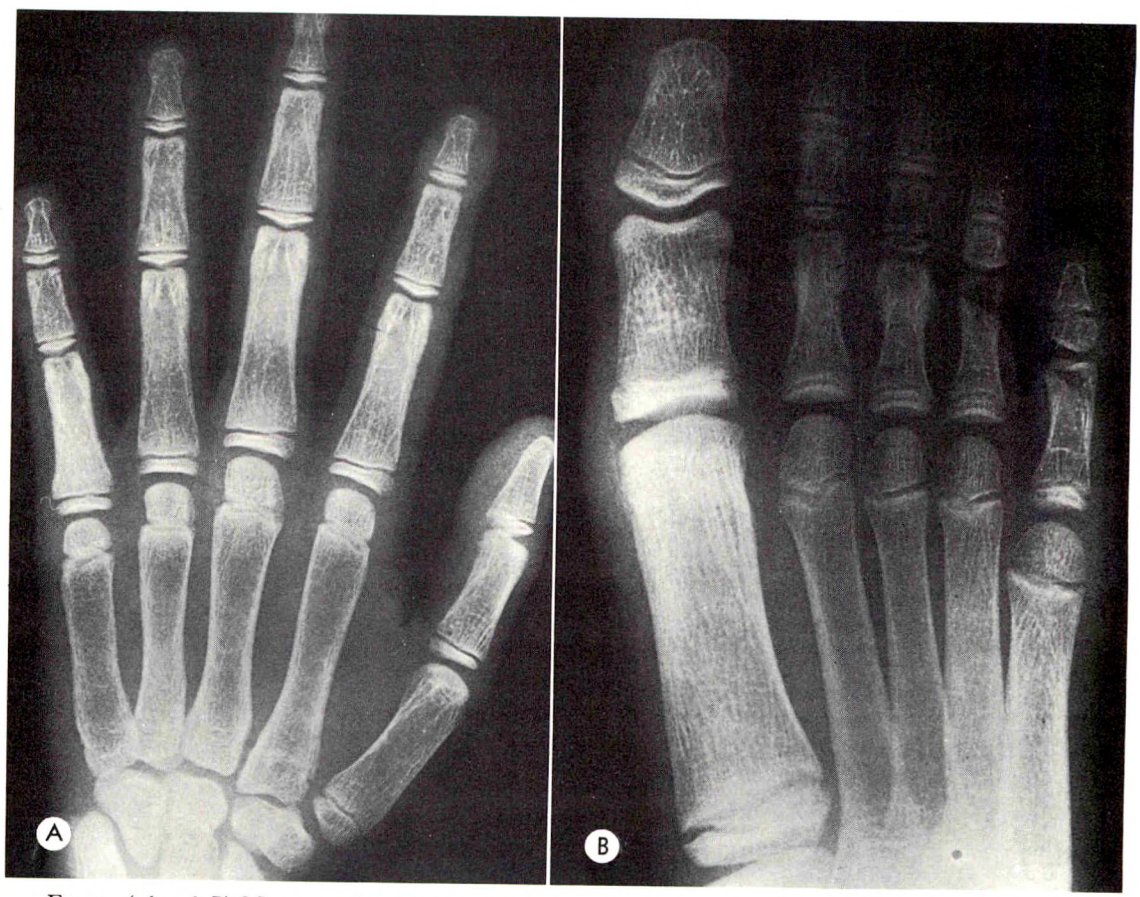


FIG. 3. (*A* and *B*) Metacarpals and metatarsals are rectangular with absence of normal tubulation.

DISCUSSION

This case demonstrates a striking combination of roentgenographic, biochemical, and histologic abnormalities in an essentially asymptomatic 13 year old male. Neither the multiplicity of the calvarial lesions nor their association with metacarpal and metatarsal "squaring" has been previously documented. The significance of the markedly elevated alkaline phosphatase remains uncertain and its relation to the osseous pathology is unknown.

The histologic pattern of the calvarial biopsy specimen differed from those previously described,[1,3] and remains nondiagnostic. Pathologic consultation suggested diagnoses which were clinically untenable. While certain isolated features of this case may be characteristic of specific disease entities such as eosinophilic granuloma and histiocytosis-X, the findings present in this patient have not been satisfactorily collated under a single diagnosis.

SUMMARY

Osseous and biochemical abnormalities are described in an essentially asymptomatic 13 year old male.

Roentgenographically similar bone lesions have been documented as isolated occurrences, but the combination of skeletal and biochemical findings noted herein has not been presented previously.

The etiology of these findings remains obscure.

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REFERENCES

1. Keats, T. E., and Holt, J. F. Calvarial "doughnut lesion": previously undescribed entity. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *105*, 314–318.
2. Keats, T. E. Personal communication.
3. Middlemiss, H. Tropical Radiology. William Heinemann, Ltd., London, 1961.

THE MAXILLARY ARTERY IN CRANIOFACIAL PATHOLOGY*

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THE maxillary artery and its branches comprise a complex arterial system which traverses, supplies, and delineates a large number of deep facial structures. Many facial lesions are characterized by their insidious onset and late diagnosis resulting from their inherent inaccessibility to early clinical detection. The value of facial polytomography is well established, yet at times inconclusive in evaluating extension of such lesions into the surrounding compartments.

Presently available angiographic techniques such as selective catheterization, magnification, and subtraction offer the potential of earlier detection and diagnosis of some deep facial lesions and, in many instances, a more sensitive method of evaluating the extent of involvement.

A previous report delineated the normal arteriographic anatomy of the maxillary artery and its branches.[1] This information has provided a basis for the evaluation of deep facial lesions as pertaining to their location, etiology and routes of extension, and has proved useful in establishing the optimum mode of therapy.

It is the purpose of this report to describe topographic changes in the various segments of the maxillary artery as they relate to facial lesions, and to emphasize the role of the distal branches and their collaterals in intracranial as well as deep facial pathology.

ANATOMY OF THE MAXILLARY ARTERY

The main trunk of the maxillary artery arises at the distal bifurcation of the external carotid artery. It is divided anatomically into 3 major segments based on its relationship to the external pterygoid muscle. The first (mandibular) segment is proximal to this muscle and medial to the ramus of the mandible. The second (pterygoid) segment courses forward through the infratemporal fossa, usually superficial but occasionally deep to the external pterygoid muscle. The third (pterygopalatine) segment is situated within the confines of the pterygopalatine fossa where it gives rise to the distal facial branches.

MANDIBULAR SEGMENT

The first (mandibular) segment of the maxillary artery is relatively fixed in position between the ramus of the mandible and sphenomandibular ligament; it is rarely displaced by mass lesions. A small parapharyngeal process of the parotid gland wraps around the back of the neck of the mandible and the mandibular segment of this vessel is imbedded within it. Consequently, parotid tumors may alter this segment either by direct involvement of the parapharyngeal process of the gland or possibly by displacement of this process (Fig. 1). The main bulk of the parotid gland is lateral to the trunk of the maxillary artery and separated from it by the mandible and its associated muscles. It would be extremely unusual for any other type of soft tissue tumor to directly affect this segment of the maxillary artery.

PTERYGOID SEGMENT

The second (pterygoid) segment courses through the infratemporal fossa, supplying branches to the muscles of mastication. In

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two-thirds of individuals it runs lateral to the external pterygoid muscle and in one-third medial to this muscle.[14] For this reason one should use caution in evaluating medial or lateral displacements without other evidence of a mass lesion. In addition to the maxillary artery, the infratemporal fossa contains some of the muscles of mastication, the mandibular nerve and the chorda tympani. Masses arising directly within this fossa are extremely rare. Invasion of the infratemporal fossa by tumors of nasopharyngeal origin is occasionally encountered.[10] In such cases the second portion of the maxillary artery may be displaced laterally and downward. Nasopharyngeal carcinomas are the most common lesion found to invade the infratemporal fossa. These have sparse vascularity, but will displace and distort normal vasculature (Fig. 2). A vascular nasopharyngeal lesion extending into the infratemporal fossa in young males is most likely to be a juvenile angiofibroma[27] (Fig. 5*C*). Some

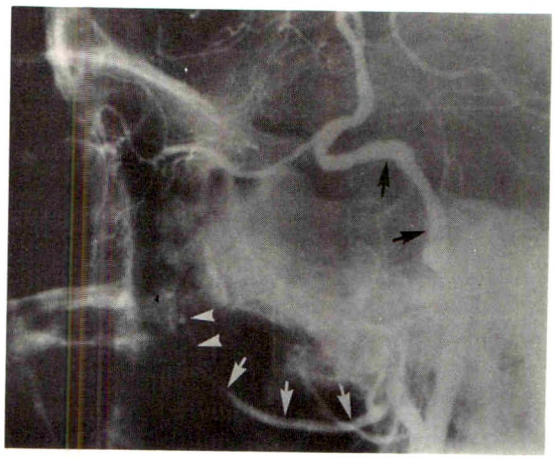


Fig. 2. Displacement of the pterygoid portion of the maxillary artery. Lateral common carotid arteriogram showing inferior displacement of the main trunk of the maxillary artery (white arrows) by a large nasopharyngeal carcinoma indicating spread of tumor into the infratemporal fossa. The terminal pterygopalatine portion is also displaced inferiorly and forward by invasion of tumor into the pterygopalatine fossa (white arrowheads). The petrous and cavernous portions of the internal carotid artery are displaced backward and superiorly consistent with intracranial extension (black arrows).

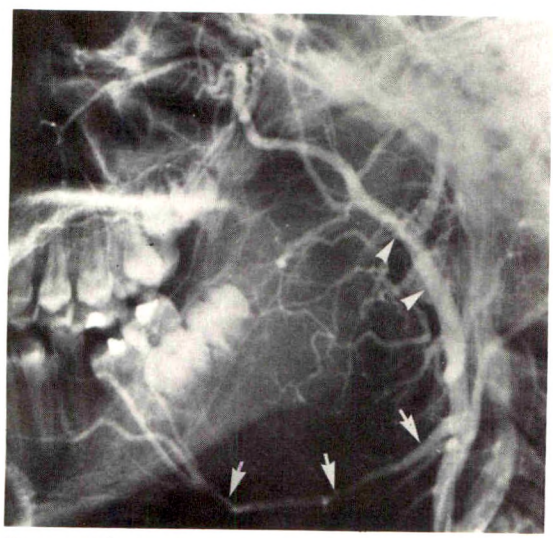


Fig. 1. Displacement of the mandibular portion of the maxillary artery. Lateral carotid arteriogram in a patient with a large avascular parotid tumor. The distal external carotid artery and the mandibular portion of the maxillary artery are displaced by the tumor (arrowheads) consistent with involvement of the parapharyngeal process of the parotid gland. The facial artery is displaced inferiorly (arrows).

malignant tumors may show increased vascularity such as a lymphoepithelioma (Fig. 3*B*) or rhabdomyosarcoma. Other vascular lesions to be considered in this location are hemangiomas and chemodectomas.[17]

Parotid tumors, arising lateral to the infratemporal fossa, are unlikely to involve the pterygoid segment of the maxillary artery unless tumor mass presents medial to the mandible. In the majority of cases the facial artery is involved and the maxillary artery is spared (Fig. 1). The angiographic appearance of salivary tumors in this location varies from an avascular mass displacing vessels to that of a hypervascular pattern.[16] Encasement or occlusion of the maxillary artery may be seen especially with malignant varieties (Fig. 13). Cylindromas of the salivary glands may display neovascularity and tumor stain. These more commonly arise in the minor salivary glands and are easily detected clinically.

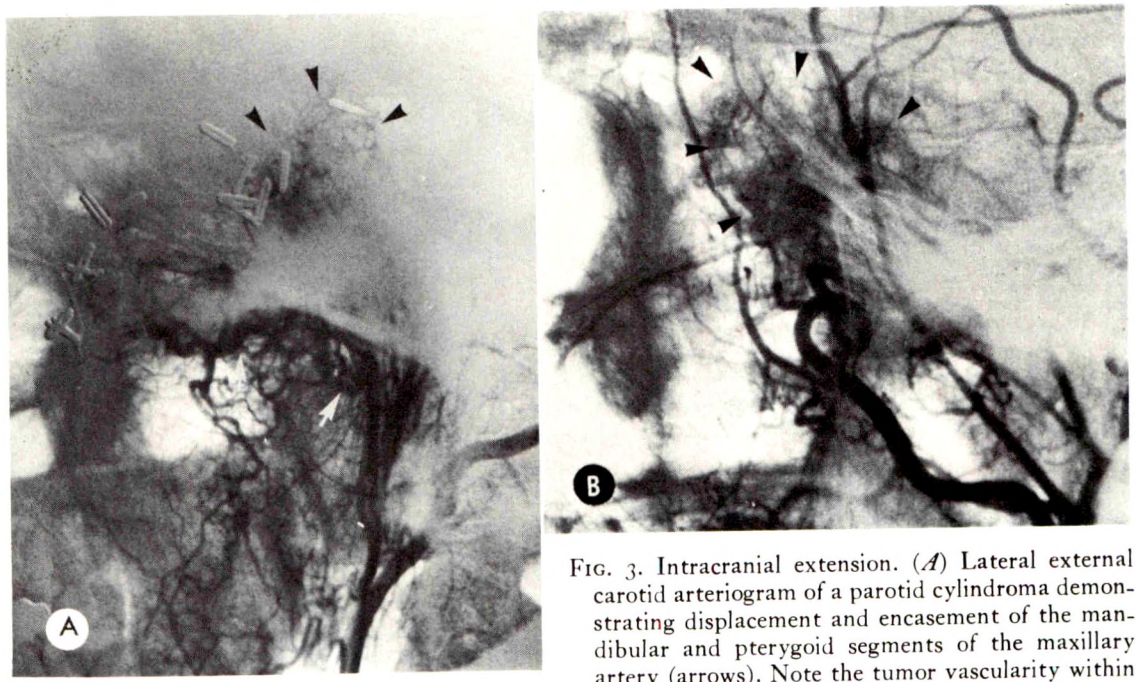


FIG. 3. Intracranial extension. (*A*) Lateral external carotid arteriogram of a parotid cylindroma demonstrating displacement and encasement of the mandibular and pterygoid segments of the maxillary artery (arrows). Note the tumor vascularity within the middle cranial fossa (arrowheads). (*B*) Lateral external carotid arteriogram in a case of nasal lymphoepithelioma showing extension of tumor into the orbit, ethmoid and sphenoid sinuses (arrowheads).

Nevertheless, they are known to spread by perineural metastasis and this route of spread is often overlooked, contributing to failures in therapy.[3,4,25] Although extension along the cranial nerves may produce changes in bone foramina and fissures, this is not always found. In such cases selective external carotid angiography may be the only method of detecting intracranial involvement by this mode of tumor extension (Fig. 3, *A* and *B*).

PTERYGOPALATINE SEGMENT

The terminal portion of the maxillary artery leaves the infratemporal fossa by turning medially through the pterygomaxillary fissure to enter the confines of the pterygopalatine fossa. Within this narrow conical structure it is well protected by bone partitions. Any alteration in its position would indicate either invasion of the fossa or erosion of its walls by tumor. This fact has proven helpful in detecting involvement of the pterygopalatine fossa by adjacent neoplasms, in particular, nasopharyngeal (Fig. 2; 4, *A* and *B*; and 5, *A–C*) and maxillary sinus carcinoma (Fig. 6*C*; and 7*C*), which are usually avascular on angiography. In addition to vascular displacement, encasement or truncation of its branches within the fossa are also indicative signs of tumor invasion[12] (Fig. 7*C*). Lesions displaying tumor neovascularity are not frequently encountered in this region. An example is the juvenile nasopharyngeal angiofibroma which characteristically invades and expands the pterygopalatine fossa and thus displaces the vascular structures within it accordingly (Fig. 5, *A–C*). Neoplasms may insidiously invade various facial compartments without producing plain film roentgenographic changes. Lymphoepitheliomas, for instance, are known to spread in this manner through foramina and fissures. Intracranial involvement is not uncommon and angiography may be the only means of detecting this type of spread (Fig. 3, *A* and *B*).

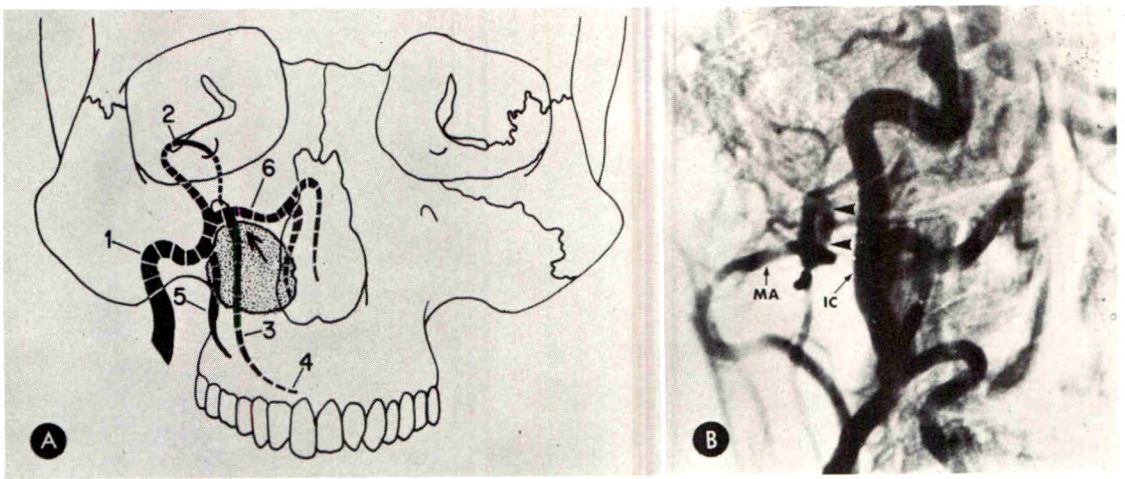


Fig. 4. Lateral displacement of the pterygopalatine portion of the maxillary artery. (*A*) Diagram of a nasopharyngeal tumor displacing the distal maxillary artery laterally. 1. Maxillary a.; 2. infraorbital a.; 3. descending palatine a.; 4. major palatine a.; 5. posterior superior alveolar a.; 6. sphenopalatine a. (*B*) Anteroposterior common carotid arteriogram in a patient with a large nasopharyngeal carcinoma which has displaced the distal portion of the maxillary artery laterally (arrowheads) indicating invasion of the pterygopalatine fossa. In this projection the distal maxillary artery is normally superimposed upon the internal carotid artery (IC). MA = maxillary artery.

VASCULAR TOPOGRAPHY OF FACIAL COMPARTMENTS

MAXILLARY SINUS

Several of the anteriorly directed branches of the third portion of the maxillary artery leave the pterygopalatine fossa to form a vascular framework around the maxillary sinus.[1] LaMarque *et al.*[12] referred to this vascular configuration as the "cercle chirurgical du sinus maxillaire." These vessels may be altered by lesions invading the walls of the maxillary sinus. The roof conducts the infraorbital branch along the infraorbital groove and canal. The greater and lesser palatine arteries descend along its posterior wall and the major palatine artery conforms to the floor of the sinus (Fig. 6*A*). The posterior superior alveolar artery travels around its lateral wall and the posterior lateral nasal branches of the sphenopalatine artery conform to its medial wall (Fig. 9*A*).

Displacement or encasement of several of these vessels by maxillary sinus carcinoma have been demonstrated on arteriography. Such findings have proven useful in detecting or confirming the direction and degree of spread of tumor, an important adjunct in the proper staging of maxillary sinus carcinoma.[12] This is especially critical in the evaluation of lesions involving the suprastructure of the maxillary sinus, where the presence of invasion into the orbit or pterygopalatine fossa has an important bearing on the proper mode of therapy. Extension into the orbit may involve the infraorbital branch of the maxillary artery either by displacement or encasement (Fig. 6*D*; and 7*B*). Invasion of carcinoma through the posterior wall into the pterygopalatine fossa can be detected by similar changes in the greater palatine artery (Fig. 6*C*; and 7*C*). LaMarque *et al.*[12] demonstrated posterior extension of maxillary sinus pathology into the pterygopalatine fossa in 10 out of 12 cases either by displacement, pressure effect or stenosis of the distal maxillary artery and its branches within the fossa. Inferior extension would be indicated by alterations in the major palatine artery (Fig. 6*B*; and 7*A*). Lateral or medial wall involvement may produce similar changes

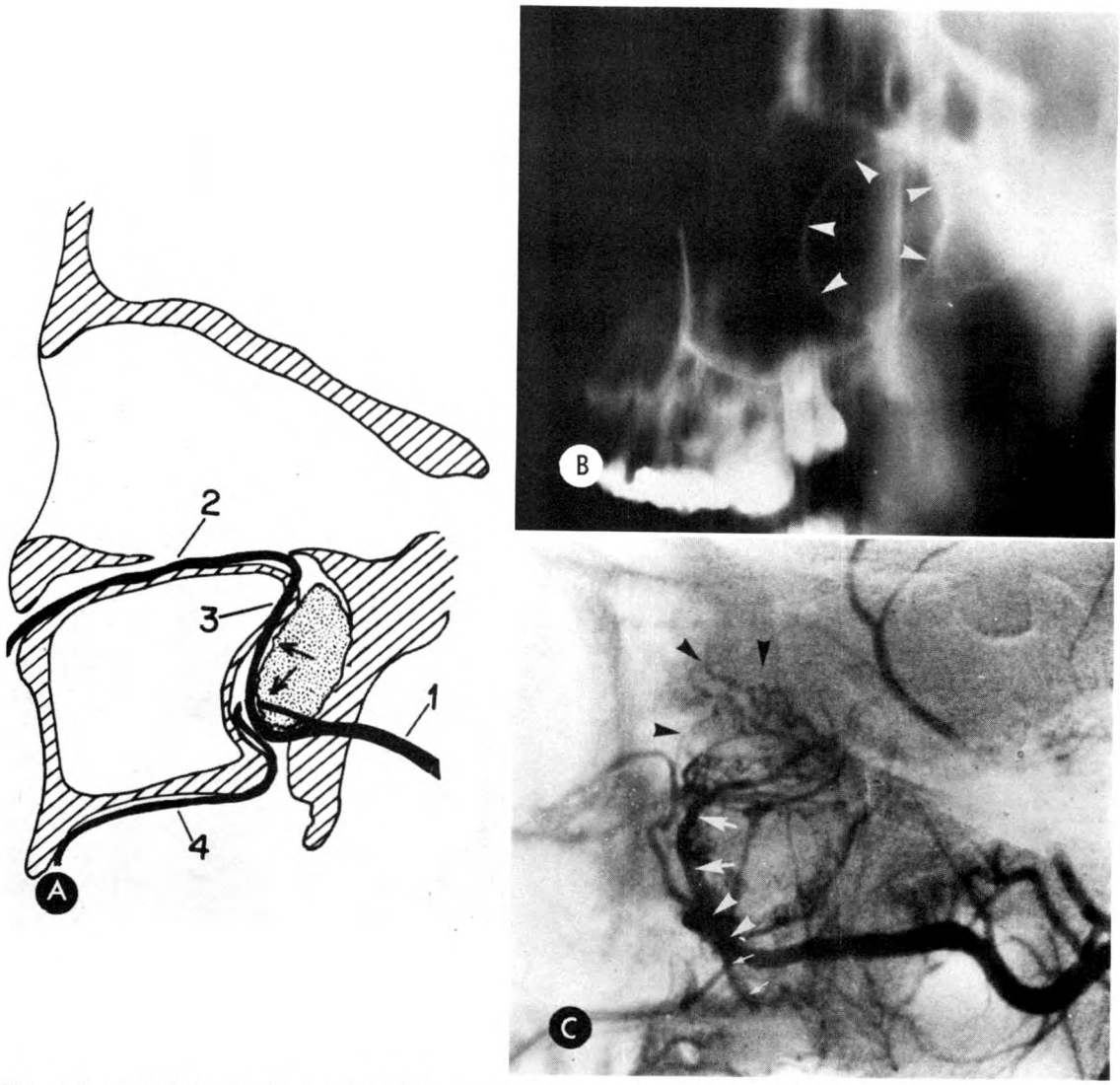
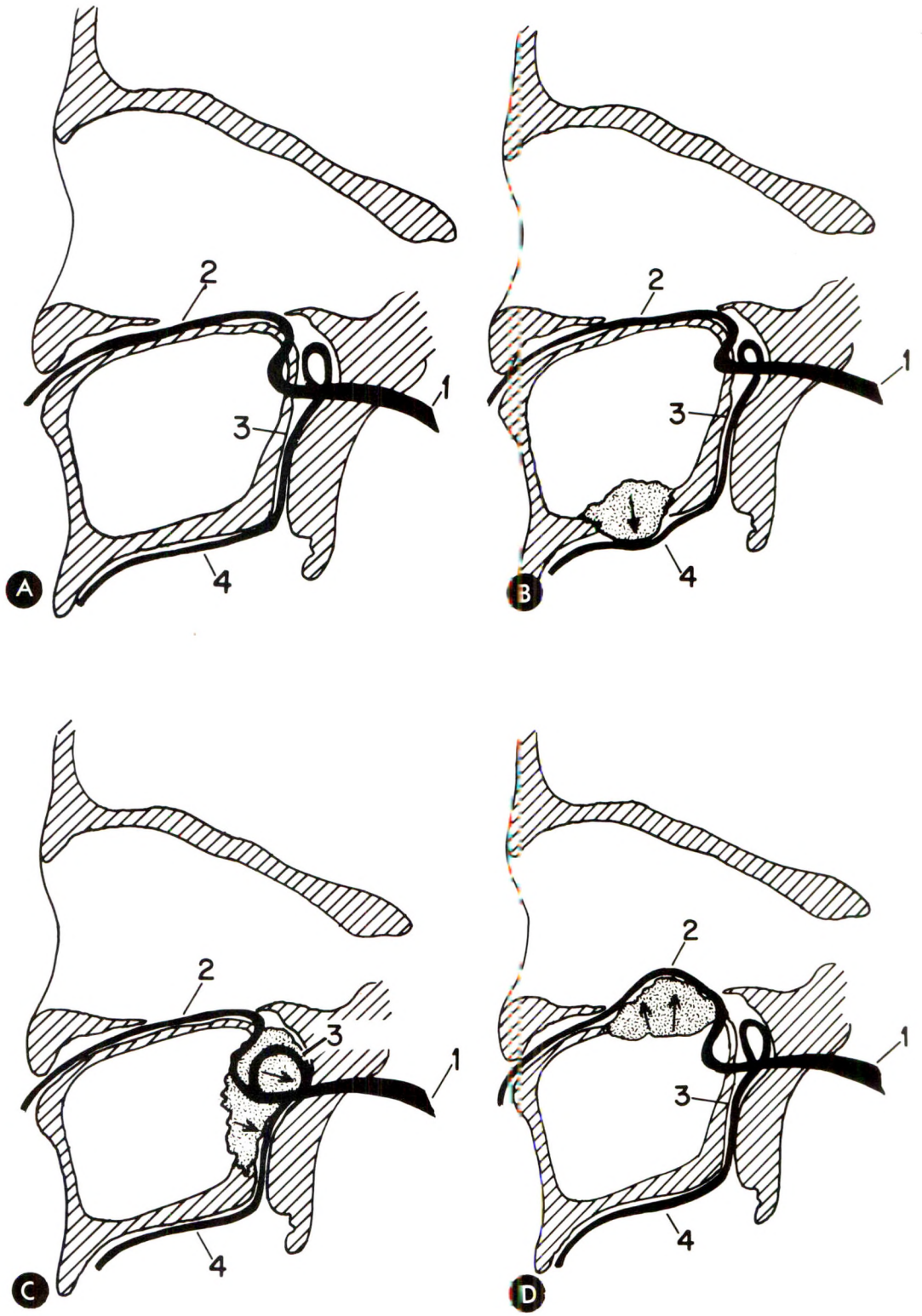


FIG. 5. Anteroinferior displacement of the pterygopalatine portion of the maxillary artery. (*A*) Diagrammatic representation of expansion of the pterygopalatine fossa by tumor with displacement of the vessels contained within it. (*B*) Lateral laminagram showing marked expansion of the fossa (arrowheads) characteristic of a juvenile nasopharyngeal angiofibroma. (*C*) Lateral external carotid arteriogram showing displacement of the distal maxillary artery downward and forward by the tumor mass (white arrowheads). The sphenopalatine artery (large white arrows) and descending palatine artery (small white arrows) are bowed anteriorly. Note the tumor vascularity within the orbit (black arrowheads). 1. Maxillary a.; 2 and 3. infraorbital a.; 4. major palatine a.

FIG. 6. Diagrammatic representations of vascular changes produced by spread of maxillary sinus tumors. (*A*) Normal. (*B*) Inferior spread through the hard palate displacing the major palatine artery. (*C*) Posterior extension into the pterygopalatine fossa displacing the descending palatine artery. (*D*) Superior extension into the orbit elevating the infraorbital artery. 1. Maxillary a.; 2. infraorbital a.; 3. descending palatine a.; 4. major palatine a.


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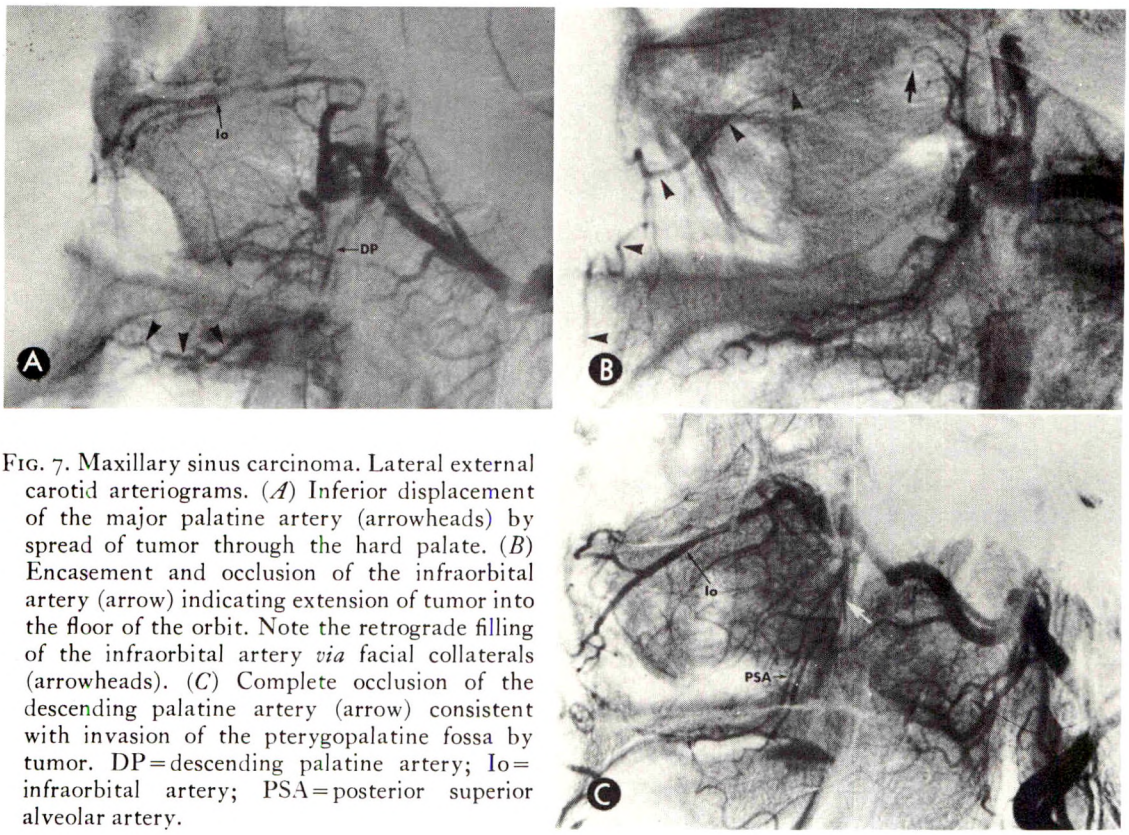


FIG. 7. Maxillary sinus carcinoma. Lateral external carotid arteriograms. (*A*) Inferior displacement of the major palatine artery (arrowheads) by spread of tumor through the hard palate. (*B*) Encasement and occlusion of the infraorbital artery (arrow) indicating extension of tumor into the floor of the orbit. Note the retrograde filling of the infraorbital artery *via* facial collaterals (arrowheads). (*C*) Complete occlusion of the descending palatine artery (arrow) consistent with invasion of the pterygopalatine fossa by tumor. DP = descending palatine artery; Io = infraorbital artery; PSA = posterior superior alveolar artery.

in the posterior superior alveolar and posterior lateral nasal arteries respectively.

ORBIT

The infraorbital artery leaves the pterygopalatine fossa through the inferior orbital fissure. It courses over the superoposterior aspect of the maxillary sinus and then along the floor of the orbit first within a bone groove and then through the infraorbital canal. Within the orbit it gives rise to muscular branches and the anterior superior alveolar arteries. Intraorbital tumors may derive blood supply from this vessel, particularly meningiomas[11] and rhabdomyosarcomas. External carotid arteriography should be included in their evaluation. Extension of masses through the floor of the orbit into the roof of the maxillary sinus may encase or displace this vessel inferiorly (Fig. 8, *A* and *B*) and, conversely, superior extension of maxillary sinus tumors may elevate this vessel (Fig. 6*D*; and 7*B*). Spread of tumor by perineural invasion of the infraorbital nerve has been encountered.[3,9] As the nerve and artery are in close proximity along the course of the infraorbital groove and canal, vascular changes would suggest this mode of spread. Invasion through the inferior orbital fissure, transmitting the infraorbital artery, may also be detected by angiography.

NASAL CAVITY AND NASOPHARYNX

The sphenopalatine artery enters the nasal cavity through the sphenopalatine foramen and its branches conform to the walls of the ipsilateral half of the nasal cavity. The posterior lateral nasal branches course over the lateral nasal wall and the posterior septal branches over the nasal septum (Fig. 9*A*). In the lateral projection these vessels are difficult to identify; however, they can be defined on the anteroposterior or Towne projections using subtrac-

tion techniques.[1] Expansion or erosion of the nasal cavity may be identified by displacement or encasement of these branches (Fig. 9*B*).

Vascular tumors in this region are rare. Again, the most common vascular lesion in this location is the juvenile nasopharyngeal angiofibroma. This lesion is extremely vascular and receives its blood supply primarily from the sphenopalatine branches of the maxillary artery (Fig. 9*B*). Other branches of the maxillary artery may be involved, in particular, the pharyngeal artery. Supply from the ascending pharyngeal branch of the external carotid artery is also common.[27] These tumors are benign and slow growing, expanding bony facial compartments (Fig. 5; and 9*B*). They characteristically extend along foramina and fissures in all directions and displace rather than invade bony structures. Supply from ophthalmic, dural, cavernous and petrous branches of the internal carotid artery is occasionally encountered[17,27] (Fig. 12). Selective external and internal carotid angiography is extremely helpful in the preoperative evaluation of these lesions. If their entire blood supply is defined and ligated, the incidence of recurrences can be significantly reduced. Equally as important is the marked reduction in operative blood loss once the feeders are identified and controlled. If angiography shows that the supply is solely from the distal branches of the maxillary artery, bleeding can be easily controlled by a transantral maxillary artery ligation.[21]

Carcinomas in this area, as in any location, are predominantly avascular and arteriography is of little help when these tumors are confined to the nasopharynx. Occasionally hypertrophy and displacement of the pharyngeal branch of the maxillary artery are encountered (Fig. 10). This branch initially courses posteriorly through the palatinovaginal canal or groove along the inferior surface of the body of the sphenoid bone.[1] Encasement of this segment is consistent with invasion by tumor into the sphenoid or posterior ethmoid regions.

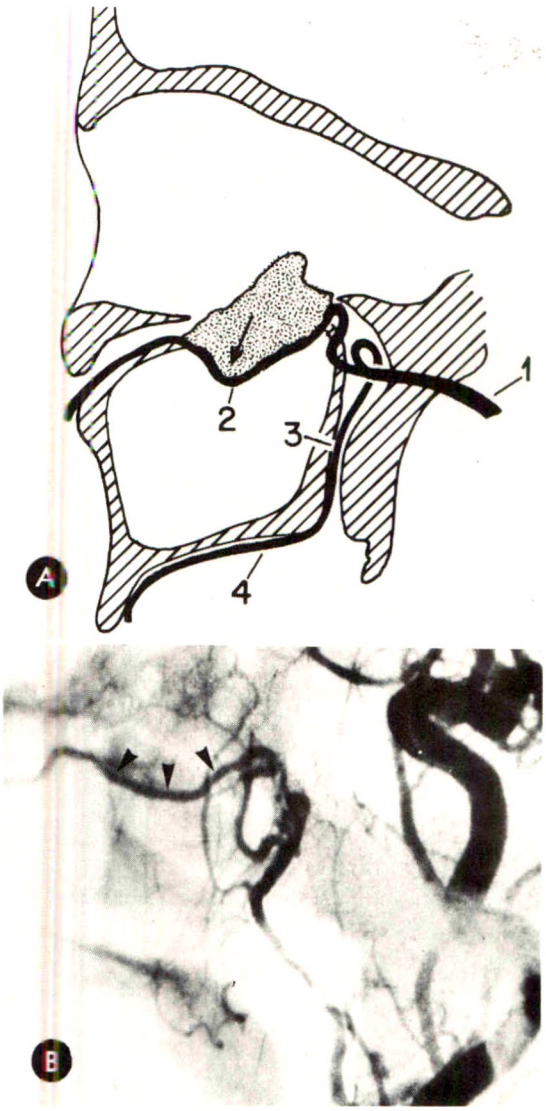


Fig. 8. Extension of orbital masses into the maxillary sinus. (*A*) Diagrammatic representation of an orbital mass depressing the infraorbital artery. (*B*) Lateral external carotid arteriogram in a patient with a blow out fracture of the orbital floor. The infraorbital artery is displaced inferiorly by the soft tissue mass (arrowheads). 1. Maxillary artery; 2. infraorbital artery; 3. descending palatine artery; 4. major palatine artery.

EPISTAXIS

Most cases of spontaneous epistaxis in adults result from sclerotic changes in branches of the maxillary artery, especially the sphenopalatine artery.[2] Uncontrollable epistaxis following trauma more often in-

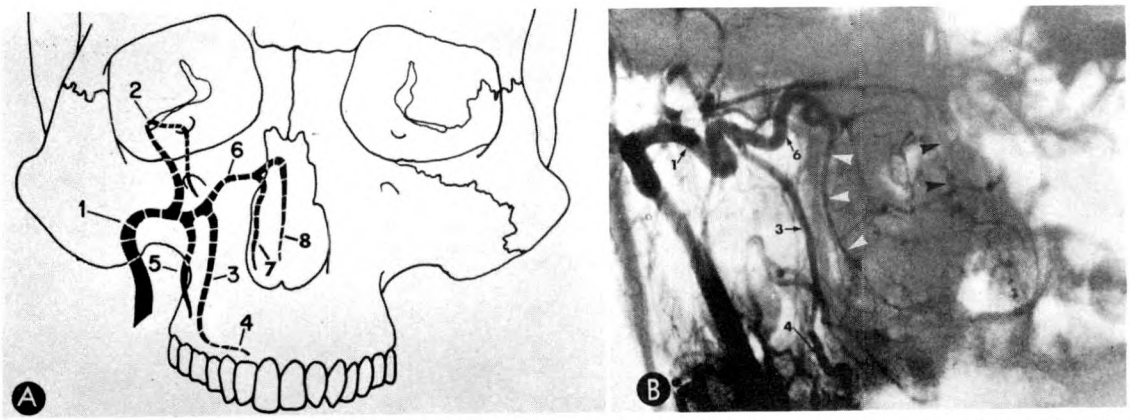


FIG. 9. Sphenopalatine branches in the anteroposterior projection. (*A*) Diagram of the normal vascular distribution in the anteroposterior projection. (*B*) External carotid arteriogram of a juvenile nasopharyngeal angiofibroma. The tumor was supplied solely from the sphenopalatine branch. Expansion of the nasal cavity by tumor is manifested by the lateral displacement of the posterior lateral nasal branch (white arrowheads) and the displacement of the posterior septal branch across the midline (black arrowheads). 1. Maxillary a.; 2. infraorbital a.; 3. descending palatine a.; 4. major palatine a.; 5. posterior superior alveolar a.; 6. sphenopalatine a.; 7. posterior lateral nasal branch; 8. posterior septal branch of the sphenopalatine a

volves the ethmoidal branches of the internal carotid system; however, maxillary artery branches are not infrequently involved.[5] Many cases of recurrent or intractable epistaxis are treated by maxillary artery ligation without ever knowing the exact site of bleeding or the precise etiology. Clinical examination often fails to detect which vessels are responsible and even laterality may be impossible to determine. Certain vascular tumors may also be responsible, especially angiomas[22] and angiofibromas.[27]

Angiographic evaluation of the maxillary artery could provide essential information in such cases and reduce the incidence of operative failures. In addition to identifying vascular neoplasms, extravasation of contrast medium has been demonstrated and found helpful in identifying bleeding sites.[2,5] Recurrent epistaxis following surgical intervention presents a difficult problem and, in this situation, collateral systems to the maxillary artery should be evaluated angiographically.

INTRACRANIAL COLLATERAL SYSTEMS

The maxillary artery gives rise to many of the major collaterals between the external and internal carotid systems.[6,13] All 3 segments of the maxillary artery take part in these collaterals which anastomose with the petrous, cavernous and intradural portions of the internal carotid artery (Table 1).

A. OPHTHALMIC COLLATERALS

The communications with the internal

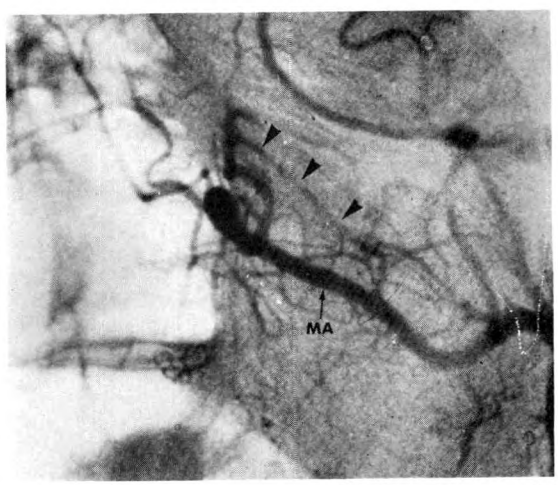
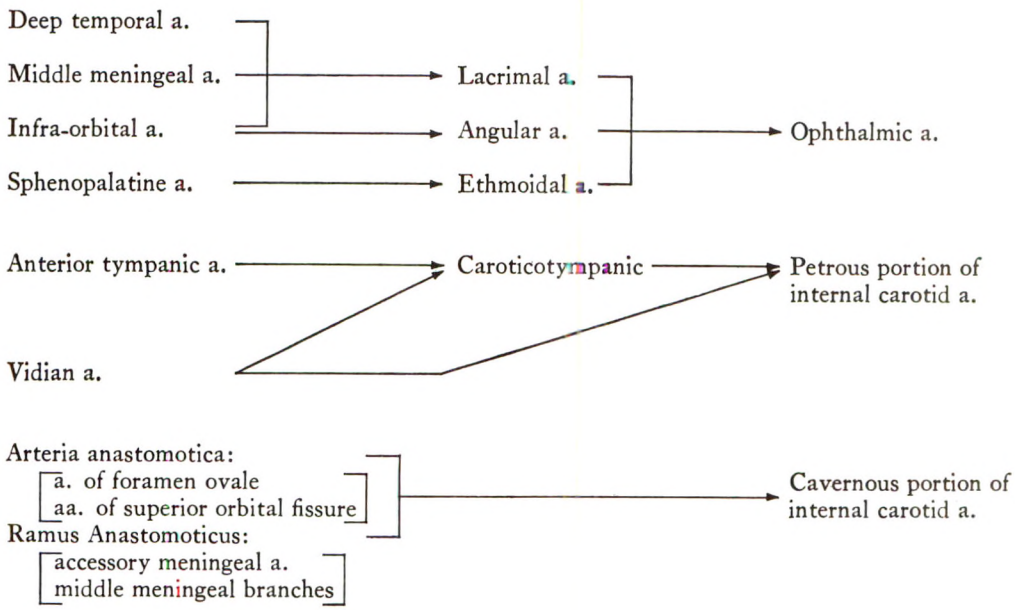


FIG. 10. Pharyngeal artery. Lateral external carotid arteriogram demonstrating the pharyngeal artery (arrowheads) which is enlarged and stretched in a patient with a nasopharyngeal carcinoma. MA = maxillary artery.

TABLE I

INTRACRANIAL COLLATERAL SYSTEMS OF THE MAXILLARY ARTERY*



* Modified from Lie, T. A.[13]

carotid artery by retrograde flow through the ophthalmic artery have been demonstrated frequently in instances of occlusion of the internal carotid artery (Fig. 11, *A* and *B*). Several branches of the maxillary artery may take part; namely, the sphenopalatine, infraorbital, middle meningeal and deep temporal arteries.[6,13]

B. PETROUS CAROTID COLLATERALS

Less well recognized are the communications with the cavernous and petrous portions of the internal carotid artery. The pterygopalatine portion of the maxillary artery gives rise to 3 posteriorly directed branches which course through openings in the sphenoid bone.[1] Medially the pharyngeal artery leaves the pterygopalatine fossa by way of the palatinovaginal canal or groove and is distributed to the upper part of the nasopharynx and the auditory tube. It anastomoses with other extracranial vessels such as the ascending pharyngeal and ascending palatine arteries. Slightly more laterally the Vidian artery (artery of the pterygoid canal) courses posteriorly through the Vidian canal toward the foramen lacerum. This vessel is a remnant of the first aortic arch and may persist as a direct collateral with the petrous portion of the internal carotid artery.[13] Although the Vidian artery is described as a branch of the pterygopalatine portion of the maxillary artery, it has also been described as arising from the petrous portion of the internal carotid artery. Regardless of its main origin, it is one and the same vessel traversing the Vidian canal. Recently it has been demonstrated angiographically and has been shown to supply nasopharyngeal tumors.[15] It is also an important vessel to recognize in the evaluation and treatment of intractable epistaxis. Retrograde filling of the maxillary artery by the Vidian artery may take place if this vessel is not included in maxillary artery ligation.[21] The Vidian artery also communicates with the anterior tympanic branch of the first (mandibular) portion of the maxillary artery and the caroticotympanic branch of the internal

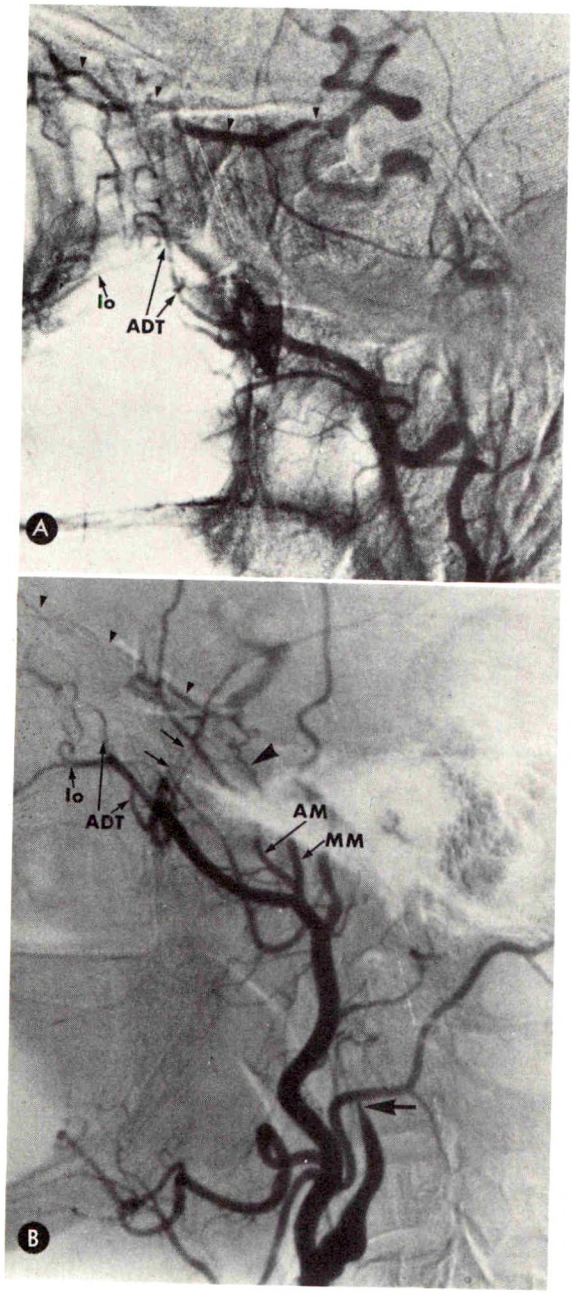


FIG. 11. Intracranial collateral systems of the maxillary artery. (*A* and *B*) Lateral carotid arteriograms with complete occlusion of the internal carotid artery (arrow). The intracranial circulation is reconstituted by retrograde ophthalmic flow (small arrowheads) filling from branches of the infraorbital (Io) and anterior deep temporal (ADT) arteries. In *B* there may be filling of the internal carotid from a ramus anastomoticus (large arrowhead) and the arteria anastomotica (small arrows). AM = accessory meningeal a.; MM = middle meningeal a.

carotid artery within the tympanic cavity.[7,13] Other communications of the maxillary artery with the petrous portions of the internal carotid artery may be encountered angiographically, especially in cases of vascular nasopharyngeal tumors (Fig. 12).

C. CAVERNOUS COLLATERALS

Further laterally, a third posteriorly directed branch, the artery of the foramen rotundum, enters the cranial vault through its corresponding foramen. It is thought to anastomose with the cavernous portion of the internal carotid artery, in particular, with the artery of the inferior cavernous sinus.[8,15,21] Other cavernous collaterals traverse the lateral aspect of the superior orbital fissure. Collectively these posteriorly directed branches through the foramen rotundum and superior orbital fissure have been referred to as the arteria anastomotica[13] (Table 1). Retrograde filling of the maxillary artery through such collaterals has been seen following occlusion of the proximal maxillary artery[15] (Fig. 13). These vessels may also assist in filling the intracranial circulation in cases of occlusion

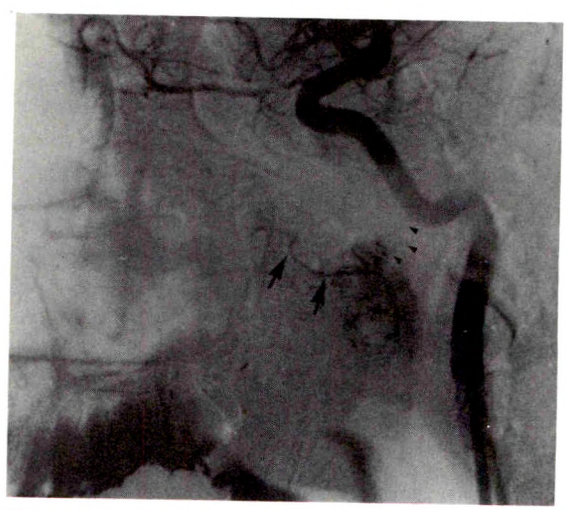


FIG. 12. Petrous collateral. A selective internal carotid arteriogram in a patient with a juvenile nasopharyngeal angiofibroma. The tumor is partly supplied by a branch from the petrous portion of the internal carotid artery (arrowheads) with some retrograde filling of maxillary artery branches (arrows).

of the internal carotid artery[6,13] (Fig. 11*B*).

Another potential anastomosis with the cavernous internal carotid artery is the accessory meningeal artery which enters the cranium through the foramen ovale. This vessel, plus cavernous communications from the middle meningeal artery have been collectively referred to as the ramus anastomoticus[13] (Table 1).

SUPPLY TO INTRACRANIAL LESIONS

Basically, the arteria anastomotica and the ramus anastomoticus represent meningeal collaterals as they contribute to the supply of the dura of the cavernous sinus and surrounding regions along the base of the skull. It is not surprising, therefore, that these branches of the maxillary artery may contribute to the vascular supply of intracranial neoplasms and vascular malformations involving the dura in these areas. These branches have been shown to supply dural arteriovenous shunts in the region of the cavernous sinus[18,19] and contribute a significant supply to carotico-cavernous fistulas[8] (Fig. 14*A*). Consequently, selective external carotid arteriography is mandatory in the evaluation of these lesions if therapy is to be successful. Intracerebral arteriovenous malformations may also derive vascular supply from the meningeal collaterals of the distal maxillary artery (Fig. 14*B*).

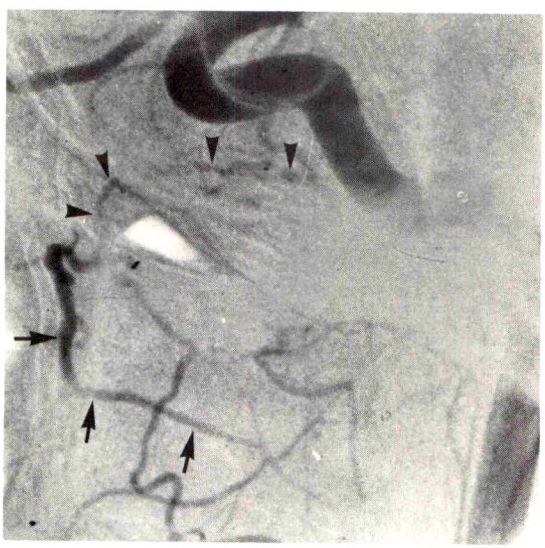


Fig. 13. Retrograde filling of the maxillary artery (arrows) through an arteria anastomotica (arrowheads) arising from the cavernous portion of the internal carotid artery. The proximal maxillary artery had been occluded by a parotid tumor.

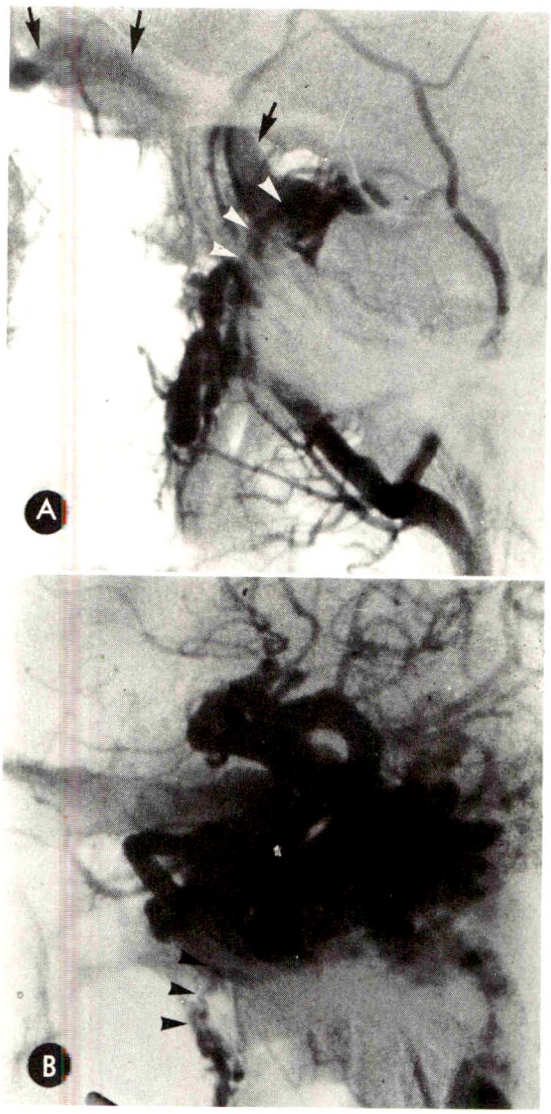


Fig. 14. Supply to intracranial vascular lesions. (*A*) A large tortuous meningeal collateral (arteria anastomotica) arises from the distal maxillary artery (arrowheads) supplying a carotico-cavernous fistula. Note the opacification of the cavernous sinus and ophthalmic veins (arrows). (*B*) A similar meningeal branch (arrowheads) acting as a feeder to an arteriovenous malformation of the temporal lobe.

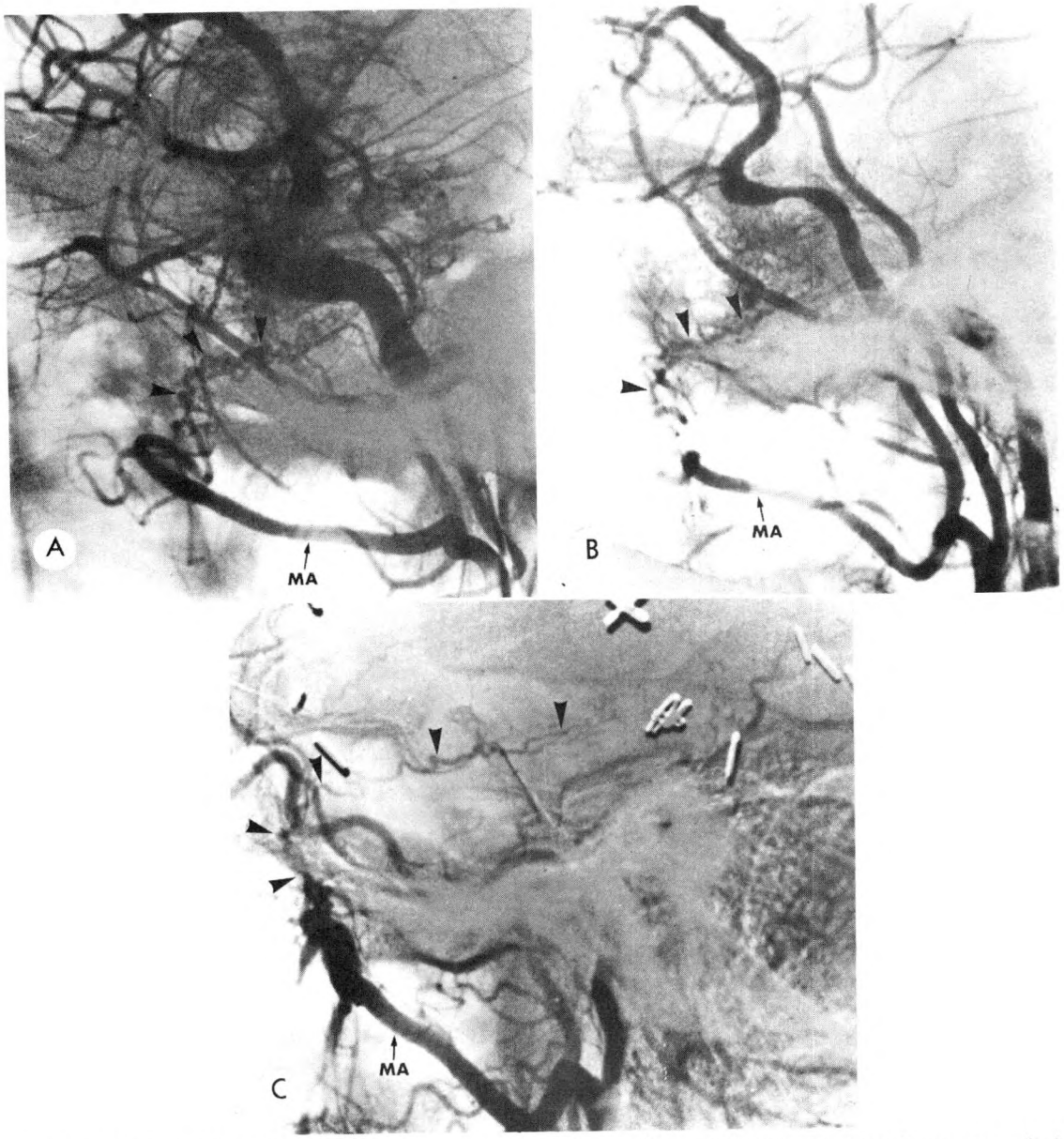


FIG. 15. Meningeal branches of the pterygopalatine portion of the maxillary artery (arteria anastomotica). (*A* and *B*) Two separate cases of sphenoid ridge meningiomas supplied by a large tortuous meningeal branch (arrowheads) arising from the distal maxillary artery. (*C*) Lateral selective external carotid arteriogram showing a large meningeal branch (arrowheads) from the distal maxillary artery supplying a recurrent tentorial meningioma.

Neoplasms in the region of the cavernous sinus and surrounding areas may derive some of their blood supply from these meningeal collaterals if such lesions involve the dura. Rarely gliomas may have a dural supply. Characteristically parasellar and sphenoid ridge meningiomas are supplied by branches of the middle meningeal artery and/or the meningohypophyseal trunk. Supply by meningeal collaterals from the accessory meningeal artery (ramus anastomoticus) and from the branches of the pterygopalatine portion of the maxillary artery (arteria anastomotica) might be ex-

pected of meningiomas in this location (Fig. 15, *A* and *B*). In some instances the distal meningeal branches of the maxillary artery represent the major vascular supply. In 1 case the distal maxillary artery gave rise to a meningeal feeder to a tentorial meningioma (Fig. 15*C*).

SUMMARY

An angiographic evaluation of the maxillary artery and its branches provides a means of detection and diagnosis for many facial as well as intracranial lesions.

Its distal branches form a vascular framework around the maxillary sinus and nasal cavity and outline the bony septa of the adjacent orbital floor, pterygopalatine fossa and the roof of the nasopharynx. Extension of tumors across these facial compartments and into the cranial fossa, often difficult to detect by other techniques, can be detected by this method.

Although most tumors of the nasopharynx and paranasal sinuses have sparse vascularity, changes in vascular topography will provide information concerning the direction and degree of spread. This information has proved useful in establishing the optimum mode of therapy.

The collateral systems of the maxillary artery are reviewed and their importance in the evaluation of intractable epistaxis, occlusive vascular disease, vascular malformations and shunts, and facial as well as intracranial neoplasms are discussed.

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REFERENCES

1. Allen, W. E., III, Kier, E. L., and Rothman, S. L. G. Maxillary artery: normal arteriographic anatomy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 517–527.
2. Coel, M. N., and Janon, E. A. Angiography in patients with intractable epistaxis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 37–40.
3. Dodd, G. D., Dolan, G. A., Ballantyne, A. J., Ibanez, M. D., and Chan, P. Dissemination of tumors of head and neck via cranial nerves. *Radiol. Clin. North America*, 1970, *8*, 445–461.
4. Dodd, G. D., and Jing, B. Radiographic findings in adenoid cystic carcinoma of head and neck. *Ann. Otol., Rhin. & Laryng.*, 1972, *81*, 1–8.
5. Duggan, C. A., and Brylski, J. R. Angiographic demonstration of bleeding in intractable traumatic epistaxis. *Radiology*, 1970, *97*, 605–606.
6. Fields, W. S., Bruetman, M. E., and Wiebel, J. Collateral circulation of brain. In: Monographs in the Surgical Sciences. Williams & Wilkins Company, Baltimore, 1965, pp. 183–259.
7. Gray's Anatomy. Thirty-fourth edition. Edited by D. V. Davis, and R. E. Coupland. Longmans, Green & Co., Ltd., London, 1967, p. 794.
8. Hayes, G. J. External carotid cavernous sinus fistulas. *J. Neurosurg.*, 1963, *20*, 692–700.
9. Jafek, B. W., and Ward, P. H. Infraorbital perineural invasion by tumor. *Radiology*, 1971, *101*, 617–618.
10. Jing, B. Tumors of nasopharynx. *Radiol. Clin. North America*, 1970, *8*, 323–342.
11. Kendall, B. Invasion of facial bones by basal meningiomas. *Brit. J. Radiol.*, 1973, *46*, 237–244.
12. LaMarque, J. L., Ginestié, J. F., Serrou, B., Fabre, J. M., and Sénac, J. P. Apport de l'artériographie carotidienne externe dans l'exploration radiologique des tumeurs du sinus maxillaire. *J. de radiol., d'électrol. et de méd. nucléaire*, 1971, *52*, 357–366.
13. Lie, T. A. Congenital Anomalies of the Carotid Arteries. Excerpta Medica Foundation, The Netherlands. 1968.
14. Lurjé, A. On topographical anatomy of internal maxillary artery. *Acta anat.*, 1946, *2*, 219–231.
15. Margolis, M. T., and Newton, T. H. Collateral pathways between cavernous portion of internal carotid and external carotid arteries. *Radiology*, 1969, *93*, 834–836.
16. Martinez-Mora, J., Bloix-Ochoa, J., and Tresserra, L. Vascular tumors of parotid region in children. *Surg., Gynec. & Obst.*, 1971, *133*, 973–977.
17. Medellin, H., and Wallace, S. Angiography in neoplasms of head and neck. *Radiol. Clin. North America*, 1970, *8*, 307–321.
18. Newton, T. H., and Hoyt, W. F. Spontaneous arteriovenous fistula between dural branches of internal maxillary artery and posterior cavernous sinus. *Radiology*, 1968, *91*, 1147–1150.
19. Newton, T. H., and Hoyt, W. F. Dural arteriovenous shunts in region of cavernous sinus. *Neuroradiology*, 1970, *1*, 71–81.

20. PAULUS, D. D., JR., and DODD, G. D. Roentgen diagnosis of tumors of nasal cavity and accessory paranasal sinuses. *Radiol. Clin. North America*, 1970, *8*, 343–360.

21. PEARSON, B. W., MACKENZIE, R. G., and GOODMAN, W. S. Anatomic basis of transantral ligation of maxillary artery in severe epistaxis. *Laryngoscope*, 1969, *79*, 969–984.

22. PUPP, L., and HUSZAR, L. Angioma of internal maxillary artery producing massive recurrent epistaxis. *J. Laryng. & Otol.*, 1972, *86*, 147–154.

23. RIZZUTI, R. J., and WHALEN, J. P. Nasopharynx. In: Radiology of the Skull and Brain. Book I. Volume I. Edited by T. H. Newton, and D. G. Potts. C. V. Mosby Company, St. Louis, 1971, pp. 241–259.

24. SAKAI, S., SHIGEMATZU, Y., and FUCHIHATA, H. Diagnosis and TNM classification of maxillary sinus carcinoma. *Acta oto-laryng.*, 1972, *74*, 123–129.

25. SWASH, M. Invasion of cranial nerves by cylindroma: four cases treated by radiotherapy. *J. Neurol., Neurosurg. & Psychiat.*, 1971, *34*, 475–480.

26. TEAL, J. S., RUMBAUGH, C. L., SEGALL, H. D., and BERGERON, R. T. Anomalous branches of internal carotid artery. *Radiology*, 1973, *106*, 567–573.

27. WILSON, G. H., and HANAFEE, W. N. Angiographic findings in 16 patients with juvenile nasopharyngeal angiofibroma. *Radiology*, 1969, *92*, 279–285.

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OS SUPRA PETROSUM OF MECKEL*

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AMONG the various types of calcifications which may be encountered at the tip of the petrous bone is a little known ossicular structure first mentioned by J. F. Meckel[4] in 1748, and named *os supra petrosum* by Gruber[2] in 1869. Staurenghi[6] (1900) employed the term *ossicolo petroso prearmillare o preganglionare* based on its relation to the trigeminal ganglion (ganglion prearmillare). Le Double[3] (1903) described it in some detail under a similar name. The most recent review of the subject is that by Redlich[5] (1963) in a paper reporting the first roentgen demonstration of the ossicle in 5 living children.

DESCRIPTION

The os supra petrosum (O.S.P.) is described as a small ossicle, pea-sized at the most, located on the anterosuperior surface of the petrous bone, near the tip of this bone and just anterior and medial to the ganglion of Gasser. It lies under the dura either independent from it or adherent to it. It is usually bilateral and symmetrical and has apparently been observed at all ages. Its incidence is not established. It has been said[1] to occur as frequently as in 10 per cent of individuals, but this figure is probably exaggerated and at least roentgenographically it is an uncommon finding as already pointed out by Redlich.[5] Its nature and mode of development are also unknown. Le Double[3] considered it as a localized ossification of the dura from which it becomes independent only secondarily. The finding does not seem to have any clinical significance, but may be important in the differential diagnosis of intracranial calcifications.

MATERIAL

We have had the opportunity to observe this ossicle in the skull roentgenograms of 5 children with a 5 year follow-up in 1 of the cases (Case 1), and a detailed postmortem study in another (Case v). The findings are illustrated in Figures 1–5 and the main clinical features are given in the legends. In this series of cases, the ossicle was best seen in the straight anteroposterior roentgenograms with the petrous ridges projecting through the orbit, and in the Stenver's view. In both views, the ossicle was superimposed on the upper border of, or pro-

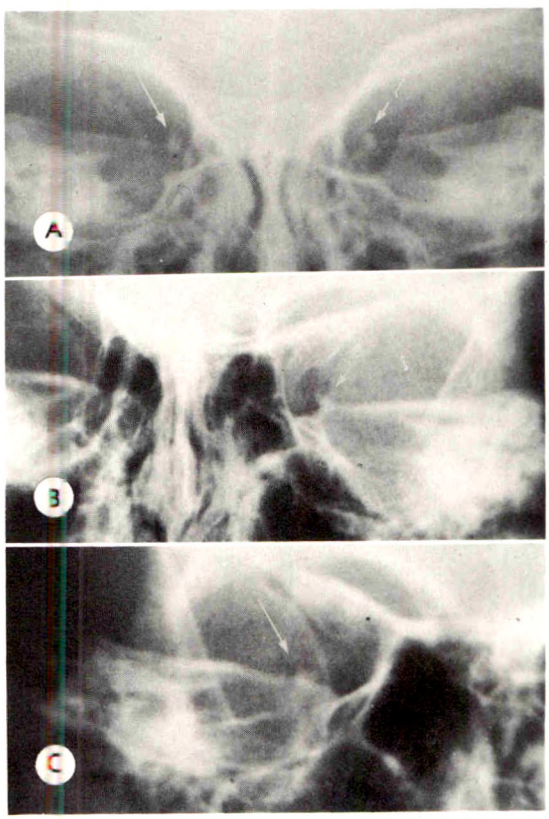


FIG. 1. Case 1. Two year old female.
(*A*) Bilateral os supra petrosum (O.S.P.) was noted in skull roentgenograms obtained because of convulsions. (*B* and *C*) Re-examination 5 years later showed identical findings.

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FIG. 2. Case II. Ten month old male.
Bilateral O.S.P. was noted in skull roentgenogram obtained because of multiple congenital anomalies.

jected slightly above, the ossified tip of the petrous bone. It could also be identified in the lateral view slightly anterior to the tip of the petrous bone which normally projects on, or slightly anterior to the sphenooccipital synchondrosis.

In the specimen obtained at postmortem in Case V, the region of the right petrous tip was dissected, and the region of the left tip was decalcified and embedded for sectioning in a plane parallel to petroclinoid ligament. Upon lateral reflection of the right trigeminal ganglion the right os supra petrosum lay exposed (Fig. 6, *A* and *B*). It was a slightly movable oval structure

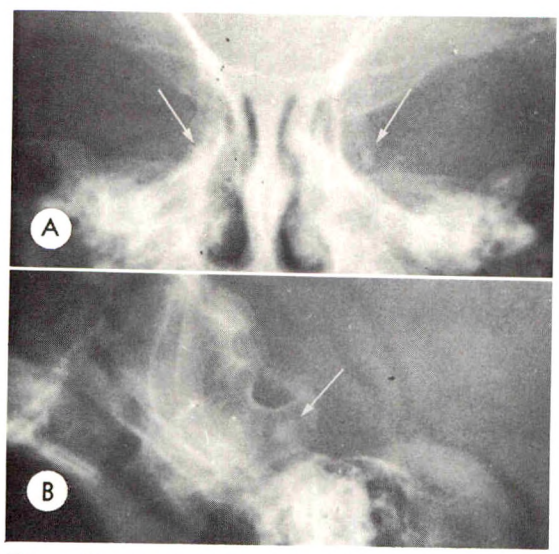


FIG. 3. Case III. Three month old male.
(*A*) O.S.P., at least on the left, was noted on skull roentgenogram obtained following head trauma. (*B*) O.S.P. in the lateral view of skull.

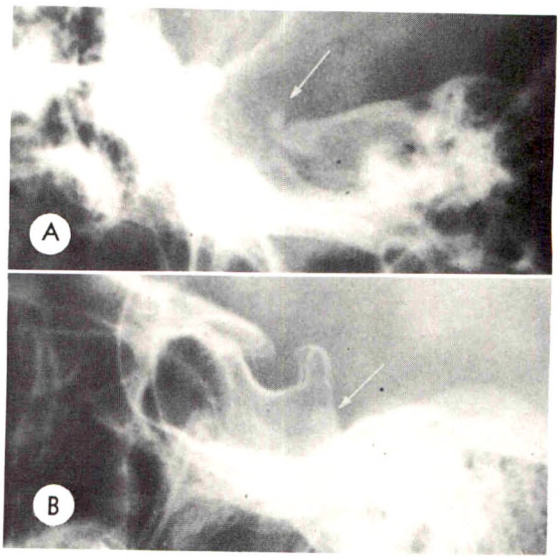


FIG. 4. Case IV. Five year old female.
(*A*) O.S.P., at least on the left, was noted in roentgenogram of the sinuses and mastoids obtained because of fever of undetermined origin. (*B*) O.S.P. in the lateral view of skull.

closely enveloped by the dura. It projected 1 mm. above the floor of the trigeminal recess. It lay in an angle formed by the anterior margin of the portio minor of the trigeminal nerve running beneath the ganglion and the inferior surface of the anterior limb of the ganglion. A delicate fold of dura extended laterally from the

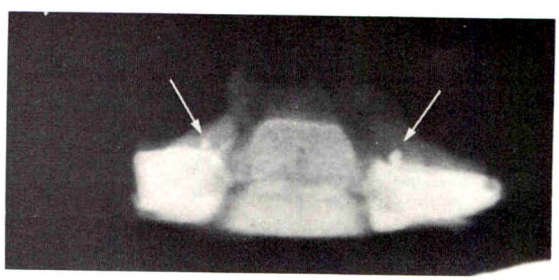


FIG. 5. Case V. Three month old female.
Bilateral O.S.P. was noted on skull roentgenogram obtained because of convulsions and clinical findings of increased intracranial pressure later found to be caused by a cerebral teratoma. The patient expired following surgery. At postmortem examination, a block of bone including the area of each petrous tip, the basal occiput and posterior sphenoid was removed.
Roentgenogram of this specimen clearly shows bilateral O.S.P.

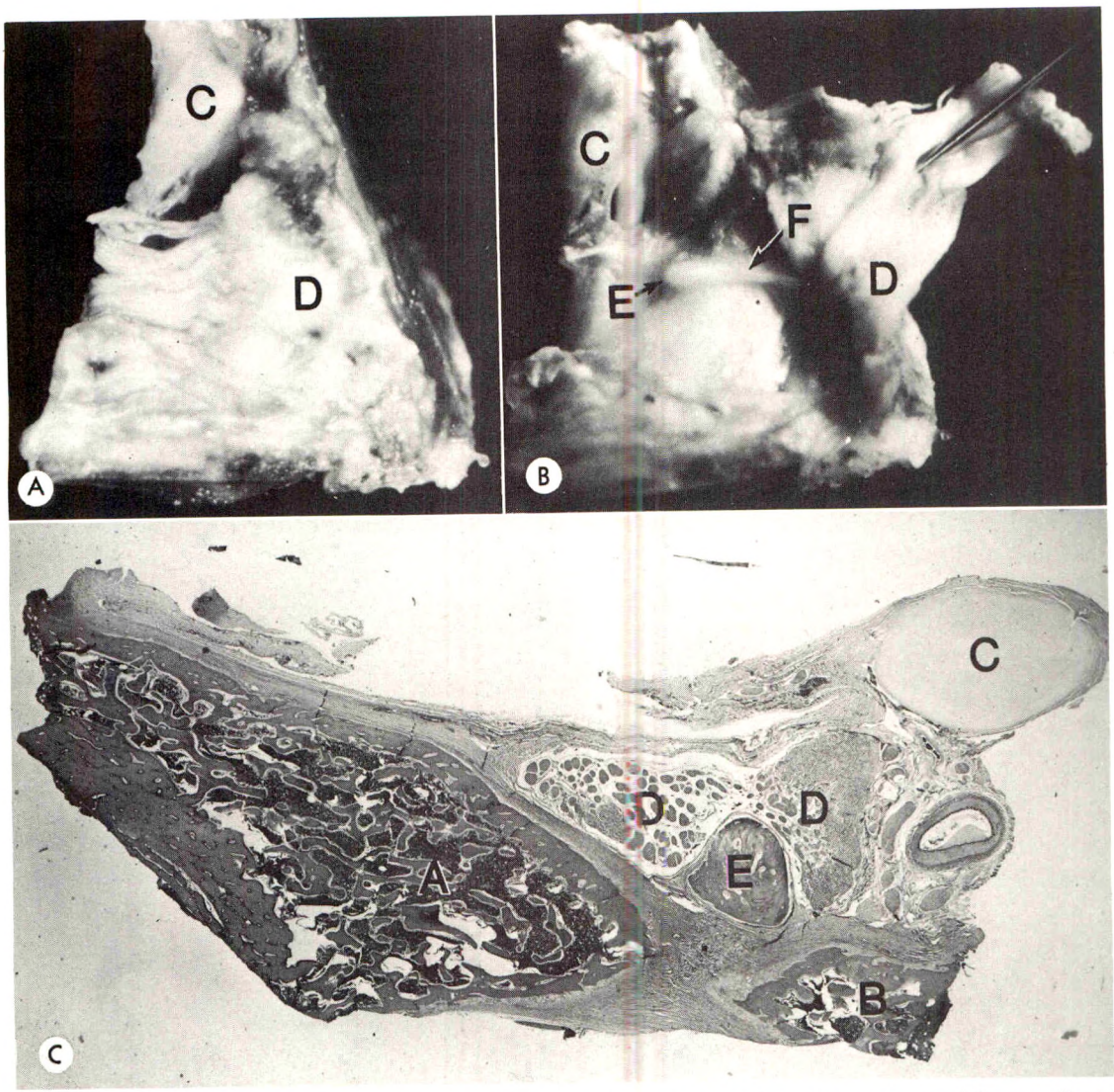


Fig. 6. Postmortem study in Case v. (*A*) The right trigeminal ganglion viewed from above with petroclinoid ligament and overlying dura removed. (*B*) Same view with ganglion reflected laterally. The portio minor of the trigeminal nerve courses beneath the ganglion. The O.S.P., covered by dura, projects from the floor of the trigeminal fossa. A fold of dura (E) extends laterally. (*C*) Section through left O.S.P. parallel to petroclinoid ligament. Note the thin investment of dura around the bone. The carotid artery is seen beneath the posterior clinoid. (A) Petrous bone; (B) sphenoid bone; (C) posterior clinoid; (D) trigeminal ganglion and nerve; (E) O.S.P.; (F) ligament of O.S.P.

os along the margin of the portio minor. This ran across the foramen lacerum to the sphenoid bone where it joined the dura encircling the foramen ovale. This fold of dura could be referred to as the ligament of the O.S.P. A less prominent fold of dura ran from the os to the posterior margin of the superior orbital fissure. The os lay above the periosteum covering the tip of the petrous bone. There was no concavity in the underlying petrous tip. The left O.S.P. was shown by histologic sections (Fig. 6*C*) to have a similar relationship to the gasserian ganglion. However, it was located slightly more anterior so as to overlie in part the dense, fibrous connective tissue of the foramen lacerum. It appeared to lie entirely within the dura.

CONCLUSION AND SUMMARY

Attention is called to a little known cranial ossicle, the os supra petrosum (O.S.P.), located within the dura at the tip of the petrous bone.

It is an anatomic variant which may be seen quite clearly in the roentgenograms. It is of no apparent clinical significance but should be recognized in the differential diagnosis of intracranial calcifications.

Five children with this roentgen finding are reported together with some postmortem observations, both gross and histologic, in 1 of the cases.

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REFERENCES

1. Caldani, F. Nuovi elementi di anatomia. Venezia, 1824. Quoted by Le Double.[3]
2. Gruber, W. Beiträge zur Anatomie des Schädelgrundes. Mémoires de l'Académie Impériale des Sciences de St. Petersbourg, VII. Série, Tome XIII, Nr. 7 (1869), P. 26–27. Quoted by Redlich.[5]
3. Le Double, A.-F. Traité des variations des os du crâne chez l'homme et de leur signification au point de vue de l'anthropologie zoologique. Vigot Frères, Éditeurs. Paris, 1903, pp. 337–338.
4. Meckel, J. F. Tract. anat. phys. de quinto pare nervorum cerebri. Goettingae, 1748. Quoted by Le Double.[3]
5. Redlich, F. H. Röntgenologischer Erstnachweiss des Os supra-petrosum (Meckel). *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1963, *98*, 599–604.
6. Staurenghi. Nuove osservazioni di craniologia. Pavia, 1900. Quoted by Le Double.[3]

PATTERNS OF INJURY AND SIGNIFICANCE OF UNCOMMON FRACTURES IN THE BATTERED CHILD SYNDROME*

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SINCE its original description,[2,9,12] the battered child syndrome has received considerable attention. Many forms of child abuse are recognized, but the roentgenologic detection of skeletal trauma still plays a prominent, if not the most prominent role.

Because of this, it was decided to review the case histories and roentgenograms in 100 physically abused children, and to document the patterns of injury. This was done in order to determine how frequently various fractures occurred, including the typical epiphyseal-metaphyseal fracture. In addition, a second group of infants with more unusual fractures was also studied; the purpose being to determine what specificity these less common injuries might have in the diagnosis of the battered child syndrome.

MATERIAL AND METHOD

Two groups of patients were studied. The first consisted of 100 consecutive infants seen at The University of Texas Medical Branch from 1967 to 1971 in whom the diagnosis of battered child syndrome was made. Ninety-five of these patients had roentgenograms available for study, and these were examined with respect to: (1) incidence of positive roentgenographic findings; (2) type, location, and particular distribution of skeletal injuries; and (3) incidence and distribution of more unusual, but potentially significant fractures.

The second group of infants consisted of those in Group I who demonstrated less common fractures, plus an additional series of abused children who demonstrated similar skeletal injury. These injuries were then assessed in terms of their potential specificity in diagnosing the battered child syndrome.

RESULTS

Detailed results are tabulated in Table 1. The age of the patients ranged from 6 weeks to 8 years, with the majority being between 1 and 1½ years of age. Forty-nine patients were males and 46 were females. Of the 95 patients in Group 1, 63 (66 per cent) had positive roentgenographic findings, but only 52 (55 per cent) had evidence of skeletal fractures. The remaining 11 per cent showed either isolated soft tissue injury or soft tissue injury with spread sutures. The long bones were involved in 34 (36 per cent) infants, but only 22 (23 per cent) had multiple fractures. Out of the entire group of 95 patients, only 14 (15

TABLE I

GROUP I

Total number of patients	100
Total number of patients with roentgenograms	95
Age	6 wk.–8 yr.
Male/female	49/46
Positive roentgenologic findings	63 (66 per cent)
Skeletal fractures	52 (55 per cent)
Long bone fractures	34 (36 per cent)
Transverse/spiral fractures	30 (31 per cent)
Epiphyseal-metaphyseal fractures	14 (15 per cent)
Calvarial changes (spread sutures, with or without fracture)	40 (42 per cent)
Skull fractures	21 (22 per cent)
Spread cranial sutures (only)	17 (18 per cent)
Soft tissue findings	9 (10 per cent)
Soft tissue (only)	4 (5 per cent)
Multiple fractures	22 (23 per cent)

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per cent) showed the typical epiphyseal-metaphyseal fractures. On the other hand, 30 (31 per cent) of the patients showed spiral or transverse fractures of the long bones. Some overlap was present in these latter 2 groups, but still it was found that in approximately one-half of the patients with long bone fractures, typical epiphyseal-metaphyseal fractures were absent. Other fractures demonstrated in the infants of Group I were: clavicle (4); sternum (1); scapula (3); ribs (8); and spine (1). Soft tissue findings were noted in 9 patients (10 per cent), and one-half of these showed soft tissue changes without evidence of skeletal trauma.

Calvarial changes were present in 40 patients (42 per cent). Twenty-one patients (22 per cent) had skull fractures (with or without associated spreading of the sutures), while 17 patients (18 per cent) showed spread calvarial sutures without fracture. Most of the skull fractures were linear, with the majority being in the parietal and posterior parietal areas. Two patients had severe, crushed, "egg-shell" type fractures, and 1 patient eventually developed a leptomeningeal cyst. Subdural effusions were documented in 10 per cent of the infants, but since all of the patients with positive roentgenographic skull findings were not subjected to subdural taps, this figure is probably low.

Less common fractures, as illustrated in the patients of Group II consisted of spinal, sternal, lateral clavicular, scapular, and rib fractures.

DISCUSSION

GROUP I

In 1946 Caffey[2] described 6 patients with chronic subdural hematomas and fractures of the long bones. Silverman[12] subsequently called further attention to the problem, and in 1962 the term "battered child syndrome" was coined by Kempe *et. al.*[9] Since that time, interest in this condition has continued to grow.[1,3–5,8,10–13,15]

Roentgenographic findings in the battered child syndrome are well documented, but while engaged in our study, it became increasingly apparent that many of our infants did not demonstrate the classic picture—*i.e.*, multiple epiphyseal-metaphyseal fractures in various stages of healing. Indeed, spiral and transverse fractures of the long bones were more common, and it appeared to us that if one relied too heavily on the presence of the classical epiphyseal-metaphyseal fracture, many infants might evade early diagnosis.

Spiral or transverse fractures most commonly involved the femur, and then the tibia. The mechanism of injury was believed to be either by direct trauma, or forceful twisting and wrenching of the extremity. These fractures are, of course, not as specific as the typical epiphyseal-metaphyseal type, but since they were so common in our series it might be of value to view them with more suspicion; especially if they occur in the prime battered child age group. This would seem particularly appropriate when such fractures are seen in conjunction with other roentgenographic evidence of trauma or suspicious clinical findings.

Multiple injuries, especially if seen in various stages of healing, are considered highly suspicious, if not classically diagnostic of the battered child syndrome.[1,8,10,11] However, only 23 per cent of our patients showed this pattern, and it may be that one should be more suspicious of solitary fractures when an incongruous clinical history is present.

Soft tissue changes, as seen in our infants, were nonspecific. They were usually seen in the acute stage of trauma and probably represented edema and/or bleeding into the soft tissues. Their chief value is usually that of directing the roentgenologist's attention to the site of trauma, but they can also aid in the diagnosis of the battered child syndrome when used in conjunction with other positive roentgenographic findings.

Spreading of the calvarial sutures with or without evidence of skull fracture de-

serves further comment. Indeed, suture spread may be the first clue to the presence of the battered child syndrome. In some cases this probably represents transient cerebral edema (contusion), but more often it is probable that subdural hematomas are present. Subdural hematoma is a common problem in the battered child syndrome, and possible brain damage due to repeated cranial trauma should receive prime consideration. Long bone fractures may heal without a trace of deformity, but brain damage is often irreversible. The presence of spread sutures, with or without evidence of a calvarial fracture, was especially helpful in those cases where spiral or transverse long bone fractures were the only fractures present.

Over-all, many of the findings in our study were somewhat surprising. Notable were the facts that only 66 per cent of the patients showed positive roentgenologic findings and that the classic epiphyseal-metaphyseal fractures were present in only 15 per cent of the patients. However, surprising as it was, it would be somewhat presumptuous to suggest that this experience is universal, but, at the same time, it would not be out of order to suggest that there may be more cases than currently suspected which are not of the "classic" type. Consequently, it might be that one's index of suspicion should be raised, even with an ordinary long bone fracture, if it is incompletely accounted for.

GROUP II

Fractures encountered in this group of infants were certainly less common, and often somewhat unusual. Nonetheless, it was determined that many were highly suspicious, if not strongly suggestive, of the battered child syndrome. In the following paragraphs each fracture is discussed in detail, and its particular value in the diagnosis of battered child syndrome is cited.

Clavicular fractures. The most common fracture of the clavicle, from any cause, occurs in the midshaft. It is, of course, not specific and should be considered significant only when associated with other features of the battered child syndrome. Fractures of the lateral end of the clavicle, however, are a different matter. They are much less common than midshaft fractures and not usually encountered except as part of perinatal trauma,[6] or the battered child syndrome. They show a specific roentgenologic configuration, and are most easily recognized in the healing phase. They might be compared to the typical epiphyseal-metaphyseal long bone fractures, for they probably result from the same type of violent shaking or twisting injury. Characteristically, the lateral end of the clavicle has a splayed or club-like appearance, and it

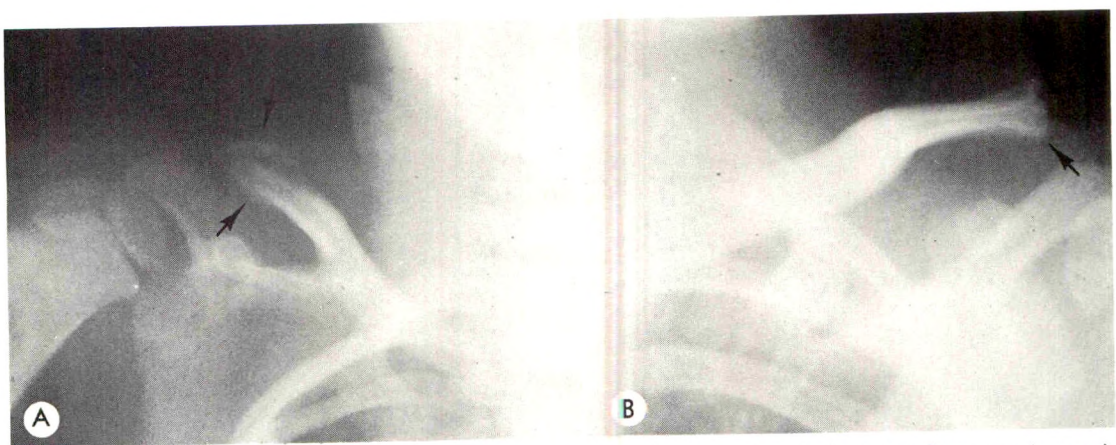


Fig. 1. (*A* and *B*) *Clavicular fractures.* Two examples of healing lateral end clavicular fractures (arrows). Note similarity to typical epiphyseal-metaphyseal long bone fractures.

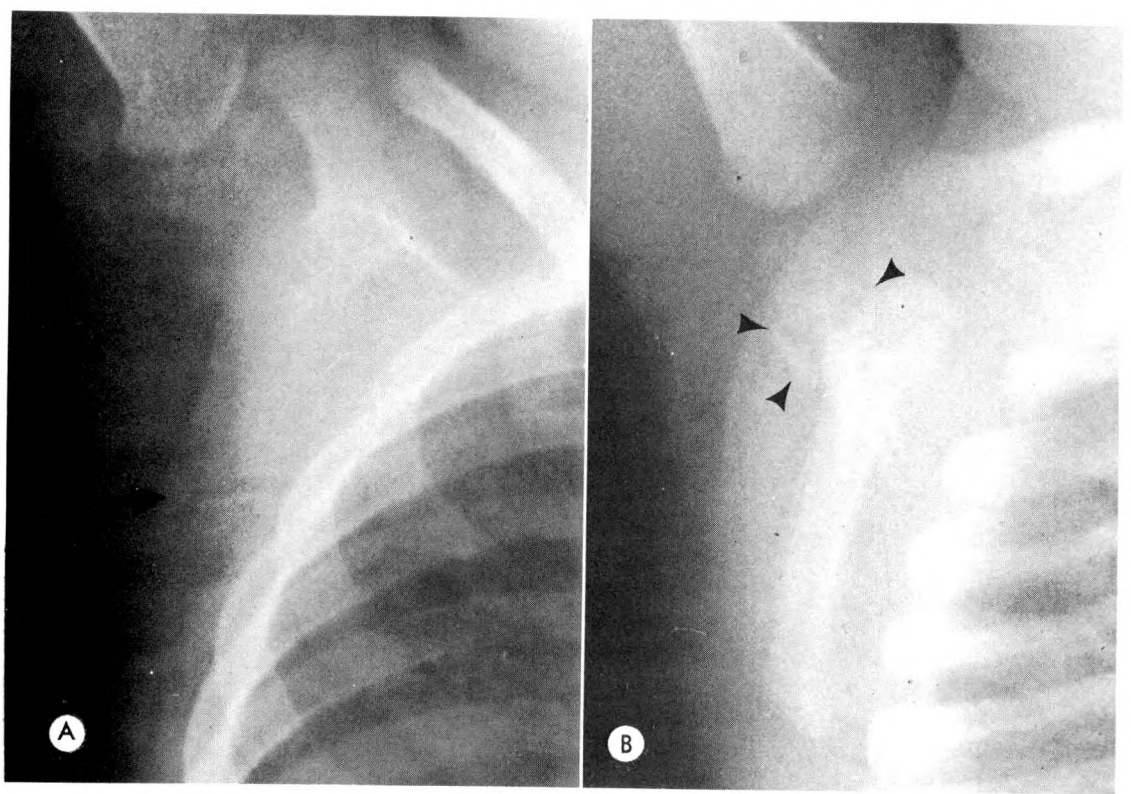
FIG. 2. *Sternal fracture.* Fracture of the sternum with fracture fragment displacement (arrows).



is suggested that this fracture be considered highly suspicious in the diagnosis of the battered child syndrome (Fig. 1, *A* and *B*).

Sternal fractures. Fractures of the sternum are unusual at any time, and most often result from severe direct blows to the chest. If such a fracture is present and there is no substantiating history to account for a severe blow (*i.e.*, automobile accident, crush injury to the chest), one should strongly consider the possibility of child abuse. In 1 of our cases, a sternal fracture was the only roentgenographic sign present (Fig. 2).

Scapular fractures. Fractures of the scapula are also unusual and 2 types occur. The first is the transverse fracture of the scapula, probably sustained by direct trauma (Fig. 3*A*), and the second is that



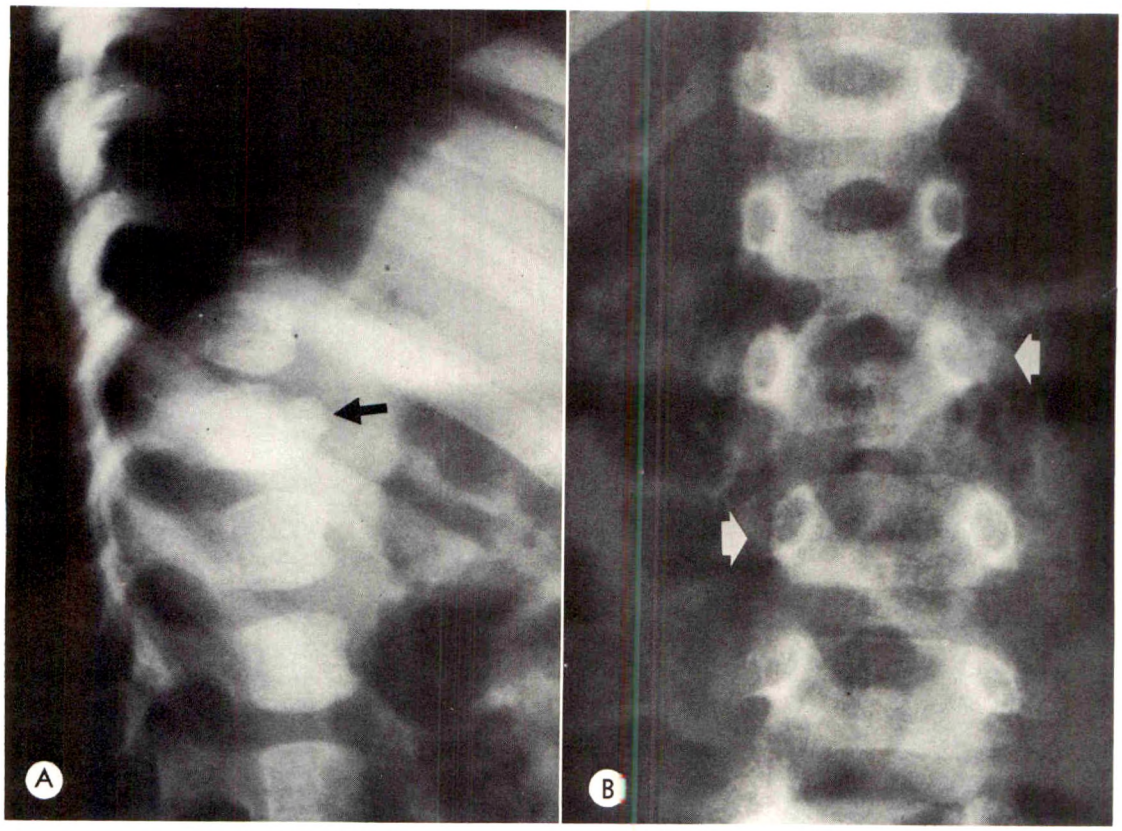


Fig. 4. *Spine trauma.* (*A*) Note notch-like deformity of vertebral body (arrow) and narrowing of the intervertebral disk space above it. This probably resulted from hyperflexion of the spine and associated anterior nuclear herniation. In other cases no intervertebral disk space narrowing is seen, but rather, anterior notching or simple compression is noted. (*B*) Fracture-dislocation of L2 and L3 (arrows), probably resulting from violent shaking of the child. (Reproduced with permission of *Radiology*.[13])

of fragmentation of the acromial process (Fig. 3*B*). The latter fracture probably results from severe twisting or shaking, much as does the typical epiphyseal-metaphyseal long bone fracture, or the fragmentation fracture of the lateral end of the clavicle. Consequently, this scapular fracture, and indeed, any scapular fracture should be viewed with considerable suspicion, especially when there is no history to substantiate the type of trauma required to produce such an injury.

Spine and spinal cord injury. Trauma to the spine and spinal cord in the battered child syndrome has been reported[13] but is not particularly common. Generally, spinal trauma can take 1 of 3 configurations: (1) anterior notching of the vertebral body (produced by hyperflexion injury with or without anterior herniation of the nucleus pulposus (Fig. 4*A*); (2) compression of the vertebral body; and (3) fracture-dislocation (Fig. 4*B*). Neither anterior notching nor simple vertebral body compression is specific for the battered child syndrome, nor are these fractures of any dire consequence; they are often subclinical in nature and notching can be seen in otherwise

Fig. 3. *Scapular fractures.* (*A*) Transverse scapular fracture (arrow). (*B*) Fragmented acromial process fracture of the scapula (arrows). Early callus formation is present.

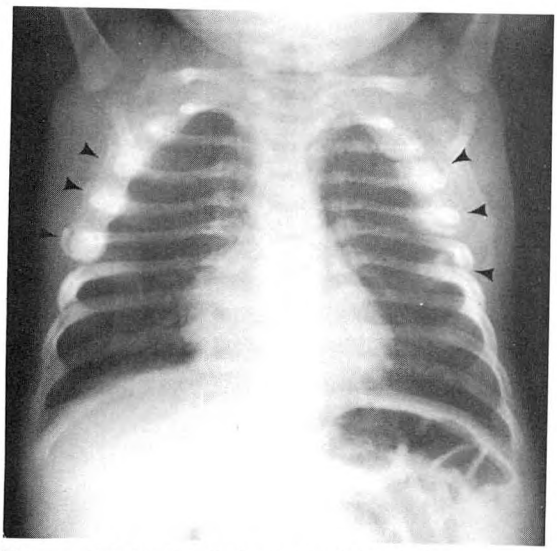


FIG. 5. *Multiple rib fractures.* This infant showed rib fractures (arrows) and spreading of the calvarial sutures only. No long bone fractures were noted.

normal infants.[13] Fracture-dislocation of the spine, on the other hand, is more significant, for it is unlikely that such injury could be sustained other than from severe, purposeful direct blows, violent shaking, or whiplashing. Spinal cord injury is, of course, much more likely to occur with such trauma.

Rib fractures. Rib fractures in children are rather uncommon, especially in the battered child age range. The normal resilience of the young thorax, plus the need for a direct blow to have occurred, probably accounts for this fact.[8,15] On the other hand, healing rib fractures can be seen shortly after birth, and most often result from perinatal injury.[14] After this time, if fractures are encountered, and a legitimate history of trauma is absent, the presence of these fractures should raise the possibility of intentional abuse. In the battered child syndrome, rib fractures are usually multiple (Fig. 5). These are most commonly seen in the posterior or lateral regions of the ribs, but anterior costochondral separations can also occur. The latter lesions are best visualized in the healing phase, whereupon the end of the rib becomes widened and clubbed (Fig. 6).

CONCLUSION AND SUMMARY

The classic roentgenographic and clinical features of the battered child syndrome were not present in most of our patients. The well known epiphyseal-metaphyseal long bone fractures, usually considered the classic findings in these infants, were less common in our series than were spiral and transverse fractures of the long bones. Multiple skeletal injuries were also less common than expected. It is not known whether these data merely represent an isolated experience, or a more universal, but as yet unappreciated phenomenon. However, because of our findings it has caused us to place more emphasis on, and be more suspicious of single, ordinary long bone fractures, especially when clinical correlation is less than plausible.

Skull fractures and spread of the cranial sutures, either alone or in combination, were quite common. These findings are obviously significant both in respect to early diagnosis and associated intracranial complications. Often skull fractures, or simple spreading of the sutures, served to focus more attention on otherwise innocuous appearing, but concurrent, spiral or transverse long bone fractures.

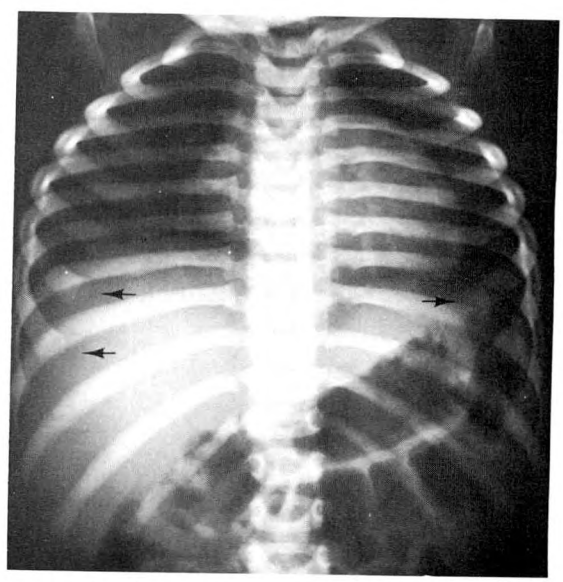


FIG. 6. *Anterior costochondral separation.* Note widened, clubbed anterior rib ends (arrows).

Certain less common fractures found in our series were felt to be highly suspicious, and might even be considered as valuable a sign of the battered child syndrome as the typical epiphyseal-metaphyseal long bone fracture. The most notable of these were fractures of the lateral end of the clavicle and fractures of the ribs and scapula. Less often, sternal and spinal fractures were useful.

It is believed that although these fractures are less common, their appreciation should aid the radiologist in diagnosing cases which might otherwise pass unrecognized for considerable periods of time.

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REFERENCES

1. Baker, D. H., and Berdon, W. E. Special trauma problems in children. *Radiol. Clin. North America*, 1966, *4*, 289–293.
2. Caffey, J. Multiple fractures in long bones of children suffering from chronic subdural hematoma. Am. J. Roentgenol. & Rad. Therapy, 1946, *56*, 163–173.
3. Caffey, J. Parent-infant traumatic stress syndrome: (Caffey-Kempe syndrome),(battered babe syndrome): First Annual Neuhauser Presidential Address of Society for Pediatric Radiology. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *114*, 217–229.
4. Fontana, V. J., Donovan, D., and Wong, R. J. "Maltreatment syndrome" in children. *New England J. Med.*, 1963, *269*, 1389–1394.
5. Fontana, V. J. Recognition of maltreatment and prevention of battered child syndrome. *Pediatrics*, 1966, *38*, 1078.
6. Förster, A. Neonatal metaphyseal injuries: typical changes at unusual site. *Ann. de radiol.*, 1971, *14*, 314–320.
7. Griffiths, D. L., and Moynihan, F. J. Multiple epiphyseal injuries in babies ("battered baby" syndrome). *Brit. M. J.*, 1963, *2*, 1558–1561.
8. Gwinn, J. L., and Barnes, G. R. Radiological case of month. *A.M.A. Am. J. Dis. Child.*, 1965, *109*, 457–458.
9. Kempe, C. H., Silverman, F. N., Steel, B. F., Droegenmueller, W., and Silver, H. K. Battered child syndrome. *J.A.M.A.*, 1962, *181*, 17–24.
10. Krige, H. N. Abused child complex and its characteristic x-ray findings. *South African M. J.*, 1966, *40*, 490–493.
11. McHenry, T., Girdany, B. R., and Elmer, E. Unsuspected trauma with multiple skeletal injuries during infancy and childhood. *Pediatrics*, 1963, *31*, 903–908.
12. Silverman, F. N. Roentgen manifestation of unrecognized skeletal trauma in infants. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1953, *69*, 413–427.
13. Swischuk, L. E. Spine and spinal cord trauma in battered child syndrome. *Radiology*, 1969, *92*, 733–738.
14. Swischuk, L. E. Radiology of the Newborn and Young Infant. Williams & Wilkins Company, Baltimore, p. 414.
15. Teng, C. T., Singleton, E. B., and Daeschner, C. W. Skeletal injuries of battered child. *Am. J. Orthoped. Surg.*, 1964, *6*, 202–207.

PHARMACOLOGIC AIDS IN ANGIOGRAPHY OF THE UPPER EXTREMITY*

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THE angiographic study of ischemia of the upper extremity has not consistently yielded definitive diagnostic roentgenograms. Much of the difficulty encountered is caused by a lack of uniformity in the technique of the angiographic procedure chosen.[3,5] Confusion exists in the classification of the multiple vascular disease entities that can affect these extremities. There is a paucity of information describing the use of Priscoline (tolazoline hydrochloride) or stellate ganglionic blocking during an upper extremity vascular study.[1,3,6]

MATERIAL AND METHOD

Six patients were studied by the transfemoral Seldinger technique. A preshaped No. 5 French polyethylene catheter was selectively placed into the subclavian artery of the affected extremity distal to the origin of the vertebral artery. Twenty to thirty milliliters of Renografin 76 per cent (depending upon the weight of the patient) was injected intra-arterially, using an automatic injector at a rate of about 10 ml. per second. The film sequence extended over a period of 30 seconds, exposing 1 film every 2 seconds.

After a 5 minute delay, a pharmacologic intervention was performed followed by a second identical contrast medium injection into the distal subclavian artery.

A stellate ganglionic block administered by an anesthesiologist was our intervention method of choice in 3 cases. The repeat contrast medium study was carried out within 30 minutes after the initiation of the block, depending somewhat upon the time it took for subjective relief to be reported by the patient.

Insertion of 50 mg. of Priscoline into the catheter followed by a 10 ml. saline flush was performed in 2 other patients. Following a 30 second pause, 20 to 30 ml. of contrast material was reinjected intra-arterially.

DISCUSSION

Vasospastic and vaso-occlusive diseases involving the upper extremity have been extensively discussed in the surgical literature. Most of the articles are therapy related with a strong emphasis placed upon clinical response and evaluation.

Various attempts at classification of such disorders are found,[4] with an adequate organization of the diseases reported by Dale and Lewis.[3] These physicians include trauma, arterial embolism related to cardiac arrhythmia, the thoracic outlet syndrome, arterial thrombosis due to arteriosclerosis, thromboangiitis obliterans, polycythemia, frost bite and collagen disease as major causes of vaso-occlusion.

Vasospastic ischemia, on the other hand, is a more difficult diagnostic problem and often a definite etiology is not established for an individual case. Arteritis and primary Raynaud's disease have been incriminated as provoking vascular narrowing in the upper extremities. It is not the purpose of this paper to differentiate Raynaud's disease from Raynaud's phenomenon.[3,4,7]

Marshall and co-workers,[6] Laws *et al.*[5] and Calenoff[1] stressed the use of brachial arteriography in the study of hand ischemia. The latter author[1] described finite vascular alterations of the hand caused by anatomic variations. Limitations of the brachial approach included iatrogenic vasospasm, in-

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consistent subclavian arterial detail and inhibited motion of the arm for roentgenographic positioning. We have therefore utilized the Seldinger femoral approach as the method of choice in studying upper extremity vascular disease. The flexibility and ease of approaching both upper extremities with a single catheter facilitate the speed of completion of the procedure.

The angiographer must decide whether an area of arterial narrowing is an actual occlusion or possibly represents spasm. A definite occlusion such as is caused by a vascular thrombosis will not require the use of a pharmacologic intervention. If, however, the narrowing is nonspecific, then the use of a vasodilating agent may increase the contrast medium flow through the vessel with better visualization.

Stellate ganglionic blockage utilizes an anesthetic agent such as Xylocaine to inhibit the sympathetic nerve supply to an affected upper extremity. If such a block is effective, vasoconstriction is eliminated. The temporary sympathectomy produced does more than release efferent vasomotor impulses. The peripheral vascular wall has, in addition, an altered response to blood gases, principally carbon dioxide. The anoxic response related to this gas produces vasodilatation and a shift of the pH to the acid side.[2]

Priscoline, an alpha blocking agent, acts by competitive blocking of vasoconstrictor receptors in the arterial wall, and vasodilatation results.

Case I illustrates the difficulty in distinguishing whether the distal ulnar tapering represented vaso-occlusion or vasospasm (Fig. 1). No filling of the digital vessels was seen. The lack of specificity in this case led us to use vasodilating agents in similar patients.

Cases II, III, and IV illustrate the effective utilization of either stellate ganglionic blockage or Priscoline in distinguishing between occluded and spastic branches (Fig. 2, *A–C*; 3, *A–D*; and 4, *A–C*). The ease and speed of introduction of an alpha blocking agent into the arterial system of an upper

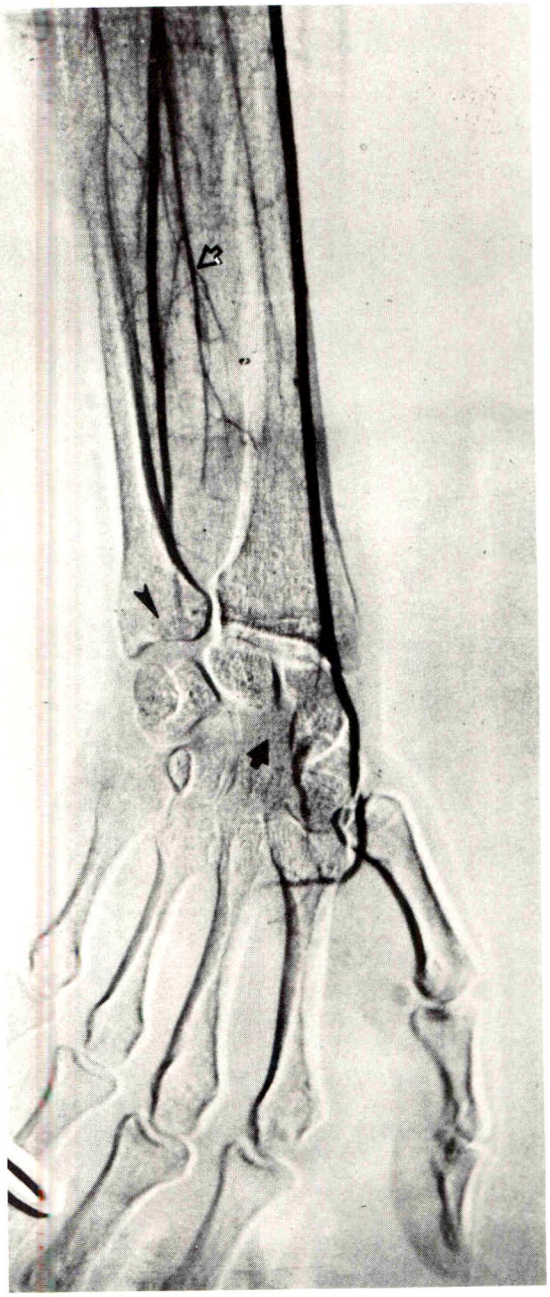


FIG. 1. Case I. Left subclavian arteriogram in a 33 year old female with coldness and bluish discoloration of her left hand.

A subtraction film taken 13 seconds following the injection of 30 ml. of Renografin 76 per cent into the left subclavian artery is shown. The radial artery is normal, but there is no filling of the palmar arch (large black arrow). Although the interosseous arteries (white arrow) and the proximal ulnar artery are normal, the distal ulnar vessel tapers markedly (arrowhead). There is no filling of any of the digital branches.

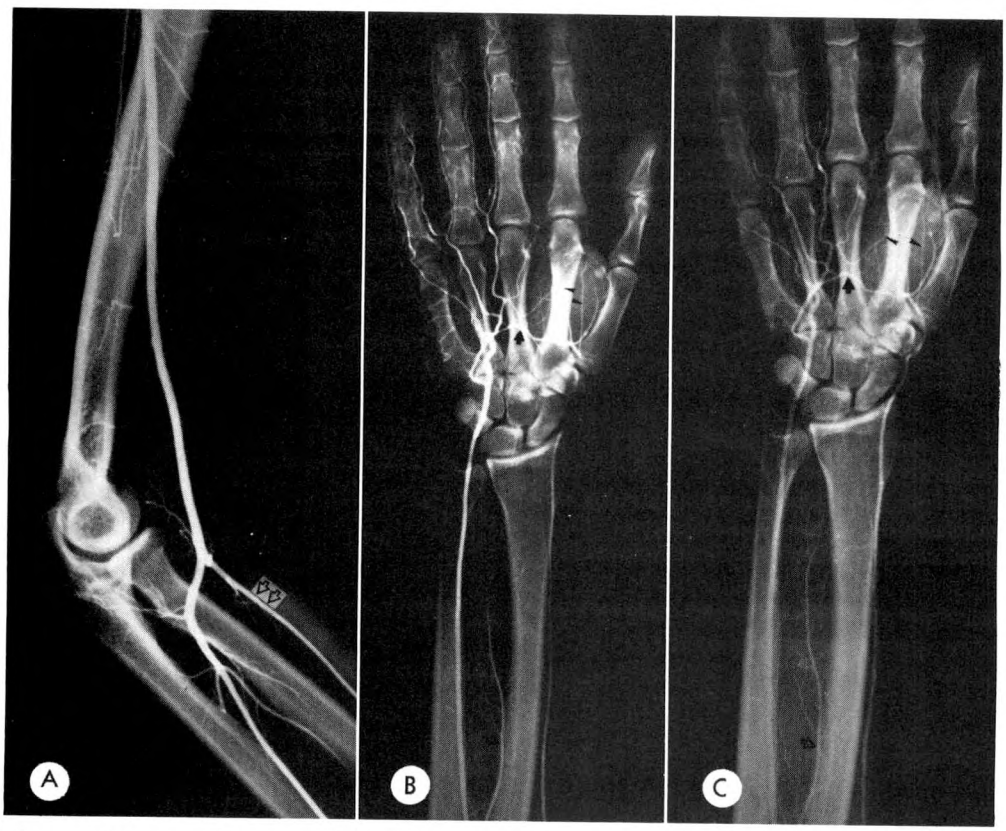


FIG. 2. Case 11. A 24 year old female with temperature loss and numbness of the right first and second digits. Right subclavian arteriography was performed.

(*A*) Normal definition of the brachial artery. The proximal part of the ulnar artery is uniformly narrowed (2 white arrows). (*B*) The right forearm and hand before the addition of intra-arterial Priscoline. The ulnar artery continues its usual course and contributes almost all of the blood supply to the palmar arch (large black arrow). The interosseous arteries (white arrow) are poorly filled. The distal portion of the radial artery tapers abnormally and gives no vasculature to the deep palmar arch. The fourth and fifth digital arteries are well perfused, but the first, second and radial side of the third digital arteries have almost no blood supply (2 small arrowheads). (*C*) Angiogram obtained following the intra-arterial insertion of 50 mg. of Priscoline. The ulnar distribution remains normal. The interosseous arterial branches (white arrow) are better filled than was noted in *B*. The radial artery is better perfused and the anastomosis with the palmar arch (large black arrow) is patent. Although the first and second digital arteries (2 small arrowheads) show little increase in caliber, there is better filling of the thenar vasculature at this time.

extremity could make this method preferable to a stellate block. However, the latter technique, although time consuming, should produce a more uniform vasodilatation.

Therapy of vasospastic upper extremity disease includes sympathectomy and the oral use of alpha blocking agents.[4,7] It would be most helpful to the clinician if he could predict which patients would benefit from such treatment. It is possible that by using our method of studying upper extremity arterial pathology, the proper therapeutic choice will be made. Even more important may be the predictability of fine arterial vascular surgery, if such becomes feasible in the near future.

SUMMARY

A method of studying upper extremity vascular disease is presented.

If vasospasm was suspected, stellate ganglionic blockage or the intra-arterial administration of Priscoline was employed to increase the arterial flow.

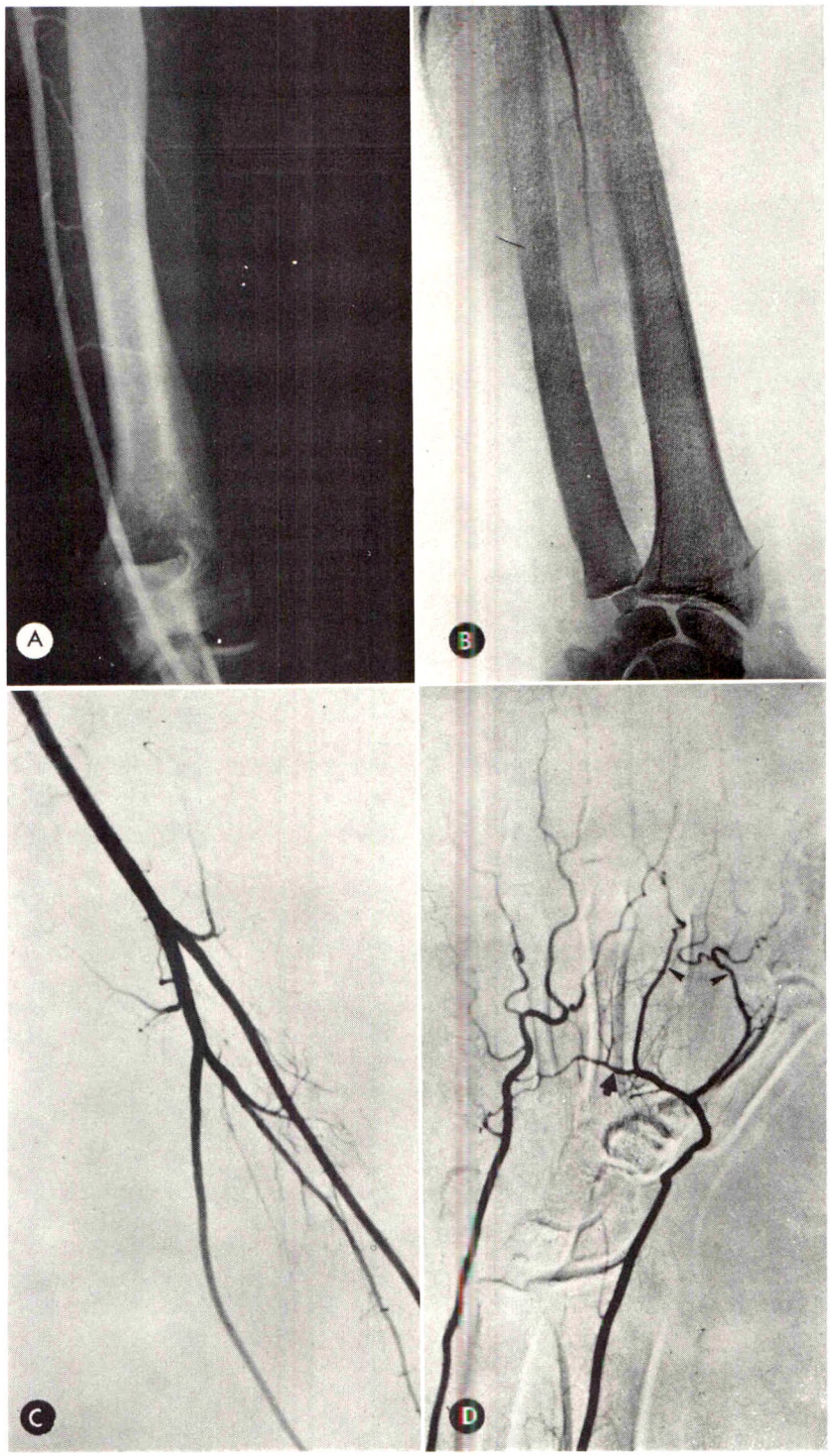


Fig. 3. Case III. A 40 year old male with substernal pain and numbness radiating down his left arm.

(*A*) Left subclavian arteriogram showing a normal flow of contrast medium down the brachial artery. (*B*) Subtraction film showing marked tapering of the proximal radial and ulnar arteries. No contrast material was seen distal to this narrowing. The patient felt no warmth in his left hand during this injection series. (*C* and *D*) Post stellate block series: there is a normal flow pattern of contrast medium through the ulnar, radial and interosseous arteries. The palmar arch (large black arrow) and the digital arteries (2 small arrowheads) are well filled.

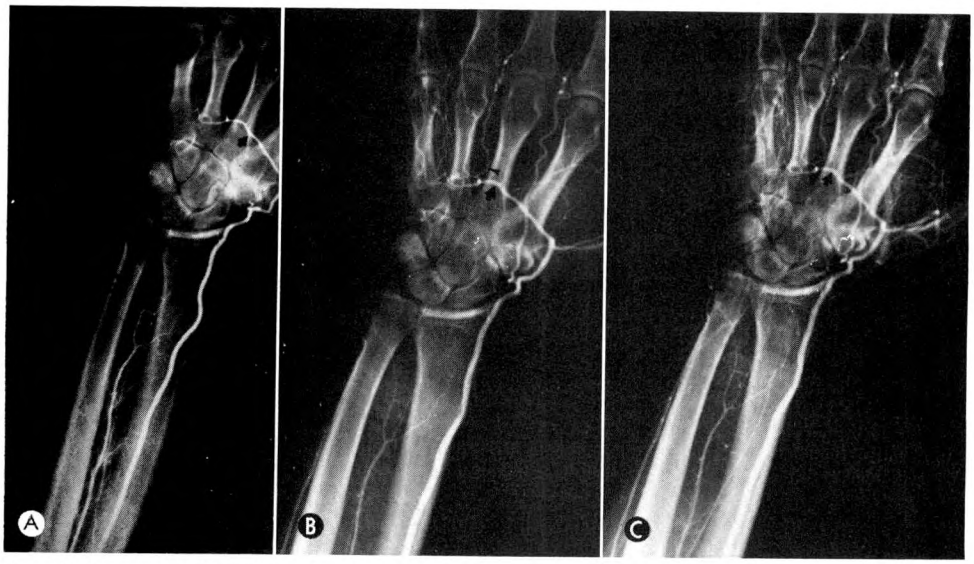


FIG. 4. Case IV. A 59 year old male with pain and numbness in his right upper extremity.

(*A*) Right subclavian arteriogram before the stellate block. There is some tapering of the distal aspect of the radial artery, although it supplies blood to the palmar arch (large black arrow). The distal portion of the ulnar artery narrows abnormally and gives no definite blood to the palmar arch. The interosseous artery is normal (white arrow). No contrast medium is noted within the digital arteries. (*B* and *C*) Right subclavian arteriograms following the stellate block. There is better perfusion of blood (and contrast material) through the radial artery than was noted on the pre-block angiogram. The ulnar artery is also better filled distally. The palmar arch (large black arrow) is well seen now, with its blood supply coming mostly from the radial vessel. The digital arteries (2 small arrowheads) have a definitely improved blood flow.

Appropriate therapy should benefit from such diagnostic knowledge.

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REFERENCES

1. CALENOFF, L. Angiography of hand: guidelines for interpretation. *Radiology*, 1972, *102*, 331–335.
2. CONN, J., BERGAN, J. J., and BELL, J. L. Hypothenar hammer syndrome: posttraumatic digital ischemia. *Surgery*, 1970, *68*, 1122–1128.
3. DALE, W. A., and LEWIS, M. R. Management of ischemia of hand and fingers. *Surgery*, 1970, *67*, 62–79.
4. HARDY, J. D., CONN, J. H., and FAIN, W. R. Nonatherosclerotic occlusive lesions of small arteries. *Surgery*, 1965, *57*, 1–13.
5. LAWS, J. W., LILLIE, J. G., and SCOTT, J. T. Arteriographic appearances in rheumatoid arthritis and other disorders. *Brit. J. Radiol.*, 1963, *36*, 477–493.
6. MARSHALL, T. R., NEUSTADT, D., CHUMLEY, W. F., and KASDAN, M. L. Hand arteriography. *Radiology*, 1966, *86*, 299–304.
7. PAABY, H., and STADIL, F. Thrombosis of ulnar artery. *Acta orthop. scandinav.*, 1968, *39*, 336–345.

GANGLIOSIDOSIS (GM_1)*

A RE-EVALUATION OF THE VERTEBRAL DEFORMITY

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WE have recently encountered a case of generalized gangliosidosis (GM_1) in which the vertebrae at L1 and L2 demonstrated the classical beaking seen in this disease and in Hurler's disease. The infant succumbed at the age of 2 years and pathologic examination of the affected vertebral bodies enabled us to gain additional insight into the pathogenesis of this deformity.

Demonstration of an accumulation of hyaline cartilage in the affected area, suggested to us that the deformity is caused by an abnormal growth process and not by herniation of a corresponding intervertebral disk.[5]

REPORT OF A CASE

A 2 year old Puerto Rican male was admitted to Kings County Hospital for the 4th and last time because of asthma and seizure disorders that began at the age of 6 months associated with severe psychomotor retardation and an enlarging liver. An evaluation for mental retardation at the age of 13 months included a rectal biopsy and discovery of aggregates of large histiocytes with foamy cytoplasm that was strongly positive for mucopolysaccharides on PAS stain. A liver biopsy was performed 2 months later and showed many enlarged Kupffer's cells with pale and faintly basophilic, vacuolated cytoplasm. Histochemical studies showed a strong accumulation of glycolipids. An analysis of the polymorphonuclear leukocytes manifested a beta galactosidase deficiency.

On this hospital admission the physical examination revealed an obese, well developed, infant undergoing repeated seizures and asthmatic attacks. The patient had difficulty holding his head upward and his facial features did not resemble the classical Hurler's syndrome.

Hepatosplenomegaly was present and the neurologic examination revealed hypertonic and spastic extremities. The reflexes were increased bilaterally. His pupils reacted slowly to light and retinoscopy revealed optic atrophy and constriction of the fundal vessels. No cherry red spot was present. The patient succumbed on the 40th day because of repeated aspiration pneumonias. Multiple repeated urinalysis failed to reveal the presence of any mucopolysaccharides.

Histochemical studies of the brain sections performed by Dr. L. Schneck at the Kingsbrook Medical Center, Brooklyn, N. Y. revealed a definite accumulation of GM_1 gangliosides. Atheromatous plaques of the ascending aorta were also noted. (Further pathologic studies of this patient are being accumulated by the Department of Pathology for another communication.)

DISCUSSION

Generalized gangliosidosis is a relatively new storage disease that has been described under a variety of names, *e.g.*, familial neurovisceral lipidosis, pseudo-Hurler's disease, Hurler variant, Tay-Sachs disease with visceral involvement, etc. The patient presently reported manifested many of the features encountered in this disease, *i.e.*, psychomotor retardation, hepatosplenomegaly beginning at 6 months of age, rapid deterioration and death due to bronchopneumonia approximately at the age of 2 years.

The quantity of mucopolysaccharides present in the urine in GM_1 gangliosidosis is usually normal. Pathologically, visceral histiocytosis is present and contains a mucopolysaccharide that is structurally similar to keratin sulfate. Of distinct importance is a 10 times normal accumulation of ganglioside GM_1 (monosialoganglioside) in the brain. This latter feature to an extent

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FIG. 1. Examination of an upper extremity reveals over-all widening of the bone marrow with marked thinning of the cortices.

resembles Tay-Sach's disease except for the accumulation of mucopolysaccharides in the viscera. Patients with gangliosidosis in general have a deficiency of β galactosidase—an enzyme required for the normal catalysis of ganglioside (GM) and mucopolysaccharide.[4]

The disease clinically is easily confused with Hurler's syndrome. However, differentiation is established by the early onset and severity of gangliosidosis, the absence of mucopolysaccharides in the urine and some of the pathologic changes encountered, particularly along the nerve sheaths and within the glomeruli. The latter demonstrate a characteristic ballooning of the glomerular epithelium.

The most characteristic radiologic findings occur in the skeleton, (Fig. 1–3), particularly in the upper extremities, pelvis and spine. Most of these changes have been described previously.[3] The changes occurring in the vertebral body have, however, incited some interest. The anterior notch deformity is located predominantly at the thoracolumbar junction. It is apparently not specific for this disease and has been found in many other conditions, *e.g.*, Hurler's disease, cretinism, Morquio's disease, achondroplasia, trauma, etc. Because of the diverse distribution, Swischuk[5] contended that the deformity was caused by an anteriorly herniated intervertebral disk that resulted following the prolonged kyphosis commonly found at this level. Unfortunately, his concept was based on pure deduction with little pathologic correlation. In fact, the only case of Hurler's disease in which anterior herniation of the intervertebral disk could theoretically be documented,[1] appeared inconclusive when reviewed by us.

Microscopic examination of the vertebral body in our patient demonstrated an excessive accumulation of hyaline cartilage in the region of the deformity (Fig. 4). Columnar and palisading cartilaginous cells could be detected along the posterior superior portion of the body. The patho-

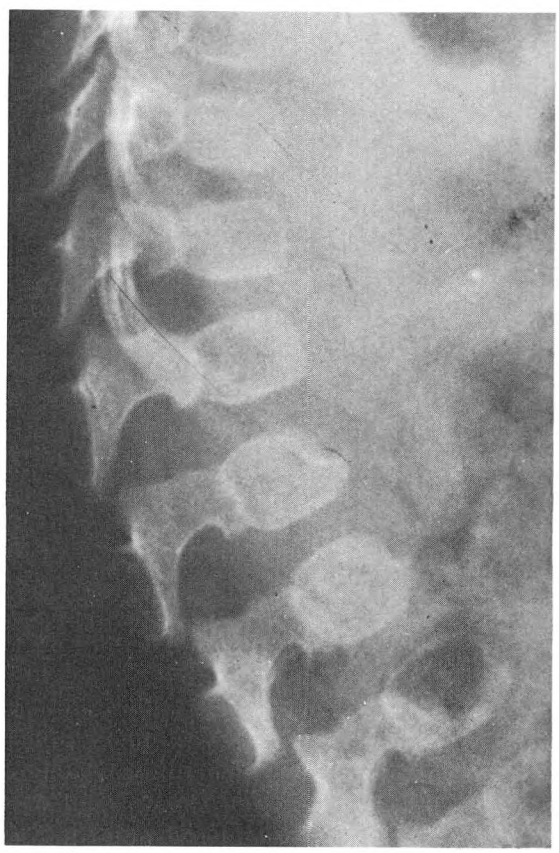


FIG. 2. Roentgenogram of the lumbar spine reveals an acute kyphosis directed at the T12–L1 area. Anterior notch defect is noted to involve the bodies of L1 and L2.

logic findings, therefore, most likely represent an asymmetric growth process. The possibility of a localized dysplasia is still highly unlikely, since the deformity is relatively specific in location and found in many other unrelated diseases.

Gooding and Neuhauser[2] demonstrated the effects of gravitational pressure and stress upon the growth and development of the vertebral body. Growth in the vertical direction occurs along the cartilaginous layers that surround the superior and inferior surfaces of the vertebral body. Decreased or absent pressure on these surfaces results in longitudinal overgrowth of the body and, by contrast, excessive stress or pressure upon the vertebral body could result in an actual decrease in the vertical growth. The frequent occurrence of this deformity at the thoracolumbar junction is no doubt related to the exaggerated and prolonged kyphosis that occurs in the T12 and L1 region. Consequently, the greatest amount of stress transmitted through the vertebral column is directed to the anterior margins of the body of L1 and to a lesser degree L2. This in turn interferes with the normal conversion of cartilage into bone along these margins and accounts for the impaired growth anteriorly and the apparent asymmetry of the vertebral body. Both sides of the vertebral body are affected, although the superior margin does predominate. Asymmetric growth of a vertebral body following uneven stress has been noted in patients with scoliosis.[2] The deformed body is in reality not hypoplastic. The vertical diameter of the posterior part of the vertebral body probably approaches normal limits.

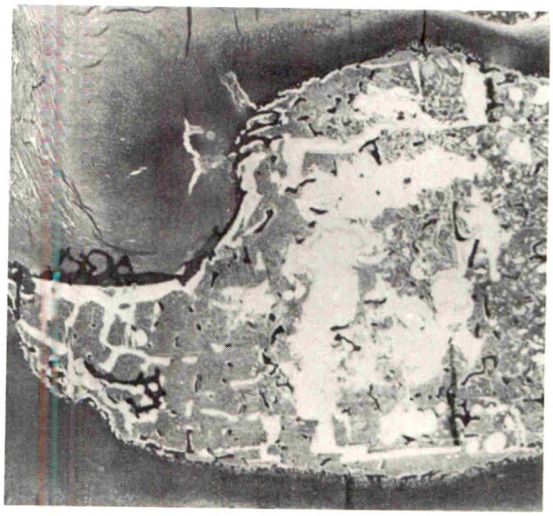


Fig. 4. Pathologic examination of the lumbar vertebrae reveals an excessive accumulation of hyaline cartilage in the notched defect. Somewhat anterior to the superior collection of hyaline cartilage are vertical strands of connective tissue which probably represent portions of a ligament and not that of a disk. (These pathologic findings were confirmed by 3 competent pathologists.)

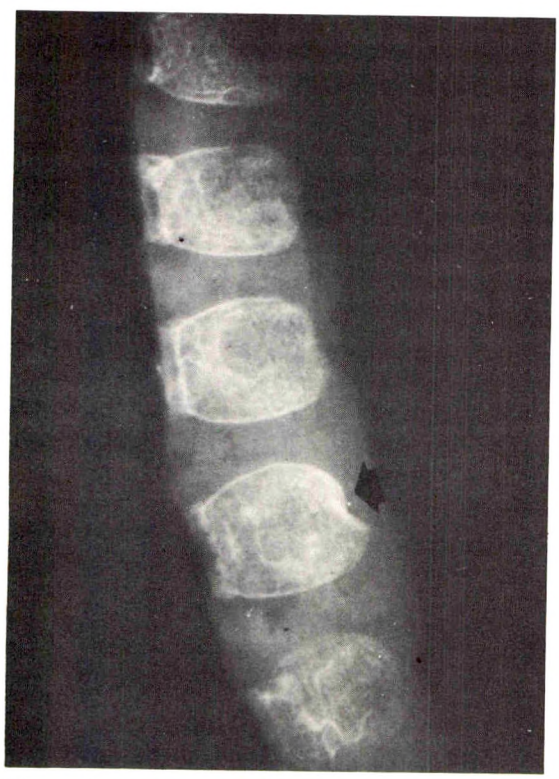


Fig. 3. Roentgenogram of the lumbar spine specimen demonstrates the findings described in Figure 2 (arrow).

SUMMARY

Vertebral bodies in a patient with gangliosidosis (GM_1) demonstrating the characteristic notch deformity were examined pathologically.

Only hyaline cartilage accumulation was

noted in the area of the deformity. This finding suggested that the abnormality is due to asymmetric growth, caused by excessive stress directed anteriorly upon the vertebral bodies of L1 and L2.

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REFERENCES

1. BEGG, A. C. Nuclear herniations of intervertebral disc: their radiologic manifestations and significance. *J. Bone & Joint Surg.*, 1954, *36-B*, 180–193.
2. GOODING, C. A., and NEUHAUSER, E. B. D. Growth and development of vertebral body in presence or absence of normal stress. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *93*, 388–394.
3. GROSSMAN, H., and DANES, B. S. Neurovisceral storage disease: roentgenographic features and mode of inheritance. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *103*, 149–153.
4. O'BRIEN, J. Generalized gangliosidosis. *J. Pediat.*, 1969, *75*, 167–187.
5. SWISCHUK, L. E. Beaked, notched or hooked vertebrae. *Radiology*, 1970, *95*, 661–664.

SIZE OF PELVIC BONY OUTLET IN RENAL AGENESIS*

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A SMALL bony pelvic outlet was observed by Currarino[2] and later Rabinowitz *et al.*[6] in 4 neonates with severe respiratory distress (Fig. 1). All of the patients had clinically obvious multiple anomalies including imperforate anus. All died a pulmonary death. Autopsies revealed renal dysplastic malformations or actual agenesis incompatible with life; pulmonary hypoplasia was marked and complicated by interstitial pulmonary emphysema, pneumomediastinum and pneumothorax. Both authors suggested that the small pelvic bony outlet reflected the small size of the bladder, the latter reflecting the absence of urine formation *in utero*.

In this report, 3 patients will be presented. Two had renal agenesis and showed immediate respiratory distress with pneumothorax and pneumomediastinum; the bony pelvic outlet was *normal* despite autopsy evidence of renal agenesis and bladder hypoplasia. In the third case, an infant with *segmental sacral agenesis* was treated for respiratory distress syndrome at birth. The patient survived and showed evidence of a small pelvic bony outlet and small bladder, although urographic studies showed normally functioning kidneys.

In an attempt to clarify the relative contribution of bladder size, renal function and sacral malformations to the size of the pelvic bony outlet, these cases and the previous cases of Currarino[2] and Rabinowitz *et al.*[6] were reviewed, as well as recent cases of renal agenesis published by Stern *et al.*[8] (Tables I and II).

REPORT OF CASES

CASE I. *Renal Agenesis with Normal Pelvic Bony Outlet and Sacrum.*

This 2,010 gram male infant was born after a 34 week gestation period; oligohydramnios was noted at the time of delivery and the patient developed almost immediate respiratory distress. The infant lived 3 hours. Physical examination during life revealed ear deformities and "Potter's facies" with flattened nose, recessed chin, webbed neck, large spade-like hands and club feet. Roentgenographic examination showed bilateral pneumothoraces and pneumomediastinum (Fig. 2*A*); the lumbosacral spine and pelvis were normal (Fig. 2*B*).

Autopsy showed the lungs to be hypoplastic. Pulmonary interstitial emphysema, as well as pneumothorax and pneumomediastinum was noted. The kidneys were absent and the bladder was hypoplastic.

Comment. The odd appearance of the patient and the oligohydramnios alerted all concerned to the possibility of renal agenesis. The pulmonary symptoms were in keeping with that diagnosis, as such patients commonly have hypoplastic lungs if the renal anomaly is associated with oligohydramnios. The facial appearance and club feet are all "oligohydramnios" deformities and not specifically related to renal agenesis.[1,7]

CASE II. *Renal Agenesis with Normal Pelvic Bony Outlet and Sacrum.*

This 1,950 gram male infant was born after a 37 week gestation. As in Case I, amniotic fluid was absent and placental examination showed vernix granulomas which represent adhesion of fetal skin to the amniotic membranes and are a part of the "oligohydramnios syndrome."[1] The infant had almost immediate respiratory distress at birth and lived 57 hours. He was noted to have "Potter's facies" and other oligohydramnios deformities, including club feet and genu recurvatum. Roentgenographic examination of the chest (Fig. 3*A*) showed bilateral pneumothoraces in the presence of a normal lumbosacral spine and pelvis (Fig. 3*B*). An intravenous pyelogram failed to show any renal structures and no urine was passed during life.

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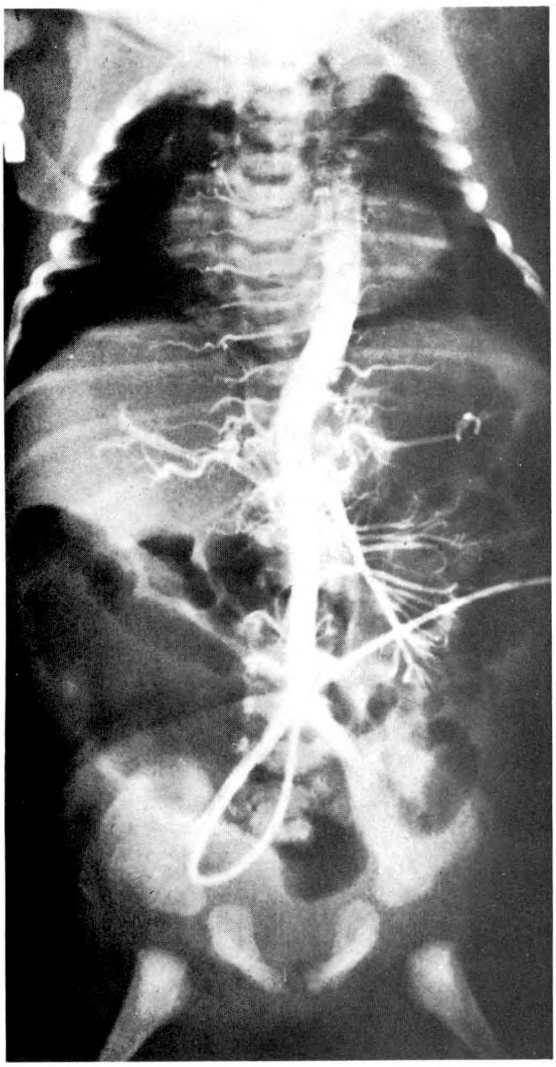


FIG. 1. This infant had imperforate anus with rectovesical fistula, segmental absence of sacrum, hypoplastic lungs, pneumomediastinum and pneumothorax. The kidneys were present as tiny dysplastic remnants. Note small pelvic bony outlet. The umbilical aortogram shows absence of filling of the renal arteries. (Courtesy of Dr. H. Kaufman, Philadelphia.)

At autopsy the lungs were markedly hypoplastic. Pulmonary interstitial emphysema, as well as pneumothorax and pneumomediastinum were noted. Renal and ureteral agenesis were noted and the bladder was hypoplastic.

Comment. As in Case 1, the prompt onset of the respiratory distress in an odd-looking infant with the "oligohydramnios" group of anomalies suggested the presence of lethal renal malformation. This was confirmed at autopsy. The 2 cases exhibited interstitial pulmonary emphysema and its progression to pneumomediastinum and pneumothorax as part of "airblock;" the "hypoplasia" of the lungs in both cases was limited to observation of their weight and size and not further detailed in terms of bronchial or alveolar development.[1]

CASE III. *Small Pelvic Bony Outlet in a Neonate with Segmental Sacral Agenesis and Normal Kidneys.*

This 1,800 gram male infant was born after a 32 week gestation. There was a normal amount of amniotic fluid and the infant had a normal appearance. Respiratory distress developed only hours after birth and the initial

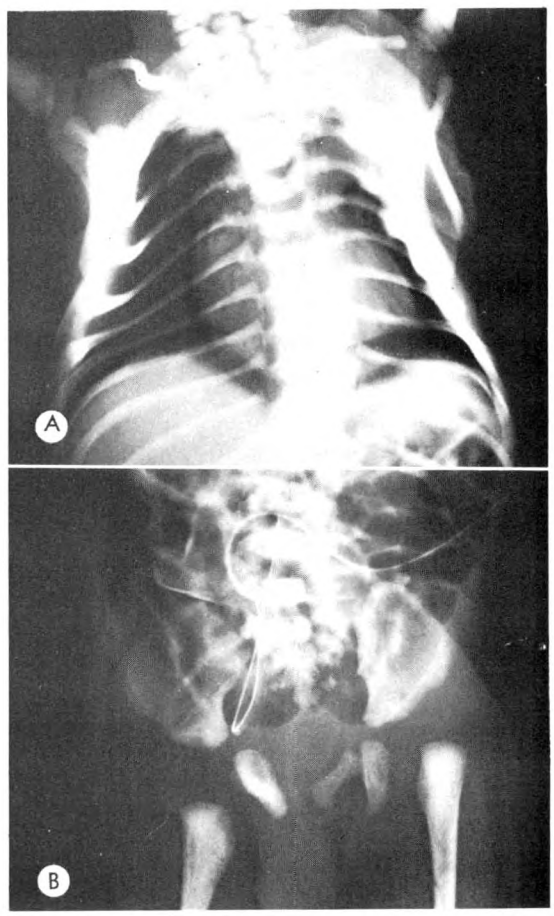


FIG. 2. Case 1. *Renal agenesis.* (*A*) Frontal chest roentgenogram shows right pneumothorax; at postmortem examination, mediastinal emphysema and hypoplastic lungs were also noted. (*B*) Frontal roentgenogram of the pelvis shows normal sacrum and normal pelvic bony outlet.

roentgenographic studies showed typical granular densities and air bronchograms of the "respiratory distress syndrome" (*i.e.*, hyaline membrane disease). After an initially stormy course (with intubation, oxygen and respirator therapy) he went through a stage of pulmonary overinflation (characterized by focal and general hyperaeration) probably due to oxygen toxicity. Finally, the chest roentgenograms returned to normal.

Roentgenogram of the chest showed healing phase of the respiratory distress syndrome (Fig. 4*A*). The patient was found to have absent anal sphincter tone and diminished reflexes in the lower extremities. Intravenous pyelogram obtained at several weeks of age showed normal kidneys (Fig. 4*B*) with a small bladder. The latter was confirmed at catheter cystography (Fig. 4*C*), which also showed the small pelvic bony outlet. Vesicoureteral reflux was absent.

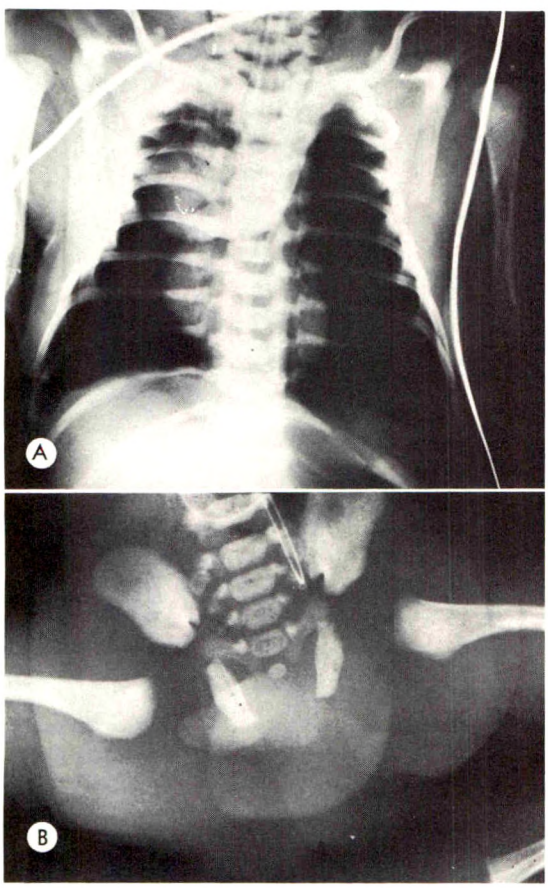


Fig. 3. Case 11. *Renal agenesis.* (*A*) Frontal chest roentgenogram shows mediastinal emphysema and bilateral pneumothoraces. At postmortem examination hypoplastic lungs were noted. (*B*) Frontal roentgenogram of the pelvis shows normal sacrum and pelvic bony outlet.

At 6 months of age a neurologic examination showed an autonomous bladder, poor rectal sphincter tone and bilateral club feet. Surgical exploration was undertaken since the clinical level of neurologic deficit was higher than that to be expected solely with segmental sacral agenesis. Myelography performed preoperatively had shown an enlarged conus. At operation the conus was needled, but no fluid could be aspirated; further surgery was not done.

Follow-up roentgenographic studies have shown continued normal renal growth. Vesicoureteral reflux is still absent, the bladder being still somewhat small with overflow incontinence. Interestingly, the bony pelvis has assumed a more normal configuration with growth of the child (Fig. 4*D*).

Comment. This patient, like those of Currarino[2] and Rabinowitz *et al.*[6] had a small bony pelvic outlet. He also had segmental sacral agenesis, *as did all of their patients*. Unlike theirs, he lacked the renal malformations that proved to be incompatible with life. He also lacked the high type of imperforate anus with a rectovesical fistula that was found in *all of their cases*.

DISCUSSION

The previous descriptions of a contracted bony pelvic outlet[2,6] in patients with respiratory distress at birth were in patients who died and proved to have a complex of malformations including renal agenesis or dysplasia and hypoplasia incompatible with survival (Table 1) (Fig. 1). All the patients had high anorectal malformations (with a fistula between rectum and bladder) and segmental sacral agenesis. Club feet and Potter's facies were commonly present not as "congenital" anomalies but as oligohydramnios deformities. The pulmonary hypoplasia is probably also related to this and is common to any renal anomaly associated with oligohydramnios whether there are kidneys or not.[1]

Cases 1 and 11 were of interest since the patients had bilateral renal agenesis, oligohydramnios, pulmonary hypoplasia, but a *normal bony pelvis*. We initially wondered if the obligatory oligohydramnios might have been the cause of the normal appear-

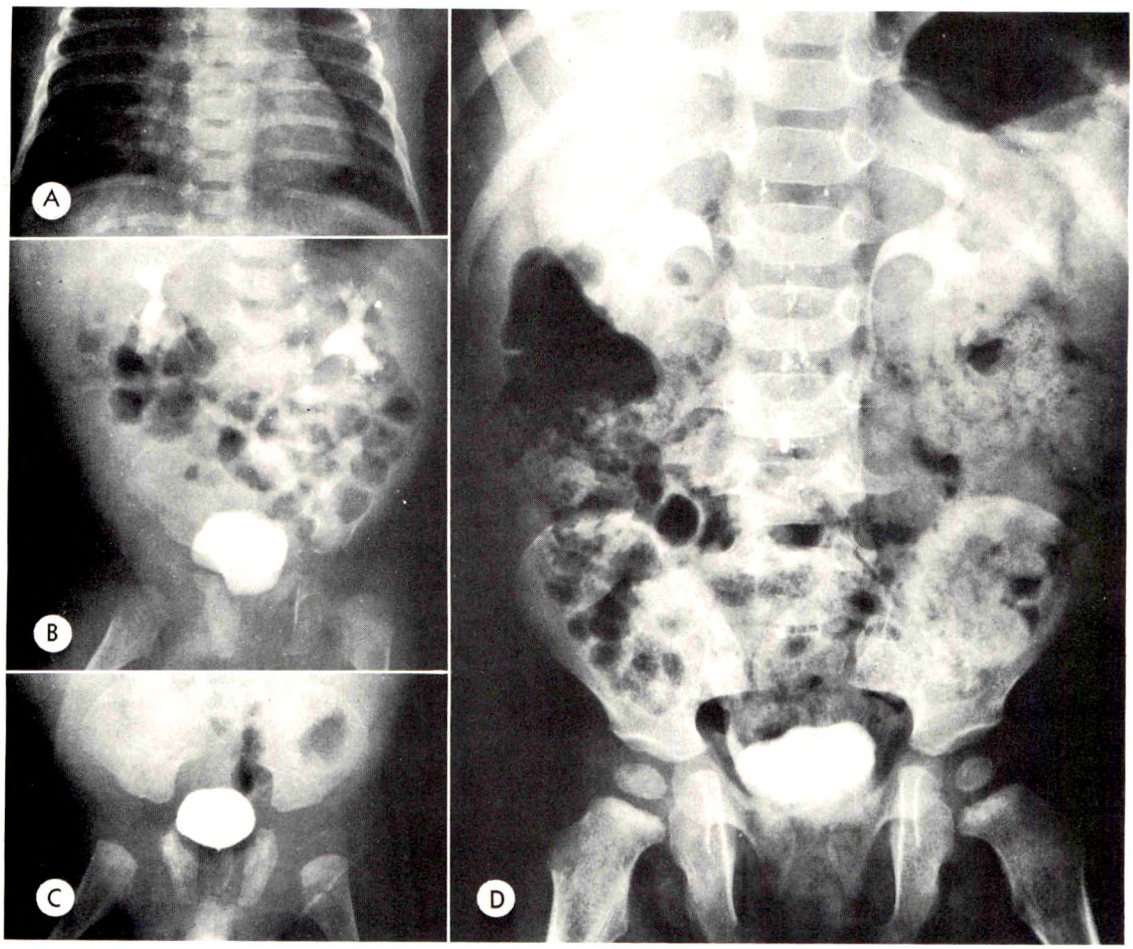


FIG. 4. Case III. *Segmental sacral agenesis.* (*A*) Frontal chest roentgenogram during recovery phase of respiratory distress syndrome. (*B*) Excretory urogram shows normal kidneys. (*C*) Cystogram shows small bladder without vesicoureteral reflux and small pelvic bony outlet. (*D*) Follow-up excretory urogram done at 21 months of age shows continued normal renal growth. Note growth of pelvic bony outlet to normal appearance.

ing bony pelvic outlet. Possibly one infant might be compressed in one fashion so as to separate the pubic bones, where another might have them squeezed together.[1] Case III still seemed to fit Currarino's[2] observation of small bladder-small pelvis, even in the presence of normal kidneys. The finding in Case III of segmental sacral agenesis did not assume a prime role in our thinking until Magnus[5] published her dissections of stillborn and newborn cases of rectovesical fistula.

Five of her cases had a small bony pelvic outlet identical to those of Currarino[2] and Rabinowitz *et al.*[6] All 5 had total or virtual absence of the levator ani muscles, and 4 of the 5 had segmental sacral agenesis (Table II). The obturator internus muscles virtually approximated each other in the midline. She speculated that the small bony pelvis led to the levator underdevelopment.

We would suggest the *opposite*. The common recurring denominator in all cases of the small pelvic bony outlet (Table II) is the maldevelopment of the levator ani muscles, virtually always heralded on plain roentgenograms by segmental sacral agenesis. Absence of adequate supporting muscle tone in these patients may allow approxi-

Table I

NORMAL PELVIC BONY OUTLET: RELATION TO SACRAL AND RENAL AND ANORECTAL ANOMALIES

Author	Pelvic Outlet	Sacrum	Bladder Size	Renal Function	Imperforate Anus
Fellows *et al.*					
Case I	normal	normal	small	nil	no
Case II	normal	normal	small	nil	no
Stern *et al.*[8]					
4 Cases					
No. 1, 2, 4, 8	normal	normal	small	nil	no

mation of the pelvic bones. This concept is reinforced by Case III where the kidneys are normal, the tone of the anal sphincter lacking, and the pelvic bony outlet and the bladder are small.

To further check these observations, it was possible to review additional cases of bilateral renal agenesis with pulmonary distress at birth, recently published by Stern *et al.*[8] In 4 of their 8 patients, roentgenograms of the pelvis were available[4] and *all 4 had a normal sacrum and normal bony pelvic outlet despite marked bladder hypoplasia* (Table I). These findings were identical to Cases I and II in this report.

Finally, the specific influence of the imperforate anus with rectovesical fistula on the appearance of the pelvis appears to be insignificant. Two cases, seen at Babies Hospital, of rectovesical fistula with a *normal* sacrum had a *normal* pelvic bony outlet (and normal kidneys). Bladder size in this anomaly can be small to normal to large, depending on renal function and the amount of meconium passed *in utero* from the rectum into the bladder.[5]

Duhamel's "Syndrome of Caudal Regression"—Its Relation to Segmental Sacral Agenesis and Anorectal Malformations

Duhamel[3] observed a high incidence of anomalies of the lower half of the body in infants with imperforate anus. These included lumbosacral spine anomalies, renal anomalies, and lower limb anomalies. He suggested that a spectrum existed, the severest form being the mermaid or sirenome-

Table II

SMALL PELVIC BONY OUTLET: RELATION TO SACRAL AND RENAL AND ANORECTAL ANOMALIES

Author	Pelvic Outlet	Sacrum	Bladder Size	Renal Function	Imperforate Anus
Currarino[2]					
Case I	small	abnormal	small	nil	yes
Case II	small	abnormal	small	nil	yes
Rabinowitz *et al.*[6]					
Case I	small	abnormal	small	nil	yes
Case II	small	abnormal	small	nil	yes
Magnus[5]		4/5			
5 Cases	small	abnormal	variable	nil	yes
Fellows *et al.*					
Case III	small	abnormal	small	normal	no

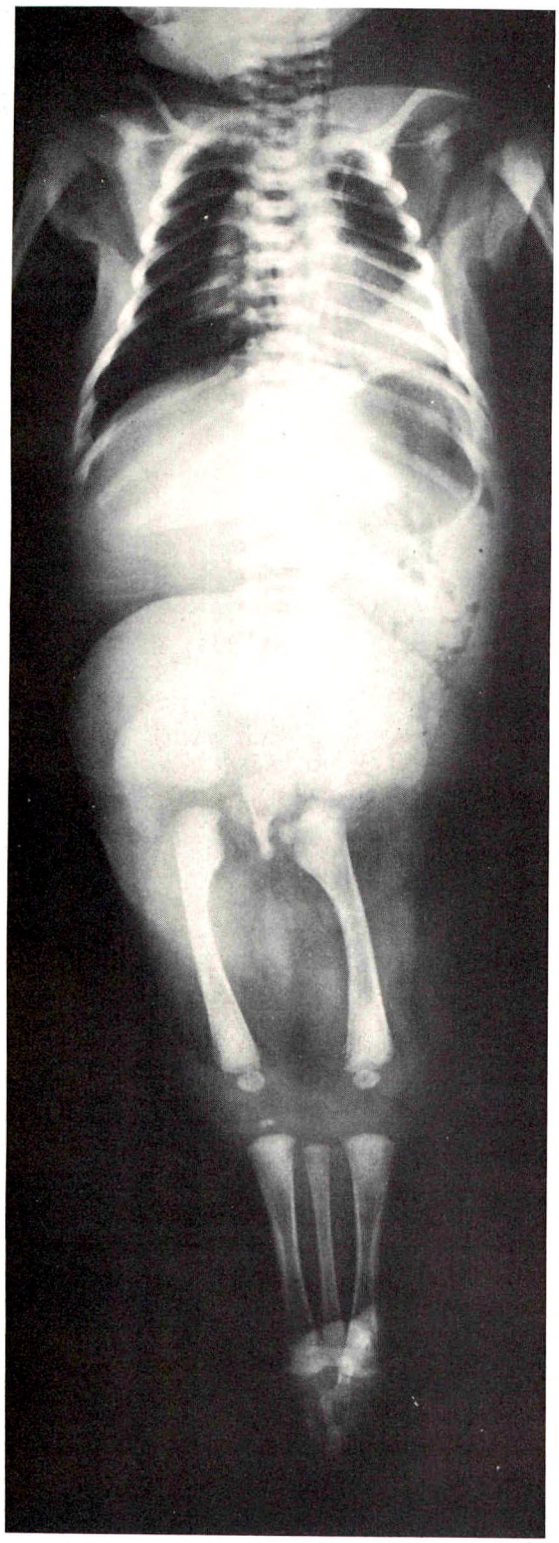


FIG. 5. *Mermaid or sirenomelus monster.* Note fused lower extremities and bony tail connected by fibrous strand to remnant of sacrum. Postmortem examination revealed hypoplastic lungs with pneumomediastinum, imperforate anus with rectovesical fistula, and tiny dysplastic renal remnants, hypoplastic bladder and patent urachus.

lus monster born with fused lower limbs, absent kidneys, rectal atresia and pulmonary hypoplasia (Fig. 5). His published example of sirenomelus monster showed segmental sacral agenesis and a small pelvic bony outlet identical to our Case III and those of Currarino[2] and Rabinowitz *et al.*[6]

Duhamel quoted Wolff,[9] who had noted that experimental selective destruction of different portions of the caudal region of the chick embryo could produce sirenomelus malformations, failure of cloacal differentiation and low spinal and limb malformations. Duhamel concluded that patients with this complex of lower spinal, lower limb, and anorectal and renal anomalies fell within the syndrome of "caudal regression."

We would utilize Duhamel's concepts to explain the roentgenographic observations of Currarino[2] and Rabinowitz *et al.*,[6] who were actually illustrating the "syndrome of caudal regression" since all of their cases had the sacral and renal and anorectal anomalies noted by Duhamel (Fig. 1). We propose that the pulmonary distress is indeed part of the renal underdevelopment, but that the undersized bony pelvic outlet relates not to the renal-bladder problem, but to the levator ani underdevelopment accompanying the segmental sacral agenesis commonly found in all such cases.

SUMMARY

A small bony pelvic outlet had previously been noted in newborns with lethal respiratory problems who had absent or nonfunctioning kidneys and bladder hypoplasia. The suggestion was made that the small pelvic bony outlet reflected the bladder development, and that these roentgenographic findings were a clue to renal agenesis in newborns with respiratory distress. All the patients also had imperforate anus

with rectovesical fistula and segmental sacral agenesis.

Three patients are reported, all of whom had small bladders. In 2 there were renal agenesis and bladder hypoplasia, but the bony pelvis was normal. In the third case, bladder hypoplasia and a small pelvic outlet were present; the patient had segmental sacral agenesis with normal kidneys and has survived.

Review of all prior cases of small bony pelvic outlet reveals the common denominator to be neuromuscular deficit accompanying segmental sacral agenesis rather than renal agenesis or bladder hypoplasia, When imperforate anus with sacral and urologic anomalies are present, the patients illustrate the syndrome of "caudal regression." Renal agenesis as an isolated anomaly is *not* associated with sacral or pelvic outlet abnormalities; bladder size *per se* plays *no* role in the appearance of the pelvic bones.

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REFERENCES

1. Blanc, W. A., Apperson, J. W., and McNally, J. Pathology of newborn and of placenta in oligohydramnios. *Bull. Sloane Hosp. Wom.*, 1962, *8*, 51–64.
2. Currarino, G. Association of congenitally small pelvic outlet with hypoplasia of bladder and urethra, and absent kidneys. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 399–402.
3. Duhamel, B. From mermaid to anal imperforation: syndrome of caudal regression. *Arch. Dis. Childhood*, 1961, *36*, 152–156.
4. Fletcher, B. D. Personal communication.
5. Magnus, R. V. Congenital rectovesical fistula and its associated anomalies. *Australian & New Zealand J. Surg.*, 1972, *42*, 197–204.
6. Rabinowitz, J. G., Pelzman, H., and Robinson, T. Small pelvic outlet associated with underdevelopment of urinary tract and other anomalies. *Radiology*, 1971, *101*, 629–630.
7. Renert, W. A., Berdon, W. E., Baker, D. H., and Rose, J. S. Obstructive urologic malformations of fetus and infant: relation to neonatal pneumomediastinum and pneumothorax (air-block). *Radiology*, 1972, *105*, 97–105.
8. Stern, L., Fletcher, B. D., Dunbar, J. S., Levant, M. N., and Fawcett, J. S. Pneumothorax and pneumomediastinum associated with renal malformations in newborn infants. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 785–791.
9. Wolff, E. La Science des Monstres. Gallimard, Paris, 1948.

NEONATAL TRANSPLACENTAL RUBELLA SYNDROME*

ITS EFFECT ON NORMAL MATURATION OF THE DIAPHYSIS

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ROENTGENOGRAPHIC descriptions of the mid-diaphysis in the perinatal period are controversial. The relatively thick cortex with relatively narrow medullary cavity has been called physiologic sclerosis.[9] Reports on the mid-diaphyseal density, in erythroblastosis fetalis are contradictory. Follis *et al.*,[10] Ritvo *et al.*,[15] and Bishop[6] all stressed the finding of increased density, whereas Brenner and Allen,[8] in a review of 100 cases, found a 5–10 per cent incidence of increased density which was similar to that in healthy neonates. The literature on skeletal changes in intrauterine viral diseases has concerned mainly the metaphysis,[13,14,16] with little mention of the diaphysis.

The present report concerns the mechanisms of normal human diaphysis maturation as evaluated by roentgenographic and histologic techniques and the influence of transplacental rubella on this maturation.

MATERIAL AND METHOD

Femurs of 30 abortuses and newborn infants were obtained at necropsy. Each femur was dissected free of soft tissues and roentgenographed on a Faxitron unit employing a 0.5 cm. focal spot. Exposure factors were 30 kvp. and 3 ma. for 3 minutes. A 3 mm. thick mid-sagittal slice of the left femur was then excised on a bandsaw and the slices were roentgenographed with the same technique, but with 2 minutes of exposure time. Total diameter and medullary width were recorded at the narrowest level of the diaphysis. Medullary width and medullary width as percentage of total diameter were plotted against body weight.

The right femur was fixed in 10 per cent formalin and demineralized *in toto* under water pump vacuum in 10 per cent formic acid buffered to pH 4.5 with sodium citrate. A 2 mm. thick transverse section was excised at the narrowest level of the diaphysis and a 2 mm. thick and 20 mm. long mid-sagittal section was excised proximally to the transverse section. The slices were embedded in paraffin, cut at 6 microns and stained with hematoxylin and eosin (H&E) and with toluidine blue.

The clinical roentgenograms were reviewed in 5 culture-proven cases of neonatal transplacental rubella syndrome with weights recorded at birth and at time of roentgenography. The medullary width as percentage of total diameter was plotted against body weight and compared with standards for normal neonates.

RESULTS

ROENTGENOGRAPHIC EXAMINATION OF NECROPSY MATERIAL

The diaphyseal diameter in fetuses up to

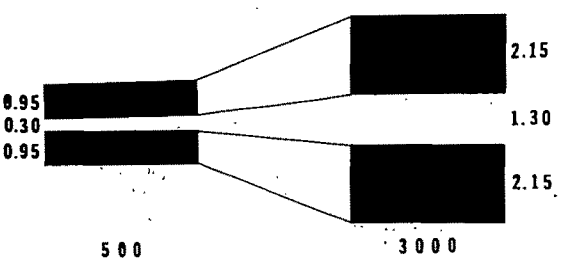


FIG. 1. Schematic illustration of relative increases in width at mid-diaphysis of cortex and medullary cavity in mm. from 500 gm. to 3,000 gm. fetus. The width of the cavity increases more than 4 times, the total diameter about 2.5 times.

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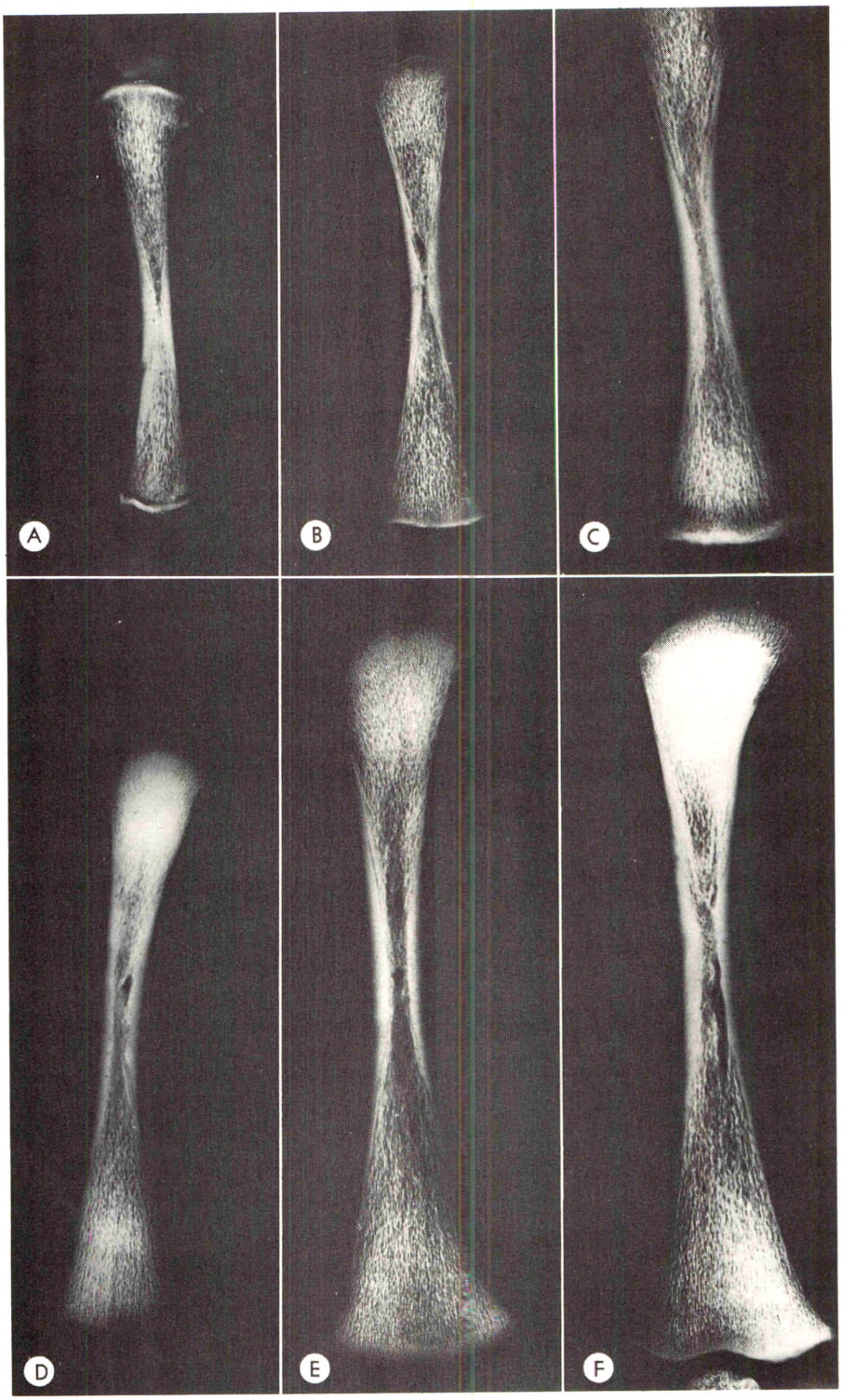


Fig. 2. (*A–F*) Roentgenograms of mid-sagittal slices of femur, necropsy specimens of fetuses weighing up to (*A*) 500, (*B*) 1,000, (*C*) 1,500, (*D*) 2,000, (*E*) 2,500 and (*F*) 3,000 gm., respectively. Progressive widening of the medullary cavity at mid-diaphysis and progressive thickening of cortices through the series. Note that the cortices are actually thinnest in the 500 gm. fetus (*A*), but the close proximity of the cortices gives an impression of sclerosis.

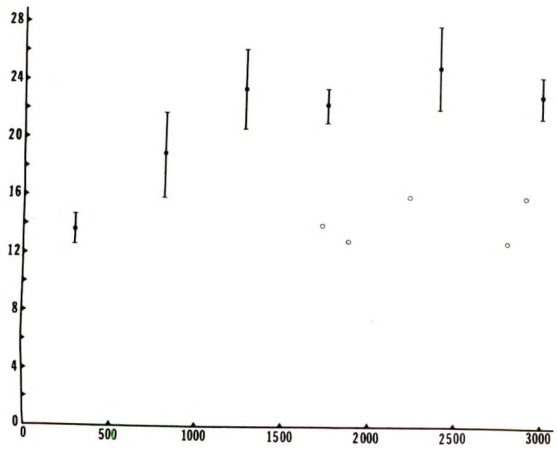


FIG. 3. Mean medullary cavity width in mm. ±s.e.m. on ordinate; as function of body weight on abscissa; location of means on abscissa shows mean fetus weight of each weight group. The number of cases in each group is 11, 5, 5, 3, 3, and 3, respectively. The values increase with increasing body weight. The difference between means of the first 2 groups is significant at $P<0.001$.

500 gm. averaged 2.2 mm. and the medullary cavity diameter 0.3 mm. The corresponding values for fetuses in the 2,500 to 3,000 gm. range were 5.6 and 1.3 mm., respectively. The relative increases in total diameter and medullary cavity are shown graphically in Figure 1. Roentgenograms of femurs from fetuses weighing up to 500, 1,000, 1,500, 2,000, 2,500, and 3,000 gm. are presented in Figure 2, *A–F*.

The medullary width as function of body weight is shown in Figure 3, and percentage medullary cavity as function of body weight in Figure 4.

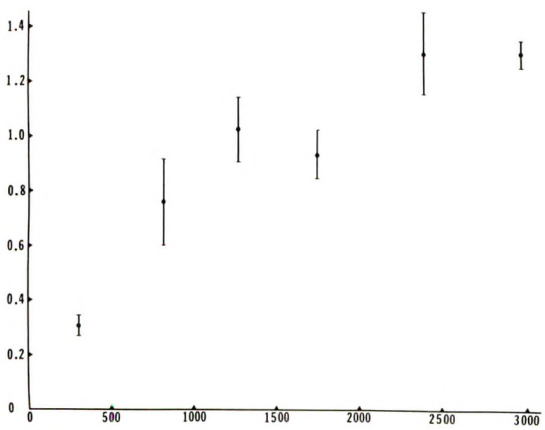


FIG. 4. Medullary cavity width as percentage of total diameter on ordinate; as function of body weight on abscissa. The symbols are the same as in Figure 3. The percentages increase with body weight. The difference between means of the first 2 groups is significant at $P<0.05$. The open circles represent 5 rubella cases. The mean rubella values are significantly ($P<0.001$) less than the mean of the normal values of the corresponding weight groups.

HISTOLOGIC EXAMINATION

The progressive increase in diaphyseal width and widening of the medullary cavity as depicted in Figure 1 reflected different apposition to resorption ratios in peripheral (subperiosteal) trabeculae and in trabeculae facing the medullary cavity. Surfaces of subperiosteal bone were lined by large osteoblasts, often in double layers. Bone

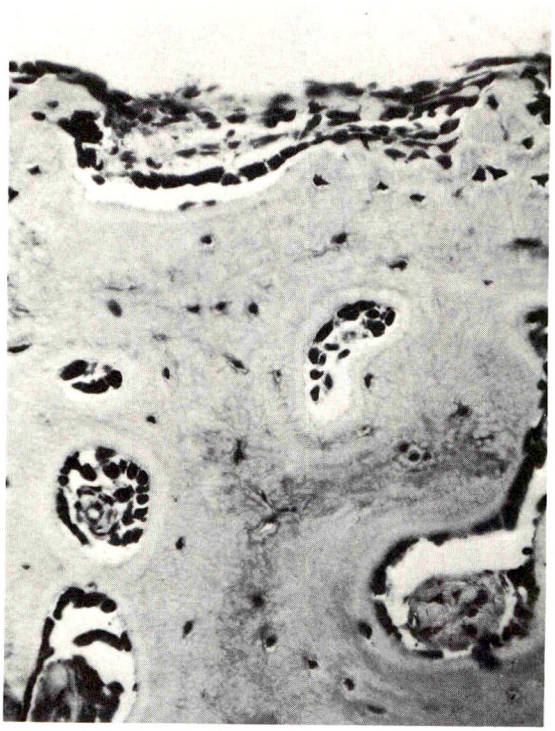


FIG. 5. Transverse section of femur mid-diaphysis of 3,000 gm. fetus. The periosteum is at top. There is a high apposition to the resorption ratio: the bone surfaces are lined by large osteoblasts, sometimes in double layers; and there are only minor signs of osteolysis in the deepest part of the trabeculae. (Toluidine blue ×250.)

resorption in this area was by osteocytic osteolysis. Deep-seated osteocytes were somewhat enlarged and their lacunar rims were sometimes metachromatic in toluidine blue stained sections (Fig. 5). The central part of this trabecular bone was diffusely but rather weakly metachromatic. The rate of osteolysis was, therefore, considered quite slow. In the bone facing the medullary cavity, the histologic picture was reversed. Apposition was minimal with surfaces either lined by rather small osteoblasts or void of osteoblasts. Osteolysis was very active (Fig. 6). The intense metachromasia typical of enhanced osteolysis in this area contrasted sharply to the staining in the middle and peripheral parts of the cortex in a low power picture of a longitudinal section (Fig. 7). A close-up of the innermost trabeculae showed, in addition to the histologic signs of increased osteolysis in the H&E stained section, a loss of birefringency with polarized light exposure (Fig. 8, *A* and *B*), reflecting degradation of collagen in the osteolytic process. Osteoclasts were not observed anywhere in the diaphysis.

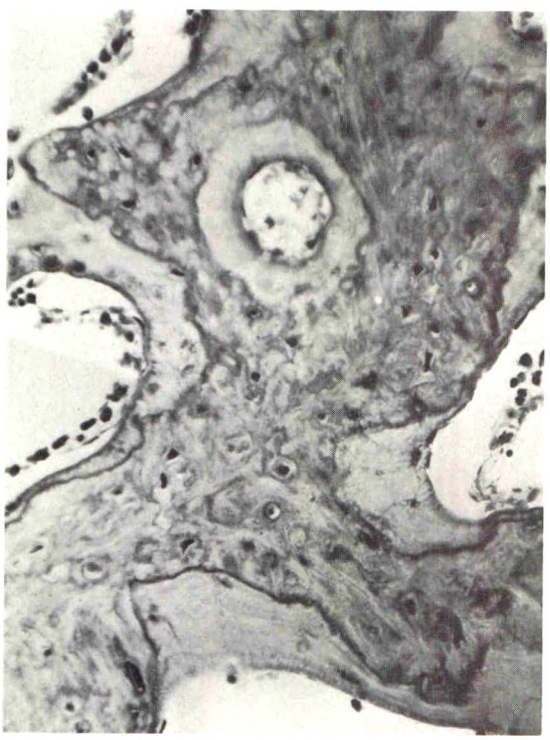


Fig. 6. Same section as in Figure 5; the trabeculae are facing the medullary cavity. There is high resorption to the apposition ratio: there is a very intense osteolysis evidenced by enlargement of the deep-seated osteocyte and metachromasia of the lacunar rims and matrix. The bone surfaces are lined by small osteocytes or void of osteoblasts. (Toluidine blue ×250.)

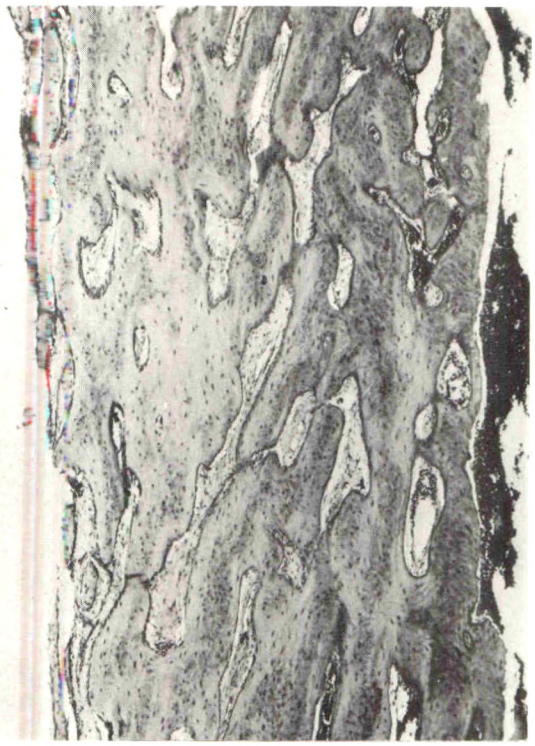


Fig. 7. Sagittal section of femur cortex at mid-diaphysis of 3,000 gm. fetus. The medullary cavity is to the right. Metachromasia, typical of osteolysis, occurs at the inner third of the cortex. (Toluidine blue ×35.)

Roentgenographic Examination of Rubella Material

The width of the medulla at birth, expressed as percentage of total diameter, in the 5 rubella cases is shown in Figure 4. All values were below the lower limits in the normal standards of similar weights. Figure 9 shows the roentgenographic picture at birth. By 8 weeks of age, the medullary width had expanded to more normal values (Fig. 10; same child as in Fig. 9). The return to normalcy coincided with disappearance of metaphyseal irregularities.

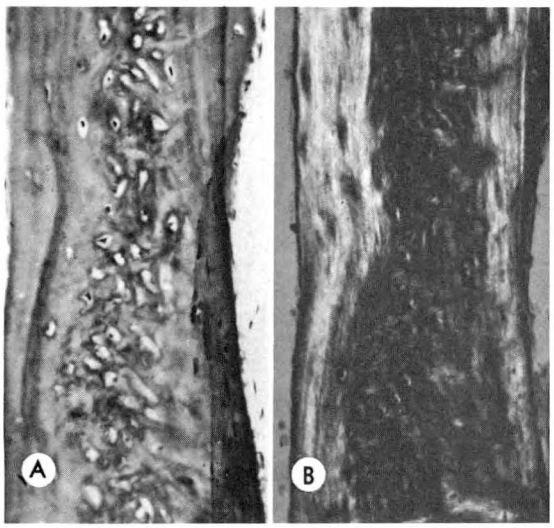


FIG. 8. (*A*) Sagittal section of trabecula facing the medullary cavity in 3,000 gm. fetus, exposed with regular light, shows intense osteolysis in center 2/3. (*B*) Same field, exposed with polarized light, shows lack of birefringency as a sign of degradation in the osteolytic process. (H. & E. ×180.)

DISCUSSION

The present study showed that the marrow cavity is expanded progressively during normal maturation of the diaphysis. The cortex also increases in width, but at a relatively less rate. Histologic examination explained this expansion of the marrow cavity by a high apposition to resorption ratio in the subperiosteal bone and a high resorption to apposition ratio in the bone facing the medullary cavity. Interpretation of the histologic picture rests on application of 2 rather recent concepts in bone metabolism, *viz.* osteocytic osteolysis and bone flow. Osteolysis defines resorption by deep-seated osteocytes which are enlarged with metachromasia and basophilia of the lacunar rim and diffusely of the adjacent matrix.[2,3,5,17] This internal resorption does not, in normal remodeling, result in internal spaces—a finding explained by the bone flow concept which holds that bone is constantly renewed by a flow from the apposition surface toward the area of osteolysis deep in the bone tissue.[1,4,12] Thus, when surface apposition exceeds internal osteolysis, the volume of the trabeculae increases; this is the situation in subperiosteal trabeculae. When internal resorption exceeds surface apposition, the surfaces will recede toward the center by the bone flow mechanism; this is the situation in trabeculae facing the marrow cavity. The shrinkage progresses to disappearance of these trabeculae and the marrow cavity expands. Osteoclasia is totally inadequate in explaining this expansion. If osteoclasia were of any importance in the modeling of the diaphysis, osteoclasts and Howship's lacunae should have been observed, no matter how short the life-span of the osteoclasts is believed to be.

The term physiologic sclerosis describes the roentgenographic picture in the normal neonate with a relatively thick cortex and a narrow medullary cavity. In transplacental rubella, the width of the medullary cavity



FIG. 9. Clinical roentgenogram at birth of femur, of 2,950 gm. white female infant with transplacental rubella syndrome. The medullary cavity is narrow with 16 per cent medullary cavity of total diameter, significantly below standards shown in Figure 4. Distal femoral ossification center, normally visible in 98 per cent of white females of similar weight,[9] is not visible in the roentgenogram. Note irregularity of the distal metaphysis.

was shown to be less than the lower range of normal values of corresponding weight. Accordingly, the diaphyseal changes in rubella should be interpreted as a delay of maturation. The delay appears to correct itself in several weeks, coincident with the correction of the metaphyseal abnormality. The latter has been described as a failure of normal modeling in that area with a diminutive distal ossification center in the femur.[18] The metaphyseal abnormality, which is not related to inflammation since there is no osteitis,[16] may then merely reflect a growth arrest. Histologically, the lesions resemble those in premature infants of mothers with severe cyanotic cardiac abnormalities.[7] This alteration of growth and failure of modeling of the metaphysis would be compatible with the delayed modeling of the diaphysis.

SUMMARY

Modeling of the femur diaphysis in the perinatal period was studied in 30 normal fetuses and infants and in 5 cases of transplacental rubella.

The total diameter and medullary cavity width increase progressively with increasing body weight. In subperiosteal trabeculae there is a high apposition to resorption ratio and bone mass increases. In trabeculae facing the medullary cavity there is a high resorption to apposition ratio; bone mass decreases and the medullary cavity expands. Bone resorption is by osteocytic osteolysis.

In transplacental rubella, the diaphyseal modeling is delayed beyond the limits of physiologic sclerosis. Normal modeling is achieved within 2 months, coincident with correction of the metaphyseal abnormalities.



Fig. 10. Clinical roentgenogram of femur of the same infant as in Figure 9, 8 weeks later. The medullary cavity is now of normal width. The distal femoral ossification center is visible and the irregularity of the distal metaphysis has disappeared.

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REFERENCES

1. Belanger, L. F. Osteolysis: outlook on its mechanisms and etiology. In: Parathyroid Hormone, Ultrastructure, Secretions and Functions. Edited by P. Gaillard, R. V. Talmage, and A. Budy. University of Chicago Press, Chicago, 1965, pp. 137–143.
2. Belanger, L. F. Osteolysis in pathological material. *Cornell Vet.*, 1968, *58*, Suppl. 1, 115–135.
3. Belanger, L. F. Osteocytic osteolysis. *Calcif. Tiss. Res.*, 1970, *4*, 1–12.
4. Belanger, L. F., and Migicovsky, B. B. Bone cell formation and survival under various conditions. *Anat. Rec.*, 1963, *145*, 385–390.
5. Belanger, L. F., Semba, R., Tolnai, S., Copp, D. H., Krook, L., and Gries, C. The two faces of resorption. Proc. 3rd Europ. Symp. Calcif. Tissues. Edited by H. Fleisch, H. J. Blackwood, and M. Owen. Springer-Verlag, Berlin, 1966, pp. 1–10.
6. Bishop, P. A. Roentgenologic diagnosis of fetal hydrops. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1961, *86*, 415–424.
7. Black-Schaffer, B. Fetal nanosomia and bone athrepsia in newborn of woman with severe cyanotic cardiovascular anomaly. *Am. J. Obst. & Gynec.*, 1950, *59*, 656–661.

8. BRENNER, G., and ALLEN, R. P. Skeletal changes in erythroblastosis fetalis. *Radiology*, 1963, *80*, 427–429.
9. CAFFEY, J. Pediatric X-ray Diagnosis. Fifth edition. Yearbook Medical Publishers, Inc., Chicago, 1967, pp. 779–780.
10. FOLLIS, R. H., JR., JACKSON, D., and CARNES, W. H. Skeletal changes associated with erythroblastosis fetalis. *J. Pediat.*, 1942, *21*, 80–92.
11. GRAHAM, B., THAL, A., and WASSUM, C. Rubella-like bone changes in congenital cytomegalic inclusion disease. *Radiology*, 1970, *94*, 39–43.
12. KROOK, L., BELANGER, L. F., HENRIKSON, P. A., LUTWAK, L., and SHEFFY, B. E. Bone flow. *Rev. Canad. Biol.*, 1970, *29*, 157–165.
13. MERCER, R. D., LUSE, S., and GUYTON, D. Clinical diagnosis of generalized cytomegalic inclusion disease. *Pediatrics*, 1953, *11*, 502–504.
14. RABINOWITZ, J. G., WOLF, B. S., GREENBERG, E. I., and RAUSEN, A. R. Osseous changes in rubella embryopathy (congenital rubella syndrome). *Radiology*, 1965, *85*, 494–499.
15. RITVO, M., SCHAUFFER, I. A., and KROSNICK, G. Clinical and roentgen manifestations of erythroblastosis fetalis. AM. J. ROENTGENOL. & RAD. THERAPY, 1949, *61*, 291–301.
16. SINGLETON, E. B., RUDOLPH, A. J., ROSENBERG, H. S., and SINGER, D. B. Roentgenographic manifestations of rubella syndrome in newborn infants. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 82–91.
17. WHALEN, J. P., WINCHESTER, P., KROOK, L., DISCHE, R., and NUNEZ, E. Mechanisms of bone resorption in human metaphyseal remodeling: roentgenographic and histologic study. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *112*, 526–531.
18. WILLIAMS, H. J., and CAREY, L. S. Rubella embryopathy: roentgenologic features. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 92–99.

Vol. 121, No. 1

BILATERAL PSEUDOTUMORS OF THE PELVIS IN A PATIENT WITH CHRISTMAS DISEASE*

WITH NOTES ON LOCALIZATION BY RADIOACTIVE SCANNING AND ULTRASONOGRAPHY

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PSEUDOTUMORS arising in patients with coagulation defects are rare, the reported incidence in several large series of hemophilic patients varying from 0.5 to 2 per cent.[1] The most common sites are the thigh and pelvis but a small number of cases have been reported in the calves, feet, arms and hands. Bilateral pseudotumors have been recorded previously in only 2 patients; these involved the cuboid bones of the feet[2] and the pelvis.[3]

In several of the case reports, including the original, there has been doubt about the diagnosis, the suspicion being that the destructive lesion might be an osteogenic sarcoma.[7–9]

The purpose of this study was to localize the tumors and determine if a hemophilic pseudotumor could be distinguished from an osteogenic sarcoma using radiologic techniques, ultrasonography and radioisotope scanning with strontium 87m and technetium 99m.

METHOD

For scintiscanning, strontium 87m (2 mc) was given intravenously and 55 minutes later the lower abdomen, pelvis and upper femora were scanned in that order using a dual headed scintiscanner. A color dot and photo scan was obtained. On a separate occasion a similar procedure was carried out 20 minutes after technetium 99m (10 mc) had been given intravenously.

Ultrasonography was performed using a Nuclear Enterprises Diasonograph.

REPORT OF A CASE

The patient is now aged 25 years and has a severe degree of Factor IX deficiency (Christmas disease) with a zero per cent assay of Factor IX. In 1956, at the age of 9 years, he developed a painful tense swelling in the left groin associated with numbness down the front of the thigh. Roentgenograms of the pelvis showed a homogeneous soft tissue mass measuring 9 cm. in diameter overlying the left iliac crest, encroaching on the upper part of the sacroiliac joint as well as the distal third of the left psoas muscle with no bone erosion. Treatment with plasma was given for a week with some slight resolution of the mass but the patient then defaulted from further attendance.

In 1963 he developed a similar pain and swelling in the right groin with a femoral nerve palsy which was treated in his home without plasma infusions and in 1964 he had a recurrence of pain at the same site. Thereafter he was reasonably well except for recurrent episodes of hematuria and acute hemarthroses of the knees, ankles and shoulders. The patient was readmitted to hospital in 1971 having fallen and fractured the right tibia and the left patella.

Examination of the abdomen showed 2 large swellings arising out of each side of the pelvis. Both were hard, nontender and nonfluctuant. The swelling on the left was largest, extending to just above the umbilicus. There were bilateral femoral nerve lesions with absent knee jerks and gross wasting of both quadriceps muscles.

Plain roentgenograms of the pelvis showed erosion of the blade of the ilium on the left side. Continuous with this area of bone destruction there was a soft-tissue mass within which were

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Fig. 1. Roentgenogram of the pelvis showing destructive lesion of the left ilium and soft tissue swelling near the left iliac crest. There are also "hemophilic" changes in the right hip joint.

areas of calcification. Osteosclerotic areas were also seen in the ilium (Fig. 1). On intravenous pyelography it was seen that the bladder and left ureter were displaced to the right. The appearance of the lower end of the left ureter suggested further that it was held in fixed dilatation as a result of its involvement by the pseudotumor (Fig. 2). A barium meal and follow-through confirmed the appearances previously noted, namely that both small and large bowel were displaced by the large left-sided pseudotumor and also by a smaller but presumably similar lesion on the right side (Fig. 3).

At this stage ultrasonography was performed with a view to confirming that there were in fact 2 separate masses. It was also considered that ultrasonic scanning would constitute a simple and safe method of assessing the effects of therapy on the size of the lesions. As expected, 2 discrete lesions were shown (Fig. 4).

Examination of the ultrasonographs showed that these lesions were poorly trans-sonic, *i.e.*, were fairly solid structures, and were of varying consistency. This, together with their rather indistinct margins, makes them indistinguishable from malignant tumors such as an osteogenic sarcoma.

The scan pictures after strontium 87m and technetium 99m injections were similar and only the strontium 87m photoscan of pelvis is shown (Fig. 5). This reveals a large round volume of abnormal uptake in the right pelvis and an elongated volume of abnormal uptake extending laterally and superiorly in the left pelvis.

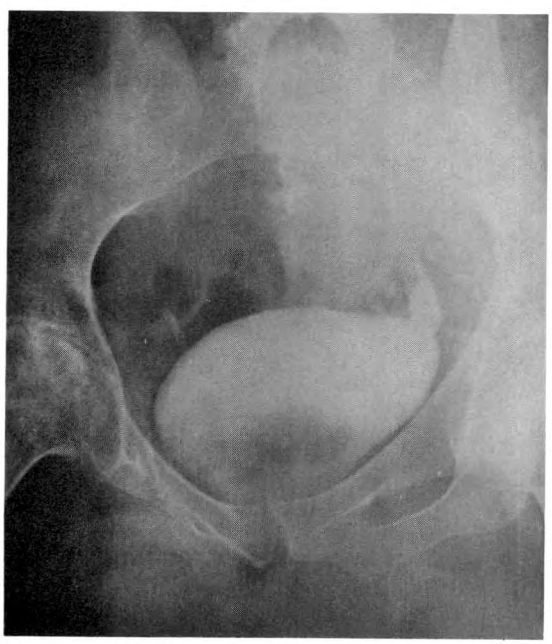


Fig. 2. Intravenous pyelogram showing dilated lower end of the left ureter with displacement of the bladder by the pseudotumors.

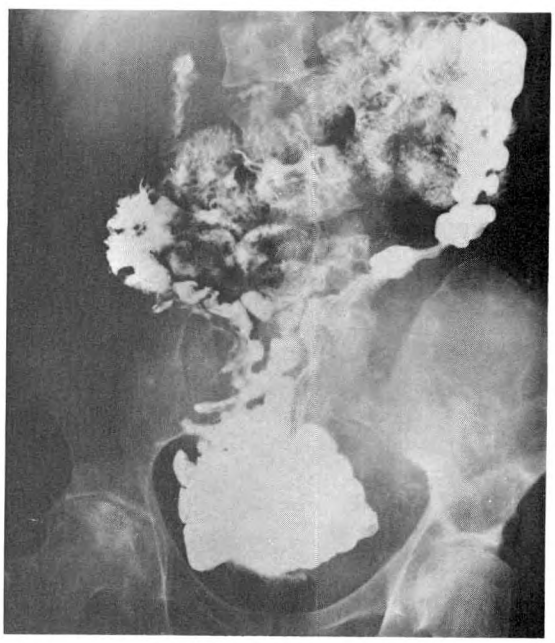


Fig. 3. Barium meal and "follow-through" examination showing displacement of the sigmoid and descending colon and the cecum.

Both regions correspond to the sides of the pseudotumors. The general appearances are similar to those we have found in known osteogenic sarcoma.

DISCUSSION

At least 49 cases of pseudotumors have been recorded in hemophiliacs since the original case report of Starker in 1918[9] and of these 20 have involved the ilium. As suggested by Valderrama and Mathews,[10] hemophilic hematomas may arise in muscle where they are confined by the tendinous attachments (simple cyst), or in a muscle with a large periosteal attachment where cortical thinning is produced by interference with bone nutrition; alternatively, they may arise from a hematoma which strips the periosteum from the cortex until limited by an aponeurotic or tendinous attachment. The end result of this is a tumor, the roentgenographic appearances of which resemble an osteogenic sarcoma. It is impossible, in retrospect, to determine the site of origin of this patient's pseudotumors, but the initial episodes of pain and femoral nerve compression suggest strongly that they started as hematomata in the iliacus muscle.[4] The lesion on the left has probably been present for 16 years and the lesion on the right for 8 years. As judged both clinically and radiologically, the left pseudotumor has grown over the years, but it is not possible to say whether this has occurred gradually or whether it is still enlarging. It seems probable from the history that the 2 tumors have arisen from separate sites and both the ultrasonic and photo scans suggest that they are discrete entities.

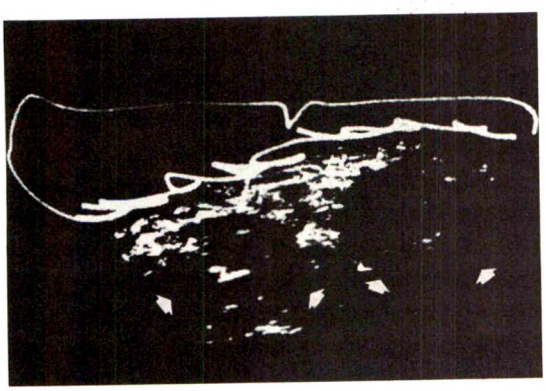


Fig. 4. Ultrasonograph of the lower abdomen (2.5 MHz, -10). This is a transverse section of the abdomen 1 inch below the umbilicus showing 2 discrete lesions. The lesions contain reflecting echoes and have indistinct margins.

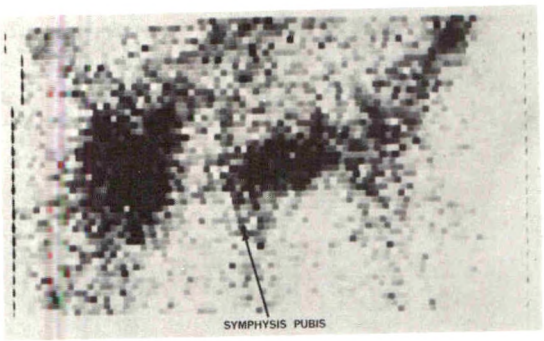


Fig. 5. Photoscan of pelvis after injection of strontium 87m showing a large round volume of abnormal uptake in the right pelvis and an elongated volume of abnormal uptake extending laterally and superiorly in the left pelvis. These areas correspond to the sides of the pseudotumors.

Although renal function, as judged by the blood urea of 25 mg./100 ml. and the creatinine clearance of 127 ml./min. is excellent, there is evidence both from the intravenou pyelogram and the isotope renogram of obstruction to the ureters. Similarly, there is as yet no interference with bowel function, but the barium meal and follow-through show distortion of the normal anatomy of the large and small bowel in the pelvis. There is 1 case report of pseudomembranous colitis in a hemophiliac associated with pseudotumor of the pelvis producing obstruction to the rectum[5] and 1 of rupture into the sigmoid colon.[6] There has been no case, to date, of renal failure resulting from compression of the ureters.

From studies of the operative case notes of the patients undergoing excision of these tumors, they appear to be extremely vascular and present major technical problems of hemostasis and in these patients macroscopic and histologic examinations confirmed the presence of large vessels in the outer fibrous zone of the capsule.[3,10] This

would explain the localization of both strontium 87m and technetium 99m in and especially around the tumors in this patient's pelvis.

Both ultrasonography and radioisotope scanning have failed to differentiate the pseudotumor from an osteogenic sarcoma, but they offer satisfactory methods of localization and measurement of the size of the tumors and should provide an objective assessment of the efficacy of treatment.

SUMMARY

A 25 year old patient with severe Factor IX deficiency was found to have bilateral pseudotumors arising from his pelvis.

Localization of these tumors and assessment of response to therapy have been attempted by a combination of conventional radiology, radionuclide scanning and ultrasonography.

An attempt has also been made to compare the characteristic ultrasonographic appearances of hemophilic pseudotumors with osteogenic sarcoma.

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REFERENCES

1. AHLBERG, A. Haemophilia in Sweden: VII, incidence, treatment and prophylaxis of arthropathy and other musculo-skeletal manifestations of haemophilia A and B. *Acta orthop. scandinav.*, 1965, *77*, 1–98.
2. CHEN, Y. F. Bilateral hemophilic pseudotumors of calcaneus and cuboid treated by irradiation. *J. Bone & Joint Surg.*, 1965, *47-A*, 517–521.
3. FRAENKEL, G. J., TAYLOR, K. B., and RICHARDS, W. C. D. Haemophilic blood cysts. *Brit. J. Surg.*, 1959, *46*, 383–392.
4. GOODFELLOW, J. W., FEARN, C. B. D'A., and MATHEWS, J. M. Iliacus haematoma: common complication of haemophilia. *J. Bone & Joint Surg.*, 1967, *49-B*, 748–756.
5. GOULSTON, S. J. M., and MCGOVERN, V. J. Pseudomembranous colitis. *Gut*, 1965, *6*, 207–212.
6. HALL, M. R. P. Haemophilia complicated by circulating anticoagulant: report of three cases. *Brit. J. Haemat.*, 1961, *7*, 340–348.
7. NELSON, M. G., and MITCHELL, E. S. Pseudotumour of bone in haemophilia. *Acta haemat.*, 1962, *28*, 137–144.
8. PAPPAS, A. M., BARR, J. S., SALZMAN, E. W., BRITTEN, A., and RISEBOROUGH, E. J. Problem of unrecognized mild hemophiliac. *J.A.M.A.*, 1964, *187*, 772–774.
9. STARKER, L. Knochenusur durch ein Haemophiles subperiostalis Haematom. *Mitt. Grenzgeb. Med. Chir.*, 1918, *31*, 381–415.
10. VALDERRAMA, J. A. F., and MATHEWS, J. M. Haemophilic pseudotumour or haemophilic subperiosteal haematoma. *J. Bone & Joint Surg.*, 1965, *47-B*, 256–265.

Vol. 121, No. 1

TECHNETIUM 99M LABELED DIPHOSPHONATE BONE SCANNING IN PAGET'S DISEASE*

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IN the intervening years since the original description of "osteitis deformans" in 1877 by Sir James Paget,[20] the efforts of numerous investigators have improved our understanding of some of the mechanisms of abnormal bone development in Paget's disease. The description of the anatomic pathology by Schmorl[25] and the observation that plasma alkaline phosphatase was markedly elevated[15] were the basis for much of the later clinical and metabolic studies. The roentgenologic aspects of the lytic and sclerotic phase of Paget's disease have been well documented,[5,9,24,29] including the observation by Sosman[27] that osteoporosis circumscripta was the destructive phase of Paget's disease in the skull.

More recently radionuclide skeletal imaging techniques have been utilized to determine the anatomic extent of the disease and to study the abnormalities of bone metabolism. Paget's disease in bone has been demonstrated with strontium 85,[17,28] strontium 87m,[28] fluorine 18,[12–28] and barium 135m[19] bone-seeking radionuclides.

The demonstration that diphosphonate strongly chemisorbs onto the hydroxyapatite crystal lattice of bone[11] contributed to its development as a carrier of a radioactive agent for use in skeletal imaging.[6]

This report summarizes the findings in 25 cases of Paget's disease which were observed during the course of over 700 bone scannings using technetium 99m diphosphonate. A more dynamic evaluation of the radionuclide concentration using a multichannel analyzer to obtain quantitative data is also included.

METHOD

The carrier molecule 1-hydroxy-ethylidene-1, 1-disodium phosphonate was synthesized in our laboratories and labeled with technetium 99m.[6] This technetium 99m diphosphonate compound is administered intravenously; the usual adult dose of 15 millicuries contains approximately 0.5 mg. of diphosphonate carrier. Skeletal images are routinely obtained of the head, trunk, and lower extremities down to the knees; when indicated, more complete imaging of the extremities is carried out. Most of the studies utilize a dual probe rectilinear scanner following a technique previously described,[14] with 2:1 reduction of the image. In a few instances scintillation camera studies were carried out with multiple anterior and posterior views of the same regions outlined in the above dual probe rectilinear scanner protocol. When a parallel hole collimator is utilized, an arbitrary time of 60 seconds is used for each gamma camera view; the time factor is increased to 90 seconds with a diverging collimator.

Dynamic studies of technetium 99m diphosphonate uptake in Pagetoid bone have been performed with a scintillation camera, whose output was digitized and recorded onto magnetic tape as a serial study. In this way a series of digitized images in time sequence for intervals up to 5 hours after intravenous injection were recorded. At the end of the storage process the images were recalled from the magnetic tape and analyzed quantitatively with a multichannel analyzer. Regions of interest were selected to include both kidneys, soft tissue, normal bone, and bone involved with Paget's disease.

RESULTS

The mixed phase of Paget's disease involving both osteolysis and sclerosis was

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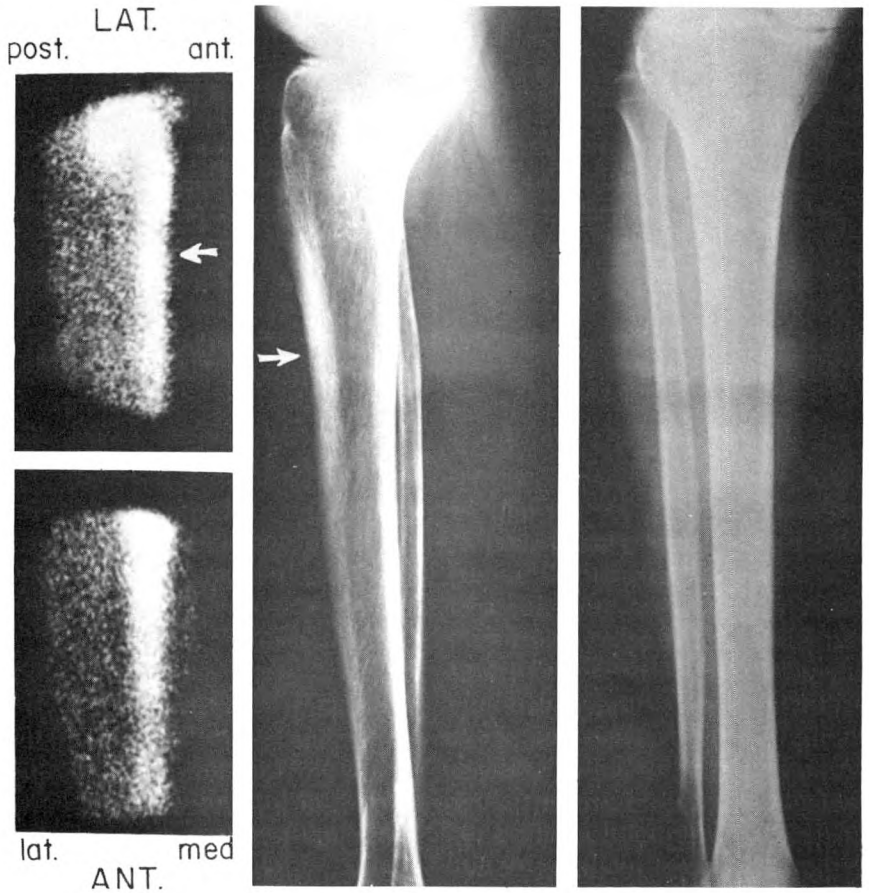


FIG. 1. Lateral and anterior views of the proximal tibia with companion roentgenograms show increased uptake along the anterior cortical margin of the tibia (arrows) corresponding to the region of Paget's disease. Uninvolved bone in the posterior part of the tibia is clearly demarcated from the diseased bone anteriorly.

the most common form of the disease in this series. The geographic extent of abnormal bone by scan usually corresponded very closely to matching roentgenograms. Bones that were often difficult to evaluate roentgenographically, such as the scapula, showed intense radionuclide uptake when involved with Paget's disease. The margin between normal and abnormal bone was more distinct on bone scans than on roentgenograms. This demarcation was useful in defining the extent of the disease in 1 patient (Fig. 1) that had increased uptake along the anterior cortical margin of the tibia. The involved portion of bone was either the periosteum or the anterior cortex of the tibia, as the medullary canal and posterior aspect of the bone were well delineated on the scan and had normal activity.

Osteoporosis circumscripta is the lytic phase of Paget's disease in the calvarium. The bone scan of 1 such patient (Fig. 2) showed considerably increased uptake corresponding to the advancing margin of the lesion. The central part of the lytic bone appeared to have lower radioactivity than the margin. This pattern is distinctly different from the scan appearance of the sclerotic phase of Paget's disease because the same bone appears to have considerably different radioactivity in different regions of the same bone.

The count rate over bones involved with Paget's disease was 3 to 5 times higher than that over normal bone. One patient in this

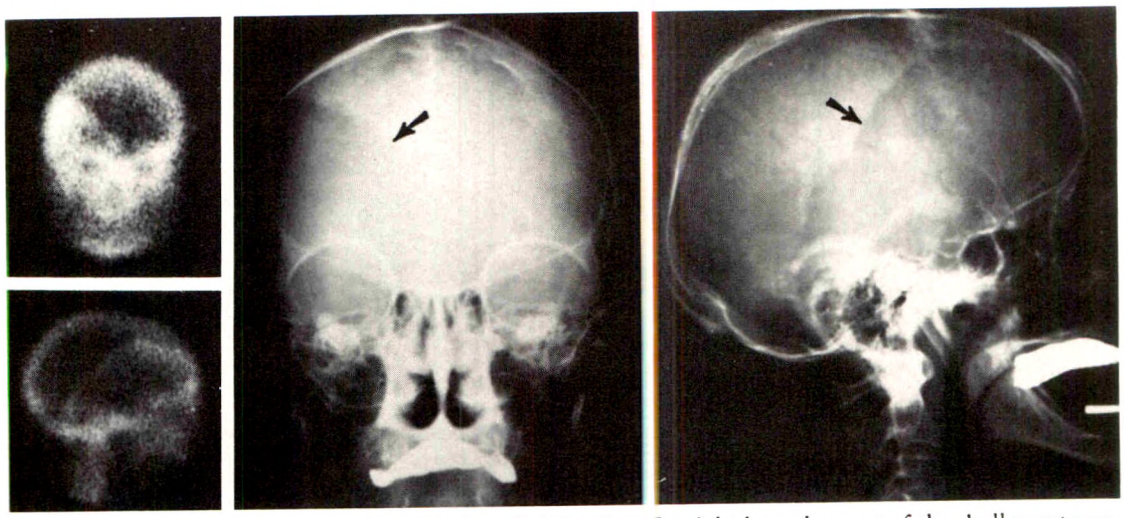


Fig. 2. Osteoporosis circumscripta (arrows) is well defined on the right lateral aspect of the skull roentgenograms. The diphosphonate bone scan shows increased uptake at the margin of the lesion with lower radioactivity in the central part of the lytic bone.

series had a count rate over the Pagetoid region which was nearly 10 times the count in adjacent normal bone. Figure 3 illustrates a dynamic study with digitized images of the gamma camera at 5 minutes, 10 minutes, 15 minutes and 5 hours after administration of technetium 99m diphosphonate. Intense radioactivity is present 5 minutes after intravenous injection in both normal and abnormal bone. The distinction between bone involved with Paget's disease and normal bone is readily apparent within minutes of the injection of diphosphonate. Figure 4 illustrates the digitized multichannel analyzer image of the 10 minute serial image. The radioactivity from both the Pagetoid bone and the kidneys is approximately comparable at 10 minutes and is well demarcated from adjacent normal bone and soft tissues. Table 1 shows the counts per channel *versus* time for the selected regions of interest. Lesion I is visible after 5 minutes and lesion II after 10 minutes. By 5 hours both Pagetoid lesions show similar technetium 99m diphosphonate concentration, both significantly higher than of normal bone. The kidneys show a gradual decrease in activity by 15 minutes and a significant reduction to background levels by 5 hours.

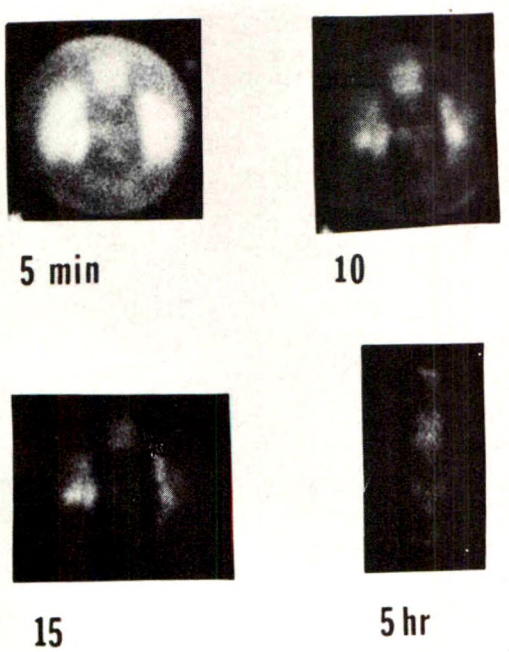


Fig. 3. Serial images of the lower lumbar spine and pelvis were stored on videotape at 5, 10 and 15 minutes, and 5 hours. Intense radioactivity is present 5 minutes after intravenous injection in the Paget's bone in the fifth lumbar vertebra and both iliac wings.

Fifteen patients with Paget's disease were also studied with fluorine 18 bone scans using 3 millicuries of the radiopharmaceutical. Five of these patients had scans with both technetium 99m diphos-

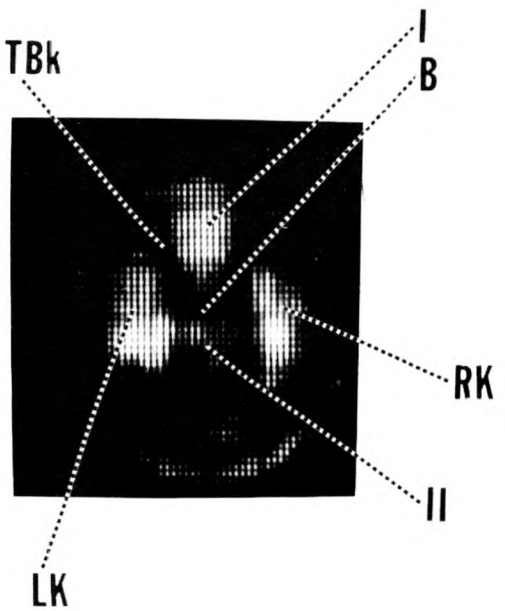


FIG. 4. Analog gamma camera scan at 10 minutes is digitized, stored and subsequently viewed with a multichannel analyzer. TBk is the tissue background; B is the normal spine; numerals I and II are lesions of Paget's disease in the lower lumbar vertebra and sacrum, respectively; and RK and LK are the right and left kidney.

phonate and fluorine 18 as fluoride. Although the scans are not quantitatively comparable because the amount of activity for each was different and the efficiency for the detection of the 140 KeV and 510 KeV energies is different, the skeletal patterns of radioactivity were comparable for both agents (Fig. 5).

DISCUSSION

Although Paget's disease of bone has been studied for nearly a century, the basic abnormality has not been clearly defined.[2,8] More recently Rasmussen and Bordier[22] have constructed an intriguing model of cellular phenomena at the endosteal surface centered around a transformation of osteoclasts into osteoblasts. From studies of the histology, bone turnover (resorption and formation) as well as the metabolic effects of such agents as calcitonin, they conclude that Paget's disease is a hormone sensitive benign tumor of bone.

Several agents such as calcitonin[13] and ACTH[1] have been used in the medical therapy of the disease in an attempt to alter the detectable biochemical abnormalities. Using therapeutic levels of diphosphonates, Smith *et al.*[26] have demonstrated a reduction in plasma alkaline phosphatase and hydroxy proline in patients with Paget's disease.

The diphosphonates, which contain a P–C–P bond, appear to modify the rate of turnover of the hydroxyapatite lattice. These compounds retard the rate of dissolution of apatite crystals[10] and inhibit the formation of calcium phosphate crystals.[11] The diphosphonate apparently is strongly chemisorbed onto the apatite lattice, thus preventing the addition of ions into the lattice.[6]

Radionuclide scintimetry has been employed to define the extent of Paget's dis-

TABLE I

REGIONS OF INTEREST*

Area	5 min.	10 min.	15 min.	5.0 hr.
Lesion I	+152	+294	+268	+119
Lesion II	−44	+37	+68	+114
Bone	−59	−9	+12.0	+48.5
Rt Kidney	+511	+448	+312	+8.8
Lt Kidney	+502	+414	+338	+10.3
Whole area	5.75×10^3	3.58×10^3	3.40×10^3	1.39×10^3 (corrected for decay)

* Regions of interest were selected on the patient illustrated in Figure 4. Counts per region of interest *versus* time are listed for 2 lesions of Paget's disease, normal bone, and both kidneys.

C. R., Jr., White, R. I., Giargiano, F. A., and Wagner, H. N., Jr. Absence of anatomic arteriovenous shunts in Paget's disease of bone. *New England J. Med.*, 1972, *287*, 686–689.

24. Seaman, W. B. Roentgen appearance of early Paget's disease. Am. J. Roentgenol. & Rad. Therapy, 1951, *66*, 587–594.
25. Schmorl, G. Über Osteitis Deformans Paget. *Virchows Arch. path. Anat.*, 1932, *283*, 694–751.
26. Smith, R., Russell, R. G. G., and Bishop, M. Diphosphonates and Paget's disease of bone. *Lancet*, 1971, 945–946.
27. Sosman, M. C. Radiology as aid in diagnosis of skull and intracranial lesions. *Radiology*, 1927, *9*, 396–404.
28. Spencer, R., Herbert, R., Rish, M. W., and Little, W. A. Bone scanning with ^{85}Sr, ^{87m}Sr and ^{18}F. *Brit. J. Radiol.*, 1967, *40*, 641–654.
29. Steinbach, H. Paget's disease. In: Radiology of the Skull and Brain. Volume 1. Book 2. C. V. Mosby, St. Louis, 1971, pp. 755–762.
30. Subramanian, G., McAfee, J. G., Boll, E. G., Blair, R. J., Mara, R. E., and Relston, P. H. ^{99m}Tc-labeled polyphosphates as skeletal imaging agent. *Radiology*, 1972, *102*, 701–704.
31. Subramanian, G., McAfee, J. G., Blair, R. J., Mehtes, A., and Connor, T. ^{99m}Tc-EHDP: potential radiopharmaceutical for skeletal imaging. *J. Nucl. Med.*, 1972, *13*, 947–950.
32. Yano, Y., McAfee, J., Van Dyke, D. C., and Anger, H. O. Tc Sn-1-hydroxy-1, 1-diphosphonate—new bone scanning agent. *J. Nuclear Med.*, 1973, *14*, 73–78.

COMPARISON OF Tc^{99m} POLYPHOSPHATE AND F^{18} FOR BONE IMAGING*

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PRIOR to the introduction of Tc^{99m} labeled phosphate compounds, F^{18} was the agent of choice for bone imaging.[2,6,11,17] F^{18} gave the highest lesion-to-normal bone differential uptake ratios with low soft tissue and blood levels and permitted diagnostic quality images to be obtained within 2–4 hours after intravenous administration.[17] The lack of widespread use of this radiopharmaceutical has been related primarily to its high cost, limited availability due to its very short half-life and the technical problems associated with imaging a moderately high energy radionuclide with conventional instrumentation.

The use of Tc^{99m} presents advantages[4,12–15] which supersede the economic and technical problems associated with F^{18}. F^{18} remains the standard imaging radiopharmaceutical with which newer agents must be compared in terms of diagnostic accuracy, tissue distribution patterns, dosimetry and target to non-target ratios. The present study was undertaken to compare one of the labeled phosphates, Tc^{99m} polyphosphate, with F^{18} in terms of these characteristics.

MATERIAL AND METHOD

RADIOPHARMACEUTICALS

The F^{18} used was cyclotron produced.[16]* It was administered as sodium fluoride in isotonic saline. Tc^{99m} labeled stannous polyphosphate was prepared from a commercially available kit containing 40 mg. of sodium polyphosphate and 1 mg. of stannous chloride.† Both radiopharmaceuticals were administered intravenously. The standard adult activity doses were 3 mc of F^{18} and 10 mc of Tc^{99m} polyphosphate. Basic decay scheme characteristics of each radionuclide are listed in Table 1.

INSTRUMENTATION

Patient imaging studies were carried out with an Ohio-Nuclear Dual Five rectilinear scanner and a Nuclear-Chicago gamma camera. A computer interfaced to the gamma camera provided for quantitative storage, analysis and display of gamma camera images. A 2×2 inch well-type NaI (Tl) scintillation crystal was used for *in vitro* specimen counting.

PATIENTS

Sixty-four patients referred for diagnostic bone scannings were included in the study. All patients presented a clinical history of malignant disease. Thirty-three patients were studied with Tc^{99m} stannous polyphosphate (Tc^{99m} PP), 26 patients with F^{18} and 5 patients received both Tc^{99m} PP and F^{18}.

TECHNIQUE

Simultaneous anterior and posterior rectilinear scans were obtained in all patients. The scan area included the axial skeleton and extended down to the distal shaft of the tibia. Scans were obtained 2 to 4 hours post-injection using 1/4 inch or 1/8 inch stepping and 5:1 minification. These scan times were selected on the basis of opti-

* Obtained from Memorial Hospital for Cancer and Allied Diseases, New York City.

† New England Nuclear Tc^{99m} Stannous Polyphosphate Kit.

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P.Q., Canada, September 25–28, 1973.

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TABLE I

RADIOPHARMACEUTICAL AND DETECTOR CHARACTERISTICS

Radionuclide	F^{18}	Tc^{99m}
Chemical form	sodium fluoride	stannous polyphosphate
Physical half-life	1.83 hr.	6 hr.
Primary γ-photon energy	511 kev.	140 kev.
Photons/100 disintegrations	194	88
Calculated NaI (Tl) photopeak detection efficiency[1]	17% (½″)	91% (½″)
	65% (2″)	98% (2″)
Collimators		
Gamma camera	410 kev. diverging (1,261 hole)	high resolution (15,000 hole)
Rectilinear scanner	5″ focal plane (31 hole)	5″ focal plane (163 hole)
geometric resolution radius	21 mm.	14 mm.

mum imaging times previously suggested.[4,17] Gamma camera images were recorded over clinically symptomatic areas and abnormal scan sites together with adjacent or contralateral control areas. These images were recorded on each patient receiving Tc^{99m} PP; a limited number of gamma camera images were recorded on 14 patients studied with F^{18}. The majority of images were stored in digital form on magnetic disk for later quantitative analysis.

Interpretation of the imaging procedures was done in the daily nuclear medicine reading session. These results were later compared to roentgenographic findings on each patient. The radiologist had the scan interpretations available for inspection at the time the roentgenograms were reviewed.

Four to 6 heparinized blood specimens were obtained from 15 patients receiving Tc^{99m} PP during the first 4 hours post-injection. Cumulative urine collections were taken on 10 of these patients for the time period extending from injection to the termination of the imaging procedures.

RESULTS

IN VITRO STUDIES

The plasma concentration curve for Tc^{99m} PP expressed in per cent dose per liter of plasma is illustrated in Figure 1. The plasma concentration curve for F^{18} in a similar clinical group of patients reported by Weber *et al.*[17] is shown in the same figure. The average Tc^{99m} PP plasma concentration was 4.9 to 2.4 per cent dose per liter over the time period of 1 to 4 hours. The reference F^{18} data show the F^{18} plasma concentration to be 2.4 to 0.8 per cent dose per liter for the same time period. An estimate of the Tc^{99m} PP erythrocyte compartment was made in 4 patients by comparing the relative activity between whole blood and plasma, making corrections for the individual hematocrits. The average of these values showed erythrocyte to plasma ratios of 0.36 at 1 hour, increasing to 0.84 at 3 to 6 hours.

The cumulative urine excretion values for 10 patients receiving Tc^{99m} PP is given in Table II. For a collection period ranging from 3 to 5 hours, measurements showed that 2.6 per cent to 43.1 per cent of the administered dose was excreted.

IN VIVO STUDIES

Significant differences were observed in the count rate levels and the over-all image quality for the 2 radiopharmaceuticals *in vivo*.

Rectilinear Scans. The typical maximum linear count rate densities and scan durations for whole body scans (1/4 inch stepping) are shown below.

			Post Injection Imaging Time
Tc^{99m} PP	85 counts/cm.	30 min.	2–4 hr.
F^{18}	40 counts/cm.	35 min.	2–3 hr.

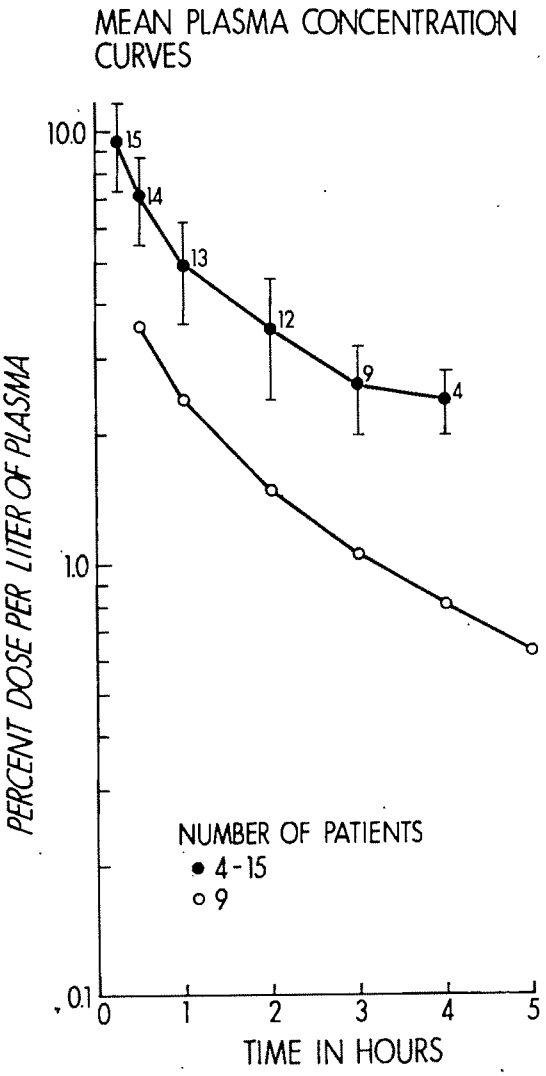


FIG. 1. Comparison of Tc^{99m} polyphosphate (upper curve) and F^{18} (lower curve, Weber *et al.*[17]) clearance from plasma.

A comparison of Tc^{99m} PP and F^{18} photoscans run on 5 patients receiving both agents demonstrated no major differences in the relative uptake distributions. However, several distinct differences in image quality were noted. Certain structures such as the ribs, clavicles and individual vertebrae, were more clearly defined on the Tc^{99m} PP scans. Visualization of the inferior angle of the scapula and better definition of the coracoid process and acromion are observed more consistently on Tc^{99m} PP scans. The use of Tc^{99m} PP also resulted in better delineation and detail of the joints. This was observed in the majority of patients at the knees, shoulders, and sternoclavicular joints. Figure 2, *A* and *B*, illustrates a typical appearance of scans on patients following the injection of Tc^{99m} PP and F^{18}.

Gamma Camera Images. The majority of gamma camera images were collected using the low-energy, high resolution collimator for Tc^{99m} and the 410 kev. diverging collimator for F^{18} (Table 1). Between 100 K and 300 K counts were collected on F^{18} and Tc^{99m} PP images. Collection times normalized to 100 K counts were as follows:

	Body Trunk	Long Bones and Joints	Imaging Time
Tc^{99m} PP	½–3 min.	2½–10 min.	2½–5 hr.
F^{18}	4–8 min.	8–16 min.	2–3 hr.

Regions of interest were flagged on camera images to provide further quantitative data on the distribution of these 2 radiopharmaceuticals. A comparison of the count rates observed over positive lesion sites to normal contralateral or control skeletal sites showed no significant differences in the range of ratios seen with Tc^{99m} PP and F^{18}. Bone to soft-tissue ratios similarly did not appear to be significantly different. (These findings are based on limited computer data on F^{18} since the longer imaging times limited the number of camera views routinely recorded.) The Tc^{99m} PP images

TABLE II

3–5 HOUR Tc^{99m} PP CUMULATIVE URINE COLLECTION ACTIVITY

Patient	Final Collection Time (hr.)	Per Cent of Injected Dose
US	2.5	43.1
ML	3	17.0
RC	3	5.4
WK	1	12.2
LD	3	26.1
MM	3	8.9
DH	4	17.2
JC	3	2.6
VH	3	7.6
LO	0.5	14.4

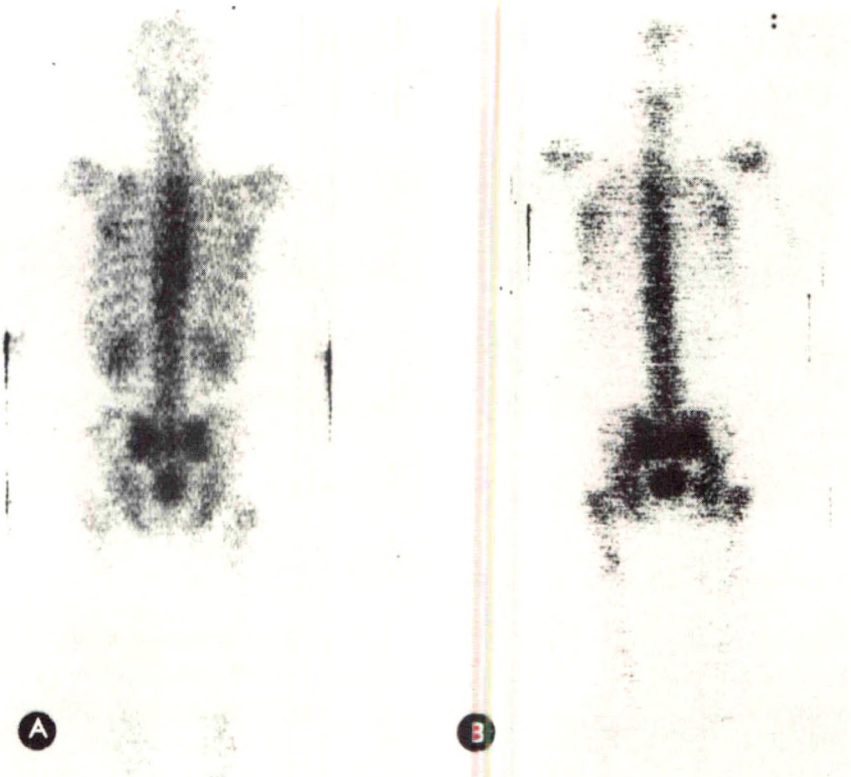


FIG. 2. (*A*) Patient PG. Example of a normal posterior Tc^{99m} PP scan ($\frac{1}{4}$ inch stepping); the patient also had a negative radiographic bone survey. Observe detail seen in the pelvis, chest and shoulder regions. The slight asymmetry in uptake over the ischium and head of the femur is due to patient positioning. (*B*) Patient SB. Example of posterior F^{18} scan ($\frac{1}{8}$ inch stepping), showing abnormal uptake in proximal left femur where a destructive lesion was seen on the radiograph. Individual bone detail is not as well resolved as observed on Tc^{99m} PP scans.

provided better detail, however, in visualizing the skeletal structures and defining the size and location of bone lesions. Figure 3, *A* and *B*, shows an example of this for a patient who received both Tc^{99m} PP and F^{18}.

Comparison Between Tc^{99m} PP Images and Roentgenograms. Table III shows a summary of the Tc^{99m} PP imaging results compared to roentgenographic findings for the 64 patients included in this study. Although the number of patients in each group is relatively small, the data suggest that Tc^{99m} PP has revealed a larger number of metastatic lesions at an earlier stage when they are not roentgenographically detectable.

Two patients had a pathologic fracture of a vertebra not accompanied by an increased uptake on bone images. Both patients had received Tc^{99m} PP. One had received radiation therapy (4,000 rads) at the site of collapse 4 months prior to the study which might have affected local vasculature. The second patient had not received any treatment to the lesion site. The appearance of 2 false negative scans with Tc^{99m} PP is not thought significant for the small patient groups studied with this radiopharmaceutical. This conclusion is based on previously documented effects of radiation therapy[3] and the comparable incidence of false negatives in earlier reported F^{18} studies.[2]

DISCUSSION

Diagnostic bone imaging requires an acceptable balance in radiopharmaceutical distribution between bone, soft-tissue and excreted radioactivity. A rapid clearance

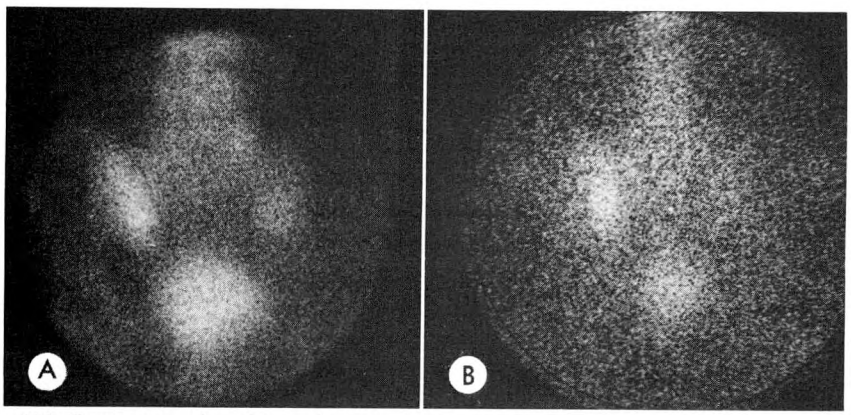


FIG. 3. Patient AN. Posteroanterior views of pelvis obtained on gamma camera following the administration of (*A*) Tc^{99m} PP (300 K counts) and (*B*) F^{18} (100 K counts). Abnormal uptake over the left sacroiliac joint is readily identified on both images. The Tc^{99m} PP image provides better detail over the pelvis and the lumbosacral region.

from blood and soft-tissue with a commensurate uptake in metabolically active bone is needed in order to take advantage of the high photon yields available from short-lived radionuclides.

Comparison of the Tc^{99m} PP blood plasma clearance rates (Fig. 1) and the average cumulative urine excretion (Table II) with earlier F^{18} studies by Weber *et al.*[17] indicates that a slower blood clearance with a comparable urine excretion is observed with Tc^{99m} PP. The Tc^{99m} PP erythrocyte activity levels also appear to be significantly higher than for F^{18}.[8] Imaging results showed that the highly variable urine excretion of Tc^{99m} PP among patients was in part due to incomplete bladder clearance. These findings suggest that a greater percentage of the administered F^{18} is available for uptake by bone and F^{18} should provide improved bone to soft-tissue ratios. Comparison of 2 earlier reports which discuss bone studies in experimental animals provide additional data to support this observation.[15,18]

Despite the more favorable F^{18} kinetics, both higher count rate levels and improved resolution of normal bone structures and bone-lesion sites were observed with Tc^{99m} PP camera and scanner imaging. These observations were felt to be the result of several additional variables; they included the

TABLE III

COMPARISON OF SCAN AND ROENTGENOGRAPHIC FINDINGS

	Normal Roentgenograms	Abnormal Roentgenograms	No Roentgenograms
Tc^{99m} PP —33 Patients			
Normal scan	6	2	9
Abnormal scan	9	6	1
F^{18} —26 Patients			
Normal scan	11	—	2
Abnormal scan	3	10	—
Tc^{99m} PP and F^{18} — 5 Patients			
Normal scan	1	—	—
Abnormal scan	—	4	—

activity, half-life and photon energy of the administered radionuclide and the type of imaging system and collimation. For example, with the activity doses used in this study, a potentially greater photon yield results from administration of the Tc^{99m} labeled preparation. Disregarding radionuclide excretion and tissue absorption of photons, it can easily be shown that the Tc^{99m} PP dose yields 2 to 5 times as many photons as the F^{18} dose between 1 and 5 hours post-injection. In addition, a much higher counting efficiency results for the lower energy photons of Tc^{99m} PP (Table 1). This count rate differential is further emphasized by the need to use thicker septa or longer collimators to obtain acceptable spatial resolution with the higher energy photons of F^{18}. With the camera, in particular, these collimator changes can result in image distortion.[7,9,10] Some loss in spatial resolution was sacrificed in this study as a result of using the 410 kev. diverging collimator.[10]

Comparable absorbed dose levels result from the prescribed administered activity doses. The absorbed radiation dose levels resulting from the Tc^{99m} PP and F^{18} bone imaging procedures were calculated using assumptions based on urine, blood and imaging data from this study in addition to *in vitro* data[17] presented earlier for F^{18}. A summary of these dose estimates follows:

Absorbed Dose in Rads

	3 mc–F^{18}	10 mc–Tc^{99m} PP
Skeleton	0.36	0.32
Bladder	0.36	0.38
Gonads	0.05	0.10

CONCLUSION

From the results obtained in this study, Tc^{99m} PP may be seen to be more advantageous than F^{18} in bone imaging. F^{18} exhibits superior metabolic properties, clearing blood and depositing on bone faster than Tc^{99m} PP. The absorbed radiation dose resulting from the administered activity levels of Tc^{99m} PP and F^{18} used in this study showed no significant differences. Tc^{99m} PP is clearly the preferred choice, however, due to the combined effects of acceptable kinetics and the low energy, high yield photons which are more compatible with conventional imaging systems. Bone images obtained with this radiopharmaceutical showed higher resolution and better counting statistics than those obtained with F^{18}. In the small number of cases reviewed for this study, the over-all improvement of image quality with Tc^{99m} PP appeared to result in earlier detection of bone lesions.

These findings in addition to the economic and practical advantages of using Tc^{99m} strongly support the use of Tc^{99m} PP in preference to F^{18} for bone imaging.

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REFERENCES

1. ANGER, H. O., and DAVIS, D. H. Gamma-ray detection efficiency and image resolution in sodium iodide. *Rev. Sc. Inst.*, 1964, *35*, 693–698.
2. BLAU, M., GANATRA, R., and BENDER, M. A. ^{18}F-fluoride for bone imaging. *Seminars Nuclear Med.*, 1972, *2*, 31–37.
3. CASTRONOVO, F. P., JR., POTSAID, M. S., and PENDERGRASS, H. P. Effects of radiation therap yon bone lesions as measured by ^{99m}Tc-diphosphonate. *J. Nuclear Med.*, 1973, *14*, 604–605.
4. CHARKES, N. D., VALENTINE, G., and CRAVITZ, B. Interpretation of normal ^{99m}Tc polyphosphate rectilinear bone scan. *Radiology*, 1973, *107*, 563–570.
5. COHEN, Y., PÉREZ, R., HENRY, R., BARDY, A., and PANNECIÈRE, C. Utilisation du pyrophosphate de sodium marqué par le technetium 99m dans la scintigraphie du squelette. *C. R. Acad. Sc. Paris*, Série D, 1972, pp. 1719–1721.
6. HARMER, C. L., BURNS, J. E., SARNS, A., and SPITTLE, M. Value of fluorine-18 for scanning bone tumors. *Clin. Radiol.*, 1969, *20*, 204–212.
7. HAYES, M. Is field size enlargement with divergent and pinhole collimators acceptable? *Radiology*, 1970, *95*, 525–528.
8. HOSKING, D. J., and CHAMBERLAIN, M. J. Studies in man with ^{18}F. *Clin. Sc.*, 1972, *42*, 153–161.
9. KRISHNAMURTHY, G. T., WALSH, C., WINSTON, M. A., WEISS, E. R., and BLAHD, W. H. Comparison of fluorine-18 bone studies obtained with rectilinear scanner and scintillation

camera equipped with high-energy diverging-hole collimator. *Radiology*, 1972, *103*, 365–369.
10. REILLY, J. A., and JOHNSON, P. M. Rapid skeletal imaging using ^{18}F and a diverging collimator. *Radiology*, 1973, *107*, 373–375.
11. ROY, R. R., NATHAN, B. E., BEALES, J. S. M., and CHISHOLM, G. D. 18 fluorine total body scans in patients with carcinoma of prostate. *Brit. J. Urol.*, 1971, *43*, 58–64.
12. SILBERSTEIN, E. B., SAENGER, E. L., TOFE, A. J., ALEXANDER, B. S., JR., and PARK, H. M. Imaging of bone metastases with ^{99m}Tc-Sn-EHDP (diphosphonate), ^{18}F, and skeletal radiography. *Radiology*, 1973, *107*, 551–555.
13. SUBRAMANIAN, G., and MCAFEE, J. G. New complex of ^{99m}Tc for skeletal imaging. *Radiology*, 1971, *99*, 192–196.
14. SUBRAMANIAN, G., MCAFEE, J. G., BELL, E. G., BLAIR, R. J., O'MARA, R. E, and RALSTON, P. H. ^{99m}Tc-labeled polyphosphate as skeletal imaging agent. *Radiology*, 1972, *102*, 701–704.
15. SUBRAMANIAN, G., MCAFEE, J. G., BLAIR, R. J., MEHTER, A., and CONNOR, T. ^{99m}Tc-EHDP: potential radiopharmaceutical for skeletal imaging. *J. Nuclear Med.*, 1972, *13*, 947–949.
16. TILBURY, R. S., DAHL, J. R., MAMACOS, J. P., and LAUGHLIN, J. S. Fluorine-18 production for medical use by helium-3 bombardment of water. *Internat. J. Appl. Radiation*, 1970, *21*, 277–281.
17. WEBER, D. A., GREENBERG, E. J., DIMICH, A., KENNY, P. J., ROTHSCHILD, E. O., MEYERS, W. P. L., and LAUGHLIN, J. S. Kinetics of radionuclides used for bone studies. *J. Nuclear Med.*, 1969, *13*, 8–17.
18. YANO, Y., MCRAE, J., VAN DYKE, D. C., and ANGER, H. G. Technetium-99m-labeled stannous ethane-1-hydroxy-1, 1-diphosphonate: new bone-scanning agent. Lawrence Berkeley Laboratory, LBL-514 Rev., August, 1972.

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Fifty-sixth Annual Meeting: Maui Surf Hotel on the Island of Maui, Hawaii, April 21–25, 1974.

EDITORIAL

CONTINUING MEDICAL EDUCATION

IMPLICATIONS CONCERNING RECERTIFICATION, RE-EXAMINATION AND RELICENSURE

CONTINUING medical education—and the documentation of it—is becoming more and more a part of life for the practicing radiologist. It is an idea, the time of which has come. The natural incentive of the dedicated physician to continue his medical education in order to provide optimal, up-to-date patient care is reinforced strongly today by a multiplicity of external pressures.

The American Academy of General Practice started it all when, in 1947, it passed a rule requiring members to engage in approved programs of continuing medical education or suffer loss of membership. This mandatory collection of "Brownie Points" was viewed with derision by some physicians, more prophetically with apprehension by others.

This apprehension was reinforced by passage of the Medicare and Medicaid Laws and the Federal program in Heart Disease, Stroke and Cancer.

Once medical care became a federally-supported human right, quality controls to protect the taxpayer were inevitable.

In 1967 (two decades after the A.A.G.P. action) the President's Commission on Medical Manpower observed that in the past "*reliance on quality controls in formal education and licensure have been the primary means of assuring that physicians use the best technique and information available. But with the increasing pace of medical advances, these 'one-time' controls are not enough; the physician's education must be continued as long as he practices.*"

Further, the Commission recommended that "*professional societies and state governments should explore the possibility of periodic relicensing of physicians and other health professionals. Relicensure should be granted upon either certification of acceptable performance in continuing education programs or upon the basis of challenge examinations in the practitioner's specialty.*"

The handwriting was on the wall.

The Department of Health, Education and Welfare in its June 1971 "Report of Licensure and Related Health Personnel Credentialing" further commented: "*The typical state requirements may provide adequate safeguards at the initial level of entry into a profession. It is a considerably less effective guarantee, however, against the growing problem of professional obsolescence.*"

Now, the PSRO provision of Public Law 92-603 (Social Security Amendments of 1972) further enforces an urgency on the medical profession to formulate its own mechanisms for peer review and surveillance of professional standards, lest the privilege of so doing be forfeited.

How is the continuing competence of practicing physicians to be established?

The American College of Radiology's Commission on Medical Services and Insurance believes that demonstration programs should be developed for continuing education and assurance of professional competence which offer both the substance of refreshed competence on the part of radiologists to patients and referring physicians *and* recognition to the radiologist for efforts devoted to continuing professional education. Further, the Commission firmly states, "It is highly desirable at this time to encourage development and trial of a

broad range of acceptable alternatives to a single, narrow 'recertification' process."

The alternative of relicensure certainly has not met with universal approbation. A *Medical Opinion* report in November, 1971 showed that, of a nationwide sampling of 933 physicians, 57 per cent deemed relicensure unnecessary, 37 per cent favored it, and 6 per cent had no opinion. (It is interesting to note that those who most strongly supported relicensure had the lowest opinion of the American Medical Association.)

The A.M.A. had, in fact, at least approached the problem in 1968 by establishing the Physicians Recognition Award. Any member of the A.M.A. who meets the requirements, has five dollars and time to fill out the form can gain this handsome certificate. Over 40,000 members have done so.

The Physicians Recognition Award requires the applicant to certify that he has acquired 150 credit hours in continuing medical education in the past three years, categorized as follows:

	Credit Hour Limit
Category 1—CME Activities with Accredited Sponsorship	No limit
Category 2—CME Activities with Non-Accredited Sponsorship	45 hours
Category 3—Medical Teaching	45 hours
Category 4—Books, Papers, Publications, Presentations, and Exhibits	40 hours
Category 5—Non-Supervised Individual CME Activities	45 hours
Category 6—Other Meritorious Learning Experience	45 hours

A total of 150 credit hours is needed to qualify for the 1972 award. At least 60 credit hours must be in Category 1.

Now, what are the incentives for individual efforts to "maintain competence" through continuing education? They include personal satisfaction, awards or other types of recognition, a safeguard against discontinuance of professional privileges, and a safeguard against discontinuance of licensure or other legal sanctions.

How could competence maintenance be achieved, or substantiated, short of re-examination—which few want? The most popular alternatives are: (1) by documentation of continuing medical education through attendance at scientific meetings, through reading of professional journals, and through participation in approved educational programs; or (2) by medical care evaluation carried out through practice observation, peer review and self-assessment tests.

The premise that continuing education improves patient care, changes habit patterns of physicians, and staves off obsolescence is accepted fairly widely. Certainly documentation of continuing education, is, for most, a more desirable method for certifying continued "competence" than re-examination. Furthermore, most physicians would prefer to establish their own requirements, rather than be subjugated to regulations handed down by an outside agency.

Perhaps in view of these pressures and trends, the Oregon Medical Association passed a rule establishing participation in continuing medical education as a requirement for membership. Others promptly followed Oregon's lead, and the state medical societies of Arizona, Florida, Kansas, Massachusetts, New Jersey and Pennsylvania have similar plans. Although differing in some details, all have the effect of requiring continuing education as a condition for membership.

The California Medical Association has developed a voluntary program that requires a high level of participation in continuing education as a qualification for the California Certificate. The state societies of Iowa and of the District of Columbia have adopted recommendations for a specific kind and amount of continuing medical education for their membership on a voluntary basis.

Two specialty societies (The American Academy of Family Physicians and the American Society of Abdominal Surgeons) require continuing medical education as a

condition for membership and at least seven more have made recommendations for voluntary participation of their membership in continuing medical education.

Members of the American Osteopathic Association must now accrue 150 hours of approved continuing medical education every three years as a condition of membership. Since one of the criteria for staff appointments in Osteopathic hospitals is membership in the A.O.A., failure to comply with the educational requirement earns potentially severe penalties.

New Mexico was the first state to unequivocally establish legal requirements of continuing education for continued licensure of physicians. (An important consideration there was recognition that state medical society regulations would not apply to the 20 per cent of New Mexico physicians who were not members, and, furthermore, suspension from the Society would not alter the individual's right to practice.) In June, 1971, New Mexico thus became the first state to reject life-long licensure. The requirements for education were patterned after the A.M.A. Physicians Recognition Award but are less restrictive in that only 120 hours of approved participation are required every three years.

Since then, the Kansas legislature has passed a law linking continuing medical education with state licensure. However, a provision of that law prevents implementation until the Board of Medical Examiners unanimously recommends it. Thus far, the Board has not so acted.

In the spring of 1973, the Maryland legislature passed a bill virtually identical with New Mexico's (but requiring 150 hours of approved C.M.E. every three years) and is implementing it.

The Illinois legislature has held hearings throughout the state with the idea of passing a similar law.

In California, a legislative approach to continuing education is in the discussion stage.

A recently passed New York State Regulation requires proof of competence (in the form of matching the educational requirements of the Academy of Family Practice) for physicians who are not *members of a hospital staff, or board certified* (emphasis mine).

State licensing boards in Arizona, Florida, Maine, Maryland, Michigan, Nevada, New Mexico, Ohio, Oklahoma, Tennessee, Vermont and West Virginia require specified amounts of continuing medical education for Osteopathic relicensure.

The Academy of Family Practice, successor to the old American Academy of General Practice, requires re-examination and recertification for its members every six years. This fine example* of social and professional responsibility is influencing other medical organizations.

On March 29 and 30, 1973, the American Board of Medical Specialties (of which the Academy of Family Practice is a member) met in Chicago. There the A.B.M.S. adopted resolutions with far-reaching implications for Diplomates of the American Board of Radiology and other specialists.

Among other actions, the A.B.M.S. adopted in principle, and urged concurrence of its member boards the policy that voluntary, periodic recertification of medical specialists become an integral part of all national medical specialty certifying programs.

It recommended that the A.B.M.S. establish a reasonable deadline by which time voluntary, periodic recertification of medical specialists will have become a standard policy and procedure of all member boards.

Furthermore, it was recommended that the A.B.M.S. consider the most appropriate manner of recognizing recertification of diplomates, including appropriate indications of such recertification in the *Directory of Medical Specialists*.

In response, The American Board of Internal Medicine will offer a Recertification Examination in General Internal Medicine to its diplomates certified in 1968 or before on October 26, 1974. The recertification

* Mark Twain said, "Few things are harder to put up with than the annoyance of a good example."

procedure, at the moment, is voluntary and is non-punitive.

The Board of Trustees of the American Society of Internal Medicine has taken a somewhat broader view of continuing competency in its December, 1973 approval of the following statement: "The diversity of activities within the broad field of internal medicine has led to a number of techniques for demonstrating accountability. ASIM believes that assessment by performance is the preferred, and the only meaningful approach to evaluating the abilities of the practicing internist. While acceptable techniques are being developed for such evaluation individual ASIM members may wish to avail themselves of other techniques: continuing medical education self-assessment and voluntary examination."

The American Board of Surgery has taken action in adopting a policy of mandatory periodic recertification of candidates certified after a given date (probably 1980) and with voluntary recertification offered to those certified before that date. The mechanism to be used to establish a candidate's evaluation for award of a certificate of recertification has not been fixed.

Dr. Claude E. Welch, President of the American College of Surgeons, in his presidential address on October 18, 1973, pointed out multiple potential pathways to recertification and recommended that any attempt to require examination as a sole method be opposed. As alternative systems he included individual participation in a variety of educational activities or a peer review system in which a surgeon's record would be considered as a basis for recertification.

Discussions at the annual meeting of the Society of Chairmen of Academic Radiology Departments in May 1973 indicated that the American Board of Radiology is considering recertification of its diplomates. The mechanism for such a recertification program has not been developed and there are no indications that precipitous action will be taken.

It is interesting to note that both the American College of Radiology and the Association of University Radiologists in their annual meetings in the spring of 1973 passed resolutions urging the American Board of Radiology to adopt documented and approved continuing medical education as the basis for recertification (as a preferable alternative to re-examination).

Further, at its annual meeting in 1973 the American College of Radiology adopted a four-point program designed to meet the needs of the individual member to insure perpetuation of his professional competency and proficiency by virtue of his efforts in continuing medical education:

I. The American Medical Association accreditation system will be used as the model for this College program since it is already acceptable to state and organizational systems in effect. The College should establish "Category 1" accreditation for its educational programs. Similarly, programs to be developed in the future should be designed to meet Category 1 accreditation requirements.

II. The ACR should assist other radiological organizations to obtain Category 1 accreditation for their appropriate educational activities. It would be expected that these organizations would reimburse the College for its activities in this service function.

III. The ACR should maintain a record of credit hours earned by individual members who participate in accredited educational programs in the various A.M.A. categories. Further, the College should furnish members with documentation of their participation in such self-improvement activities on a timely basis. The College has the capability for storage and retrieval of such information at a nominal cost to participating members.

IV. It is explicitly noted that participation of College members in this proposed program is entirely voluntary. There

is no intention at this time that such participation in any way be considered a requisite for continuing membership in the ACR. It is proposed, however, that members who meet minimum requirements in education be recognized by an appropriate certificate to be issued by the College once every three years.

Subsequently, the ACR's Committee on Continuing Evaluation of Postgraduate Education has moved forward with steps to implement this mandate.

The American College of Radiology has been accredited by the American Medical Association, so that its courses of continuing medical education can be approved for Category 1 credit. The College can in turn, through co-sponsorship, gain accreditation for meritorious educational courses offered by other radiological organizations and institutions.

The first pilot project of this nature provided for Category 1 credit to participants in the scientific sessions and refresher courses of the American Roentgen Ray Society at its annual meeting in Montreal in September, 1973.* Subsequently, the College has furnished similar accreditation of educational components of the annual meeting of the American Society of Therapeutic Radiologists, the Texas Chapter of the ACR, and the 1974 Mid-Winter Conference in Los Angeles. Plans already are made for accreditation of the American Roentgen Ray Society Meeting in San Francisco, in 1974.

Additionally, invitations have been sent to a wide variety of radiological societies and academic institutions for a cooperative co-sponsorship program of accredited continuing medical education programs in radiology.

Further, the College will embark upon a program of recording, storage and retrieval of the documented participation of its individual members in approved continuing medical education.

Timely listings of accredited courses in radiology will be provided to members.

Finally, if the American College of Radiology can act as a clearing house for most postgraduate educational offerings in our specialty, there is the potential for avoidance of some of the duplication of subject matter and overlap of dates that prevails today.

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B. G. BROGDON, M.D.
Chairman, ACR
Committee on Continuing
Evaluation of Postgraduate
Education

* Editorial: Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P.Q., Canada. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *119*, pp. 855–856.

NEWS ITEMS

INTERNATIONAL SOCIETY OF RADIOLOGY (ISR)

The new Officers of the International Society of Radiology are: *President*, J. Gómez López, M.D., Madrid, Spain; *Vice-President*, Prof. Nicola Caminha, Rio de Janeiro, Brazil; *Honorary Secretary/Treasurer*, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland; *Chairman of the International Commission on Radiological Protection* (ICRP), C. G. Stewart, M.D., Toronto, Ontario, Canada; *Chairman of the International Commission on Radiation Units and Measurements* (ICRU), Prof. H. O. Wyckoff, Ph.D., Bethesda, Maryland, USA; *Chairman of the International Commission on Stage Grouping of Cancer and Presentation of Results*, Prof. H. Anacker, M.D., München, Federal Republic of Germany; *Chairman of the International Commission on Rules and Regulations*, Prof. Arduino Ratti, M.D., Milano, Italy; and *Chairman of the International Commission on Radiological Education and Information*, Prof. Pietro Cignolini, M.D., Genova, Italy.

The 14th International Congress of Radiology will take place in 1977 in Rio de Janeiro, Brazil.

ANGIOGRAPHY OF EMERGENCIES

The Department of Radiology of Harvard Medical School will sponsor a course on Angiography 1974, "The Angiography of Emergencies." It will be held at the Sheraton-Boston Hotel, Boston, Massachusetts, October 9–12, 1974.

The chairman of the course is Herbert L. Abrams, M.D., Professor and Chairman, Department of Radiology, Harvard Medical School.

The course is designed for radiologists who participate in or intend to perform angiographic studies. As such, it will define the present state of the art with a critical look at certain technical aspects. Thereafter it will detail the application of the angiographic method to emergencies in a series of authoritative lectures and demonstrations.

Topics will include such matters as the components of the angiographic room for emergencies, angiographic technique, catheters, the role of magnification angiography, and the prevention of complications. Intrathoracic and intraabdominal emergencies which require angiographic study will be dealt with in detail, and an entire day will be spent on the gastrointestinal tract, covering the problem of gastrointestinal bleeding and of ischemic disease of the bowel. Although the course will cover established areas of angiographic examination and interpretation, it will also embody and emphasize recent advances, their potential and their limitations.

A half day seminar will be held at each of the Harvard teaching hospitals. Emergency situations will be considered. Emergency coverage, ancillary coverage, ancillary personnel, ancillary emergency drugs, and other practical problems will be dealt with.

The following distinguished Faculty will present the course: Douglas F. Adams, M.D., Associate Professor of Radiology, Harvard Medical School; S. James Adelstein, M.D., Professor of Radiology, Harvard Medical School; Stanley Baum, M.D., Professor of Radiology, Harvard Medical School; Erik Boijsen, M.D., Professor and Head, Department of Radiology, University of Lund, Sweden; Scott Boley, M.D., Professor of Surgery, Albert Einstein College of Medicine, New York; John Brooks, M.D., Professor of Surgery, Harvard Medical School; Harvey Goldman, M.D., Associate Professor of Pathology, Harvard Medical School; Florencio A. Hipona, M.D., Professor of Radiology, Harvard Medical School; Paul C. Kahn, M.D., Professor of Radiology, Tufts University

School of Medicine; Harry Z. Mellins, M.D., Professor of Radiology, Harvard Medical School; Sven Paulin, M.D., Professor of Radiology, Harvard Medical School; Joseph Rösch, M.D., Professor of Radiology, University of Oregon School of Medicine; Morris Simon, M.D., Professor of Radiology, Harvard Medical School; Jerry S. Trier, M.D., Associate Professor of Medicine, Harvard Medical School; and Manuel Viamonte, M.D., Professor and Chairman, Department of Radiology, University of Miami School of Medicine, Florida.

For further information, please contact Department of Radiology, Harvard Medical School, 25 Shattuck Street, Boston, Massachusetts 02115.

AMERICAN THERMOGRAPHIC SOCIETY

The annual meeting of the American Thermographic Society will be held at the Palmer House in Chicago, Illinois, June 22 and 23, 1974.

All sessions are open to parties interested in the applications of thermography to medicine. Although most of the members are physicians, interested engineers, scientists and paramedical personnel are invited to attend, if not participate.

Please refer all requests for abstract forms, registration forms, and any further details to: Program Committee Chairman, A. Zermeno, Ph.D., M. D. Anderson Hospital, 6723 Bertner, Houston, Texas 77025.

AMERICAN SOCIETY OF CYTOLOGY

The 22nd Annual Scientific Meeting of the "American Society of Cytology" will be held at the Statler Hilton Hotel, New York, New York, November 6–9, 1974.

For information, please contact Warren R. Lang, M.D., Secretary-Treasurer, Health Sciences Center, 130 South 9th Street, Suite 1006, Philadelphia, Pennsylvania 19107.

DEPARTMENT OF THE NAVY NAVAL UNDERSEA CENTER

The Sixth International Symposium on Acoustical Holography and Imaging will be held at San Diego, California, February 4–7, 1975.

The Abstract Deadline is September 15, 1974.

The previous symposia have been co-sponsored by the Acoustical Society of America and the IEEE. Their co-sponsorship is anticipated for this symposium.

For information please write: Dr. Newell Booth, Code 6513, Naval Undersea Center, San Diego, California 92132.

TRAINING PROGRAM IN PATHOLOGY OF CONGENITAL HEART DISEASE

A 2 to 8 week training program in congenital cardiac pathology is being offered at the Johns Hopkins University for physicians who have an interest in congenital heart disease. Physicians currently enrolled in postgraduate training programs, as well as those who have completed training, are eligible to apply.

Over 700 specimens of congenital heart disease, with correlated clinical information, are available for independent study with the assistance of the program director and the training coordinator. The collection was begun more than 25 years ago by Dr. Alfred Blalock; many of the specimens predate the development of open-heart surgery, and so are valuable for studying the natural history of congenital heart disease.

Clinical data available with the specimens include hospital and cardiac clinic records, roentgenograms, cine- and large-film angiograms, phono-, vector-, and electrocardiograms, catheterization reports, operative notes, autopsy reports, and slides showing gross or microscopic pathology. A set of reprints relating to cardiac pathology, filed by author and indexed by subject, is kept in the laboratory with the heart specimens and clinical records.

The trainees may attend conferences and rounds in the Division of Pediatric Cardiology, and also in the Departments of Pathology, Radiology, Surgery, and Medicine, depending upon their interests. They will

act as discussants or as moderators for the weekly pediatric cardiology-pathology conference, an informal meeting attended by members of the above departments.

Please direct further inquiries to Glenn C. Rosenquist, M.D., CMSC 6-124, The Johns Hopkins Hospital, Baltimore, Maryland 21205.

PATHOLOGY OF RADIATION INJURY ARMED FORCES INSTITUTE OF PATHOLOGY

This course is designed to provide the therapeutic radiologist and the pathologist with the practical knowledge necessary for an understanding of the pathogenesis of changes in normal and neoplastic tissues resulting from the actions of ionizing radiation, especially as regards those effects deriving from radiotherapeutic procedures. The course begins with a brief review of the basic principles of radiation cytopathology and histopathology and proceeds to a comprehensive consideration of specific systems responses with special emphasis upon those complications which are most frequently encountered and which may produce severe debilitation. A broad interpretation of radiation injury will be achieved through discussants from the disciplines of pathology, therapeutic radiology and radiobiology. A Course Manual will be provided each trainee and will serve as a study guideline and reference source. It will include subject information in outline form; a selected bibliography; an appendix with charts, diagrams and drawings; and a case review supplement. Both required and optional micro and lantern slide seminars are included in this course.

Applicants must be members of the Medical Corps who are board-certified, board-qualified, or otherwise exceptionally advanced in the specialty fields of radiology or pathology or other closely allied scientific disciplines wherein the acquisition of such information is particularly pertinent. Applications from qualified civilian physicians will be considered on a space available basis.

Early submission of applications is urged.

Requests for application forms should be addressed to Armed Forces Institute of Pathology, ATTN: AFIP-EDE, Washington, D. C., 20306.

GEORGE VON HEVESY PRIZE FOR NUCLEAR MEDICINE

George von Hevesy was a pioneer of Nuclear Medicine. For his studies in the field of radioactive indicator technique he received the Nobel Prize in 1943. He was one of the Founders and Honorary President of the Society of Nuclear Medicine (Europe).

At the First World Congress of Nuclear Medicine which will be held in Tokyo, September 30–October 5, 1974, under the presidency of H. Ueda, M.D., the George von Hevesy Prize for Nuclear Medicine will again be awarded. The prize amounts to Swiss Francs 10,000.

Unpublished scientific papers, preferably in English, accepted also in German and French, on the subject of Nuclear Medicine, can be submitted by authors up to the age of 40 years. The papers should be restricted to a maximum of 8 type-written pages. Upon decision of the Committee of Trustees the prize can be divided.

The manuscripts should be submitted before August 10, 1974, and be sent to Prof. Dr. W. Horst, Clinic of Nuclear Medicine, University of Zürich, Raemistrasse 100, 8006 Zürich, Switzerland.

BOOK REVIEWS

THE RADIOLOGY OF JOINT DISEASE. By D. M. Forrester, M.D., Associate Professor of Radiology, University of Southern California School of Medicine, Los Angeles County—University of Southern California Medical Center, Los Angeles, Calif.; Consultant Physician in Rheumatology, Long Beach Veterans Administration Hospital, Long Beach, Calif.; and John W. Nesson, M.D., Instructor in Radiology, University of Southern California School of Medicine, Los Angeles County—University of Southern California Medical Center, Los Angeles, Calif. Volume 2 in the Series, Saunders Monographs in Clinical Radiology. Cloth. Pp. 474, with 554 illustrations. Price, $22.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1973.

This monograph on joint disease is well written, quite informative, and contains few failings.

The most impressive asset to the book is the authors' approach to the interpretation of roentgenograms and the method used in presenting the material. The book is basically divided into 2 Parts.

Part I outlines the ABC's of Arthritis; A—Alignment, B—Bone mineralization, C—Cartilage Space, S—Soft Tissue, and is probably the most interesting portion of the whole book. By itself it demonstrates a systematic approach to the interpretation of a roentgenogram and if need be is the main selling point.

Part II which makes up the greater portion of the reading volume is organized joint by joint, or as the authors state "Arthritis from Head to Foot." However, in this section there are too many repetitive facts and sentences and a "deja vue" feeling quickly develops. The concept of a differential approach by joint is probably sound and does make for easy learning *via* repetition. Unfortunately, this results in some tedious reading toward the end of the book, although the authors' easy style compensates for this. The reproductions, which are magnificent, allow the reader to appreciate the features discussed—a rare accomplishment for most textbooks relating to bone diseases. The addition of many pathologic gross sections also supplements the reader's understanding of the many bone and joint changes existing in the various arthritides.

Despite the above criticisms, a lack of pathogenetic depth and an absence of any discussion on dysplasias, the book is definitely worthwhile reading and is highly recommended to the radiology resident, radiologist and anyone interested in the differential diagnosis of joint disease, particularly arthritis. If the reader eventually tires of rheumatoid arthritis, he can concentrate on the literary quotations which the authors offer frequently, adroitly, and without charge.

JACK G. RABINOWITZ, M.D.

COMPLICATIONS OF DIAGNOSTIC RADIOLOGY. By John F. Weigen, M.D., B.Sc., F.A.C.R., Department of Radiology, Palo Alto Medical Clinic; Clinical Assistant Professor of Radiology, Stanford University, Palo Alto, Calif.; and Sydney F. Thomas, M.D., F.A.C.R., Radiologist, Chico, Calif. Cloth. Pp. 557, with some figures. Price, $25.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

There is a group of illnesses which has increased in incidence and yet which can be lessened by every physician: iatrogenic disease. Every radiologic procedure involving the introduction of a contrast medium carries with it certain potential risks. This book is designed to help the radiologist evaluate these dangers and to anticipate, recognize and treat the complications that may occur.

The authors are to be congratulated for painstakingly integrating the extensive literature into a single, highly readable reference source of general interest. Initial chapters detail the basic and pathologic responses to intravascular contrast media and then consider prophylactic measures and the treatment of acute reactions. The sections dealing with allergy are instructive and practical. Individual roentgen studies are assessed in terms of their rates and types of complications and their recognition and treatment. These include: angiography in most of its clinical applications; excretory and retrograde urography and retroperitoneal pneumography; oral cholecystog-

raphy, and intravenous and percutaneous cholangiography; contrast studies of the gastrointestinal tract; myelography, discography, and intracranial pneumography; lymphography; bronchography, needle lung biopsy and bronchial brush biopsy; splenoportography and hepatosplenography; hysterosalpingography, pelvic pneumography, amniography and fetal transfusions. Emphasis throughout is placed on a practical understanding of the anatomic, physiologic and pathologic factors involved.

This book helps the radiologist to observe the prime injunction of clinical medicine: "Physician, do no harm."

Morton A. Meyers, M.D.

BOOKS RECEIVED

Surgical Pathology. Fifth edition. By Lauren V. Ackerman, M.D., Professor Emeritus of Surgical Pathology and Pathology, Washington University School of Medicine, St. Louis, Mo.; Professor of Pathology, State University of New York at Stony Brook, Stony Brook, Long Island, N. Y.; Consultant to the Armed Forces Institute of Pathology; and Juan Rosai, M.D., Associate Professor of Pathology, Washington University School of Medicine, St. Louis, Mo.; Associate Surgical Pathologist, Barnes and Affiliated Hospitals, St. Louis, Mo. Cloth. Pp. 1,394, with 1,329 figures. Price, $45.50. C. V. Mosby Company, 3301 Washington Boulevard, St. Louis, Mo. 63103, 1974.

Regional Pulmonary Function in Health and Disease. Edited by B. Leonard Holman, Boston, Mass.; and John F. Lindeman, St. Louis, Mo. Volume 3 in Progress in Nuclear Medicine. Cloth. Pp. 203, with 61 figures. Price, $23.50. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1973.

Microangiography of the Lung in Infancy and Childhood. By Bengt Robertson, Assistant Professor of Pathology, Karolinska Institutet, Stockholm, Sweden. Cloth. Pp. 86, with some illustrations. Proprius, Stockholm, 1973.

Radiodermites des Mains. By F.-G. Lagrot. Paper. Pp. 232, with 81 figures. Price, 120,00 F. Doin, Éditeurs, 8, Place de l'Odéon, 75006, Paris, France, 1974.

Postgraduate Education and Training in Public Health. Report of a WHO Expert Committee. World Health Organization Technical Report Series, No. 533. Paper. Pp. 68. Price, $1.75. Q Corporation, 49 Sheridan Avenue, Albany, N. Y. 12210, 1973.

Continuing Education for Physicians. Report of a WHO Expert Committee. World Health Organization Technical Report Series, No. 534. Paper. Pp. 32. Price, $1.75. Q Corporation, 49 Sheridan Avenue, Albany, N. Y. 12210, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Southampton Princess Hotel, Southampton, Bermuda, May 4–9, 1975.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Roosevelt Hotel, New Orleans, La., April 1–5, 1974.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Chicago, Ill., June 15–19, 1974.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next year. New York, N. Y., June 16–22, 1974, New York Hilton Hotel; and Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers June 22, 1974.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1974 was September 30, 1973.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

The third examination is scheduled for Saturday, Sept. 21, 1974. Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017. The deadline for receipt of completed applications is June 1, 1974.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D. C. 20010. Annual Meeting.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting: Sonesta Beach Hotel, Key Biscayne, Fla., Oct. 30–Nov. 4, 1974.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

FOURTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Rio de Janeiro, Brazil in 1977.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Meeting in June, 1975 in Edinburgh, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting: Chicago, Ill., June 22–23, 1974.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Richard H. Greenspan, Dept. of Radiol., Yale University School of Medicine, 333 Cedar St., New Haven, Conn. 06510. Twenty-second Annual Meeting, New York, N. Y., May 8–10, 1974. Dr. Norman E. Chase is in charge of arrangements, New York Medical Center, New York, N. Y. 10016.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Charles J. Masters, Maj. MC, Department of the Air Force, United States Air Force Hospital, Wiesbaden (USAFE) APO New York 09220.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Giacomo Pantaleo, 96-13 Forest Ave., Brooklyn, N. Y. 11227. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Franco, 45 Alexander St., Lockport, N. Y. 14094. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER OF ACR
Secretary-Treasurer, Dr. John L., Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alfred Brettner, 27 Hurd Circle, Auburn, N. Y. 13021. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL TEXAS RADIOLOGY SOCIETY
Secretary-Treasurer, Dr. John M. Stoebner, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

CHICAGO RADIOLOGICAL SOCIETY, DIVISION OF THE ILLINOIS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 35 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

COLORADO RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

DELAWARE CHAPTER OF ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

EAST BAY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George N. Weston, 1515 Ygnacio Valley Rd., Walnut Creek, Calif. 94598. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

GEORGIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER KANSAS CITY RADIOLOGICAL SOCIETY
President-Secretary, Dr. Menzie H. Brown, 155 S. 18th St., Kansas City, Kans. 66102.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Richard E. Litt, 6125 S.W. 31st St., Miami, Fla. 33155. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

HAWAII RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

HEALTH PHYSICS SOCIETY
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting: Houston, Texas, July 7–11, 1974.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

IDAHO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. John R. Dehner, Reid Memorial Hosp., Richmond, Ind. 47374.

IOWA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Eli A. Etscovitz, 10 Stonewood Dr., Newton Centre, Mass. 02159.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Howard L. Copas, 827 South 131st St., Omaha, Neb. 68154. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

New England Roentgen Ray Society
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02119. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting: May 10, 1974.

New England Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference, New Orleans, La., Apr. 25–27, 1974.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 S. University Dr., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 2100 Rockwood Dr., Sacramento, Calif. 95825. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 22–24, 1974.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

San Diego Radiological Society
President, Dr. Leland E. Kellerhouse, 2466 First Ave., San Diego, Calif. 92101. Meets the first Monday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Chairman, Dr. Roy G. Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broad-

way, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. 21st Annual Meeting: Town and Country Hotel, San Diego, Calif., June 10–14, 1974.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 311 Medical Arts Bldg., Chattanooga, Tenn. 37402. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada
First European Course in Chest Radiology at the Royal College of Surgeons, London, W.C.1., May 20–23, 1974.
Hon. Secretary, Dr. Ian H. Kerr, 156 Lambeth Rd., London, S.E.1.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton, St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 8952 Bellfonte Rd., Richmond, Va. 23229.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allan, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Isaiah J. Seligman, 16 Guion Pl., New Rochelle, N. Y. 10801. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Francisco Miranbell S., Apartado 3352, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1 Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32

Welbeck St., London, W1M/7PG, England. Annual General Meeting: May 15–18, 1974.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St. Catherine St. West, Montreal 107, Que., Canada. Thirty-eighth Annual Meeting.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, Lincolns Inn Fields, London WC2A 3PN, England. Annual General Meeting: May 15–18, 1974.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 p.m.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Barranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. J. Jääskeläinen, The Children's Hospital,

Stenvăckinkatu 11, SF-00290 Helsinki 29, Finland. Annual Meeting: May 29–June 1, 1974, Helsinki.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting in Uppsala, Sweden in June 1974.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

Vol. 121, No. 1

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Head

Castroviejo, I. P.: Intracranial calcifications in infancy 210

Neck and Chest

Sobredo, E. E. *et al.:* Aspergillus fumigatus: its pathogenic action on the lung 210
Cunningham, C. D. B., and Hugh, A. E.: Pneumoconiosis in women 210
Anderson, W. J. *et al.:* Chronic pancreatic pleural effusions 210

Abdomen

Coussement, A. *et al.:* Forgotten operative fields in the abdomen: radio-clinical study ... 211
Beeckman, P., and Van De Velde, E.: Considerations on the toxic megacolon 211
Scott, J. E.: Carcinoid tumors of the colon 212
Shennan, J.: Seat-belt injuries of the left colon 212
Safaie-Shirazi, S. *et al.:* Spontaneous enterobiliary fistulas 212
Light, W.: Extraction of residual biliary calculi in the x-ray department 212
Yu, S. F., and Oon, C. L.: Percutaneous transhepatic cholangiography 213
Burwood, R. J. *et al.:* Endoscopic retrograde choledocho-pancreatography: a review with a report of a collaborative series 213

Genitourinary System

Lang, E. K.: Roentgenographic assessment of asymptomatic renal lesions: an analysis of the confidence level of diagnoses established by sequential roentgenographic investigation 214
Tavernier, J. *et al.:* Renal pseudotumors 215
Cappeville, R. *et al.:* Mucosal striations of the upper urinary system: review of 58 cases 215

Nervous System

Cheynn, K. Y.: Painless myelography: introduction of a new aspiration cannula and review of 541 consecutive studies 216

Skeletal System

Brown, C. S., and Knickerbocker, W. J.: Radiologic studies in the investigation of the causes of total hip replacement failures 217
Lequesne, M., and Cassan, P.: Necrosis of the femoral head due to contusion 217
Kolár, J., and Penn, W.: Progressive post-traumatic pelvic destructions 217
Ameratunga, B.: Melorheostosis 218
Gupta, S. K., and Srivastava, T. P.: Roentgen features of skeletal involvement in small pox 218
Lachman, R. *et al.:* Radiological findings in Niemann-Pick disease 218

Blood and Lymph System

Syme, J.: Branch involvement at aortography in dissecting aneurysm, with particular reference to the lower intercostal and renal arteries 219
Putman, C. E. *et al.:* The roentgen appearance of disseminated intravascular coagulation (DIC) 219

General

Fleurquin, A. G.: Pelvic lipomatosis, report of three cases 220

ROENTGEN DIAGNOSIS

Head

CASTROVIEJO, I. PASCUAL. Calcificaciones intracraneales en la infancia. (Intracranial calcifications in infancy.) *J. belge de radiol.*, July–Aug., 1973, *56*, 319–335. (Address: Jefe del Servicio de Neurologia [Clinica Infantil], La Paz, Madrid, Spain.)

Intracranial calcifications in infants are, as a rule, pathologic. They are encountered in the following conditions:

1. *Disontogenic processes.*
   (a) Tuberous sclerosis. The calcifications are not present at birth, but are usually detectable when the child is about 12 months old; they become denser as the child ages. Frequently they are located near the foramina of Monro, or in the basal ganglia.
   (b) Sturge-Weber syndrome. The calcifications usually appear after age 1. They are located subcortically, on the same side as the facial hemangioma.
2. *Parasitic, infectious or inflammatory processes.*
   (a) Toxoplasmosis. The congenital (intrauterine) form of this parasitosis is relatively frequent in Spain. The calcifications are already present at birth, their location is variable, most frequently periventricular, or in the basal ganglia. Stenosis of the aqueduct of Sylvius is present in the majority of cases.
   (b) Cystomegalic inclusion disease. Intrauterine infection by this virus will produce microcephaly and fine granular calcifications in the walls of the ventricles.
3. *Tumoral processes.*
   (a) Craniopharyngiomas. The calcifications are intra- or suprasellar, and there is enlargement of the sella.
   (b) Ependymomas. As a rule the calcifications are large and irregular.
   (c) Teratomas and dermoid cysts. Usually the calcifications are in the midline.
4. *Vascular malformations.* They are rarely calcified in infancy.
5. *Nonpathologic calcifications.* Pineal and choroid plexus calcifications are very rare before 10 years of age.—*Julian B. Alvarez, M.D.*

Neck and Chest

SOBREDO, ENRIQUE EDUARDO, RUSSO, RODOLFO, and FELIPPONE, NESTOR. Aspergillus fumigatus: su acción patógena sobre el pulmón. (Aspergillus fumigatus: its pathogenic action on the lung.) *Rev. argent. radiol.*, Sept.–Dec., 1972, *35*, 241–250. (From: Servicio de Radiologia Central, Hospital Municipal F. J. Muñiz, Buenos Aires, Argentina.)

Aspergillus fumigatus fungus can affect the lung through various mechanisms and thus several clinicopathologic forms have been described.

Allergic aspergillosis. The presence of the fungus in the tracheobronchial tree produces a hypersensitivity response manifested by bronchial spasm, mucus plugs, fever, eosinophilia, etc. Roentgenographically one notes areas of focal atelectasis or edema. Systemic steroids might be helpful in this form (they are contraindicated in other forms).

Primary invasive form. When large numbers of fungi colonize the lung, a serious, frequently fatal, systemic process occurs. Extensive pneumonic changes are seen in the lungs, and other organs are also involved. This is a rare form.

Secondary noninvasive form. This results from the growth of the fungus within a pre-existing lung cavity. It acts as a local saprophyte without producing systemic reaction. Roentgenographically it is manifested as a mass within a cavity ("fungus ball") or by thickening of a previously thin wall cavity.

Secondary invasive form. This is a superimposed process that occurs in patients with other lung or systemic diseases. These patients have poor defenses, due to the primary disease or to the drugs used to treat it.—*Julian B. Alvarez, M.D.*

CUNNINGHAM, C. D. B., and HUGH, A. E. Pneumoconiosis in women. *Clin. Radiol.*, Oct., 1973, *24*, 491–493. (From: North Staffordshire Hospital Center, Stoke-on-Trent, England.)

Industrial lung disease is not often seen in women and may fail to be considered in a female whose chest roentgenograms shows bizarre pulmonary changes. It should be appreciated, however, that the Pottery industry in Britain does employ large numbers of female workers who may all be exposed to silica and other dusts. More recently, better ventilation and more effective local dust extraction have resulted in improved conditions; however, dust exposure may remain appreciable.

Pneumoconiosis appears to be the same disease in women as in men and the appearance of the infiltration and complications will be the same. Pulmonary mottling with fine nodulation and irregularly shaped stellate opacities may be associated with hilar lymph node calcification and eventual fibrotic and emphysematous changes.

Considerations regarding coexistent tuberculosis and carcinoma are the same as in their male counterparts.

Complications in diagnosis similarly occur in excluding coexisting bronchial infection or congestive cardiac failure.—*R. H. Troupin, M.D.*

ANDERSON, WILLIAM J., SKINNER, DAVID B., ZUIDEMA, GEORGE D., and CAMERON, JOHN L. Chronic pancreatic pleural effusions. *Surg., Gynec. & Obst.*, Nov., 1973, *137*, 827–

830. (From: Department of Surgery, The Johns Hopkins Medical Institutions, Baltimore, Md.)

Massive pleural effusions can be a manifestation of chronic pancreatitis. The massive pleural effusions are characterized by recurrence following thoracentesis as well as containing high amylase and protein levels.

The effusions are more commonly on the left side and can be bilateral. The amylase content is elevated and the protein content is usually above 3 gm./100 ml.

Chronic pancreatic effusions are thought to be secondary to a direct communication between the pleural space and a diseased pancreatic ductal system.

The diagnosis can be substantiated by discovering high amylase and protein values in pleural effusions which recur despite repeated thoracenteses. Medical management consists of repeated thoracenteses or chest tube drainage. If this is not successful after a short interval, laparotomy should be performed.

An operative pancreatogram is helpful to delineate abnormality of the ductal system and perhaps a sinus tract. Appropriate surgical dissection can then be instituted, and can be carried out in the region of the aortic and esophageal hiatus.—*John H. Harris, M.D.*

Abdomen

Coussement, A., Blanc, P., Fontaine, Y., Coussement, N., and Faure, C. Champs opératoires oubliés dans l'abdomen: étude radio-clinique. (Forgotten operative fields in the abdomen: radio-clinical study.) *Presse méd.*, Oct., 1973, *2*, 2447–2450. (Address: Dr. A. Coussement, 7, rue Mousset-Robert, 75012 Paris, France.)

The "forgotten" or "lost" foreign body in the abdominal cavity is an accident fraught with unwelcome and serious consequences to the surgeon and the patient. Every measure should be instituted to establish an early diagnosis of a retained postoperative foreign body.

The reported incidence of retained (forgotten) postoperative foreign bodies in the peritoneal cavity varies between 1/1,000 and 1/5,000. In a series of 214 cases, the foreign body was gauze and surgical pads in 165 cases, surgical instruments in 43 cases, and surgical drains in 6 cases. In another series of 307 cases, there was a 25 per cent mortality.

The presence of a foreign body may be recognized by the radiopacity of the metallic substance it contains (instruments, wire mesh in gauze), or by the late complications which may develop—abscess, fistulas, adhesions, and intestinal obstruction, or at a much later date, calcification within the mass.

The findings in 2 patients observed by the authors which present a characteristic appearance have not been previously described.

In both patients, a plain film roentgenogram of the abdomen obtained immediately after laparotomy was not remarkable. Six months later, after the development of clinical symptoms of abdominal pain, localized tenderness, and a plate-like rigidity, the plain film roentgenograms of the abdomen revealed the presence of a circumscribed mass developing at the site of the retained gauze or swab. Gas within this mass presented a characteristic nonhomogeneous turbulent or frothy pattern.

The first patient developed symptoms 6 months after cholecystectomy. There were severe vomiting and a subhepatic rigid mass.

The second patient, 6 months after cesarean section, complained of severe left flank pain. Palpation revealed a hard adherent tender mass, 8 cm. in diameter.

The plain film roentgenograms revealed the presence of a mass with the characteristic turbulent frothy pattern located, respectively, in the right upper quadrant and left flank.

At operation, a well defined adherent fetid mass was removed. This corresponded in size and location to that observed on the plain film roentgenograms. The core of the mass was a surgical swab.

The authors favor a wire mesh in each surgical gauze or swab, but doubt the necessity of routine postoperative roentgenograms on all patients undergoing a laparotomy.

There are reproductions of 4 roentgenograms.—*William H. Shehadi, M.D.*

Beeckman, P., and Van De Velde, E. Considérations sur le mégacôlon toxique. (Considerations on the toxic megacolon.) *J. belge de radiol.*, May–June, 1973, *56*, 197–207. (Address: Dr. P. Beeckman, Laboratorium voor Röntgendiagnostiek, Academisch Ziekenhuis, De Pintelaan, 135-B 9000 Gent, Belgium.)

The well known and long recognized complications of ulcerative colitis include pericolic and perirectal abscess, stenosis, perforation, arthritis and malignant degeneration. More recently, toxic megacolon has received considerable attention and is recognized as one of the more serious complications.

Toxic megacolon may present a localized or generalized dilatation, readily recognized on the plain abdominal roentgenogram. The dilatation may be mild, moderate, or pronounced; likewise the degree of general toxicity.

While commonly associated with ulcerative colitis, toxic megacolon has been described by some authors in amebic colitis, Chagas disease, typhus, cholera, and acute bacillary dysentery.

The plain film roentgenogram often reveals segmental or localized dilatation, frequently involving the transverse colon. The diameter may vary be-

tween 7 and 17 cm. Haustral markings are partially obliterated, the wall of the colon may be irregular and indistinct due to ulceration and edema of the mucosa.

The evolution and progress of the disease can be easily followed on daily abdominal roentgenograms. This examination is done without preparation of the patient and without the administration of opaque contrast material.

The authors review the literature and present 4 cases of their own. Reproductions of 11 roentgenograms accompany this article.—*William H. Shehadi, M.D.*

SCOTT, J. E. Carcinoid tumours of the colon. *Brit. J. Surg.*, Sept., 1973, *60*, 673–675. (From: Princess Margaret Hospital, Swindon, England.)

Four cases of carcinoid tumor of the colon, an extremely rare entity, are reported. These tumors do not appear to show any predilection for a particular site within the colon and the vast majority of carcinoid tumors are in the small bowel. The one striking feature in the symptoms of the 4 patients presented was the marked weight loss.

There is apparently a high incidence of a second neoplasm in association with carcinoid tumors and this may be from 29 to 47 per cent, depending on which report is referred to. None of the cases here presented with a second lesion. No hepatic metastases were found in these patients, but when carcinoid tumor does present in the colon, this is one of the most important factors in determining the prognosis.

The site of the tumor was the ascending colon in 1, the transverse colon in another, the splenic flexure in a third, and the descending colon in the last patient.

The presenting symptoms were bleeding in 1 case, and mass formation in the latter 3 cases.—*Robert P. Andrews, M.D.*

SHENNAN, JOHN. Seat-belt injuries of the left colon. *Brit. J. Surg.*, Sept., 1973, *60*, 673–675. (From: The United Liverpool Hospitals, Liverpool, England.)

Two patients are reported in whom injury to the left colon caused by lap type seat belt is described. Both patients did well following recognition of colon perforation and hemoperitoneum.

The author reviews the literature and points out that improperly worn lap type seat belts are associated with what is known as the seat-belt syndrome and that a variety of injuries may be produced by the sudden tightening of the seat belt in road accidents.

It is pointed out that seat-belt injuries of the colon are most uncommon.

The important features demonstrated by the 2 patients were initial discomfort at the site of injury, attributed to the seat belt and causing no early concern followed by a progressive increase in the severity of the clinical picture associated with bruising along the line of the seat belt in 1 case. Many reports have described a delay in presentation, following injury to either the large or small bowel from a seat belt, so that a close observation is important.

The management is discussed and 2 classifications are presented.

Both cases indicate that a loosely worn belt is particularly likely to be associated with injury and that in some situations when the belt is very loose at impact, so-called "submarining" takes place. There is liable to be an increased amount of force exerted on the soft tissues of the abdomen.

The author concludes that properly worn seat belts are not dangerous and that the incidence of serious injury and death associated with road accidents has been dramatically reduced by their use. The emphasis should be on the proper use of the seat belt itself and recognition by attending physicians of the possibility of delayed onset of colon and bowel perforation.—*Robert P. Andrews, M.D.*

SAFAIE-SHIRAZI, SIROOS, ZIKE, WILBUR L., and PRINTEN, KENNETH J. Spontaneous enterobiliary fistulas. *Surg., Gynec. & Obst.*, Nov., 1973, *137*, 769–772. (From: Department of Surgery, University of Iowa, College of Medicine, Iowa City, Iowa.)

Spontaneous enterobiliary fistulas are troublesome and, at times, result in disastrous complications of gallbladder disease or disease of the adjacent structures. Chronic cholecystitis with lithiasis is the primary etiologic factor, reportedly present in approximately 75 per cent of patients. Duodenal ulcer disease, carcinoma of the gallbladder, echinococcal cyst, and regional enteritis generally account for the remainder of the lesions.

The authors report 92 patients with enterobiliary fistulas seen during the 30 year period ending July 1970 at the University of Iowa Hospitals. Various signs and symptoms and modalities of therapy, according to the type of fistula, are described.

Many anatomic varieties have been noted: 70 patients had a cholecystoduodenal fistula; 10 a cholecystocolic fistula; 5 a cholecystogastric fistula; 4 a choledochoduodenal fistula; and 3 a cholecystocholedochal fistula.

Multiple fistulas were present in 3 patients and all were of the cholecystoduodenocolic variety.—*John H. Harris, M.D.*

LIGHT, WOLFE. Extraction of residual biliary calculi in the x-ray department. *J. Canad. A. Radiologists*, Sept., 1973, *24*, 209–214. (From: Department of Radiology, Queen Mary Veterans Hospital, 4565 Queen Mary Road, Montreal 247, P.Q., Canada.)

Not infrequently one or more calculi are left be-

hind at surgery in the common or hepatic ducts, even though they may have been demonstrated at operative cholangiography.

An instrument has been devised to crush or extract biliary calculi.

The procedure is performed entirely in the radiology department with fluoroscopic control under sterile conditions. In 13 patients, 20 stones, including 2 intrahepatic calculi, have been removed without any morbidity or complications.—*John H. Harris, M.D.*

Yu, S. F., and Oon, C. L. Percutaneous transhepatic cholangiography. *Australasian Radiol.*, Sept., 1973, *17*, 284–296. (From: Outram Road General Hospital, Singapore, and Thomson Road, General Hospital, Singapore, Singapore.)

In the clinical management of a case of jaundice a proper history, physical examination, and biochemical tests will usually resolve whether it is due to excessive hemolysis, hepatocellular dysfunction, or extrahepatic obstruction. There remains a small number of patients in whom obstructive jaundice cannot be excluded without a laparotomy. This carries a very high risk, if hepatocellular jaundice is present.

Percutaneous transhepatic cholangiography provides a relatively simple, yet readily available preoperative test for diagnosis of extrahepatic obstruction in a jaundiced patient.

The authors analyzed 95 cases in which they obtained nearly a 100 per cent success rate of puncture when the hepatic duct was more than 5 mm. in diameter. When it was 5 mm. or less in width, the success rate dropped to 50 per cent. The only serious complications are internal hemorrhage and bile peritonitis, but the risk is justified by the benefit of a successful cholangiogram. It is stated that in experienced hands a failed attempt is a reliable indication of the absence of extrahepatic obstruction.

The technique employed a P.E. 160 polyethylene tube fastened tightly to a long thin needle. A lateral approach in the mid-axillary line was preferred. This gives less chance of puncturing the gallbladder and a longer way of travel through the liver parenchyma with better chance of spontaneous closure of the tract. Entry into the bile duct is checked, not by suction to obtain bile, but by injection of a small amount of contrast medium under image intensified screening. The polyethylene tube is fixed to the skin for drainage and removed in 24 to 48 hours in cases that are not operated.

Of 85 successful cholangiographies in this series, only one mistake of consequence was made. This involved misinterpretation of an air bubble or a gland for a stone in the common bile duct.

It is cautioned that this examination should be done only where surgical facilities are available for laparotomy if needed.

A successful cholangiography may prevent the necessity for a laparotomy.—*Arch H. Hall, M.D.*

Burwood, R. J., Davies, G. T., Lawrie, B. W., Blumgart, L. H., and Salmon, P. R. Endoscopic retrograde choledocho-pancreatography: a review with a report of a collaborative series. *Clin. Radiol.*, Oct., 1973, *24*, 397–415. (From: Departments of Radiodiagnosis and Medicine, University of Bristol and the Departments of Diagnostic Radiology and Surgery, Welsh National School of Medicine.)

The authors—a group which included radiologists, a surgeon and an internist—describe their experience with 93 patients referred for endoscopic retrograde choledocho-pancreatography (ERCP) examination. Cannulation of the papilla of Vater was achieved in 72 of these patients and additional information of clinical or diagnostic value was obtained by endoscopy in 16 patients. Of the 48 patients in whom ERCP diagnosis was confirmed at subsequent surgery or follow-up, there were 30 in whom the ERCP diagnosis was different from the clinical diagnosis, and 8 patients with jaundice, in whom demonstration of a normal duct system averted laparotomy.

The authors discuss in detail the indications and contraindications to the procedure, the specifics of radiologic technique, and the radiologic findings and various forms of disease of pancreatico-biliary ducts. The original article is strongly recommended, but some highlights are presented here in abstract form.

The clinical indications are suspected biliary tract or pancreatic disease, and the examination may be performed complementary to barium studies, selected angiography and methionine scintigraphy. It is recommended that cannulation not be done in cases of acute pancreatitis until the serum amylase has been normal for 2 weeks. Esophageal stenosis, thoracic aortic aneurysm, myocardial infarction and tight pyloric stenosis are regarded as absolute contraindications. Low kilovoltage technique fluoroscopy, video recording, spot film and overhead roentgenography are utilized. Urografin 60% is slowly injected into the pancreatic duct under fluoroscopic control, the injection being halted when there is evidence of fine pancreatic duct filling. The normal duct will accommodate between 2 and 4 ml. of contrast medium. Hypaque 25% is used for the bile duct injection with the reasoning that excessive density may obscure small stones.

The authors proceed to review the literature, as normal and pathologic descriptions of pancreatic and bile ducts are offered. In 94 per cent of patients the main pancreatic duct and the common bile duct have a common channel terminating at or near the papilla of Vater. Normal diameter of the duct of Wirsung increases with age, is under 4 mm. below the age of 40 years and between 4 and 6 mm. after that age.

There is considerable anatomic variation. In 50 per cent of the patients the duct ascends toward the left upper quadrant; in 25 per cent the course of the duct is transverse; in 15 per cent there is a sigmoid loop; and in 10 per cent there is a descending path.

Carcinoma involving the pancreatic duct exhibits a spectrum of appearances varying from irregular localized stenosis through sharp and irregular major duct obstruction. Late neoplasm may be recognized by inability to perform ERCP because of the degree of neoplastic infiltration near the papilla. The ERCP appearance of chronic pancreatitis is characterized by irregular dilatations of the main duct with multiple areas of stenosis. Irregular dilatations of the finer pancreatic radicles, pseudocyst formations, and dystrophic calcifications, are also frequent features. In instances of relapsing pancreatitis, generalized opacification of the parenchyma of the gland may result from increased permeability of the epithelium.

Choledochography has proven highly accurate in providing information in patients with jaundice. A spectrum of patients with tumors, stones and sclerosing cholangitis is described.

The potential complications and hazards of ERCP are discussed, although in fact they occur very uncommonly. The flexible endoscope diminishes the risk of esophageal perforation. Adequate sedation and endoscopic aspiration of gastric secretions are supplementary safeguards against vomiting and aspiration. Potential cross transference of infectious disease remains a risk, since it is not possible to sterilize the endoscope.

The authors took particular care to exclude patients with a strong likelihood of hepatitis. The risk of acute pancreatitis is greatly diminished by fluoroscopic visualization of filling—a feature which is not possible at the time of operative pancreatography.—*R. H. Troupin, M.D.*

Genitourinary System

Lang, Erich K. Roentgenographic assessment of asymptomatic renal lesions: an analysis of the confidence level of diagnoses established by sequential roentgenographic investigation. *Radiology*, Nov., 1973, *109*, 257–269. (From: The Department of Radiology, Louisiana State University School of Medicine, Shreveport, La.)

The expanded use of screening excretory urography has resulted in the recognition of an increased number of asymptomatic renal lesions. In the author's experience, asymptomatic space-occupying lesions of the kidney were encountered in 15 per cent of all patients admitted for prostatic surgery, and in 4.6 per cent of all patients in whom excretory urograms were obtained. This becomes an incidence of 0.4 per cent in all patients admitted to the author's institution.

The management of such asymptomatic lesions is influenced by the incidence of the pathologic entity producing the space-occupying mass. Although the majority will be benign renal cysts, an incidence rate of 5.6 per cent of neoplastic lesions is a compelling reason for definitive diagnostic investigation, preferably nonsurgical, with a minimum of cost in time, money, and risk to the patient.

A protocol for the sequential investigation of asymptomatic space-occupying lesions of the kidney is presented, arranging diagnostic investigation in a specific order designed to progressively advance and affirm the diagnosis. Following discovery of the lesion, infusion nephrotomography or diagnostic ultrasound is proposed as the first screening examination. Drip infusion nephrotomography is advocated for the sorting of lesions into those that are hypovascular with cyst characteristics, and those with hypervascular or undetermined characteristics. Ultrasound is intended to identify cystic *versus* solid characteristics. Lesions with cystic or avascular characteristics are then examined by the cyst puncture and aspiration test complex, whereas lesions with indeterminate, hypervascular, or solid characteristics are examined by arteriography and adrenalin arteriography.

The necessity of a screening procedure is founded on the following facts:

(A) The cyst puncture and aspiration test complex provides criteria which will establish the definitive diagnosis of cystic lesions. Conversely, arteriography and adrenalin arteriography provide criteria best suited to establish a definitive diagnosis of neoplasm, certain inflammatory lesions, and pseudotumors. Diagnostic investigation thus proceeds with maximum expediency.

(B) Preliminary sorting will identify hypervascular and solid masses which are then examined by arteriography; thus the criticism of indiscriminate puncture of neoplasms and possible dissemination of tumor cells along the needle track is largely circumvented.

(C) The nephrotomogram, in particular, provides an important standard for comparison to the lesion as it appears on the double contrast cyst study.

The cyst puncture and aspiration complex includes roentgenographic documentation of the lesion, histochemical analysis of the aspirate and cytologic examination. Cyst puncture is carried out under fluoroscopic control with the aspirate visually assessed for color, turbidity and translucency and examined histochemically for fat, protein, and lactic acid dehydrogenase (LDH). The aspirated fluid is then replaced by an aliquot amount of contrast medium and air. Appropriate films are then obtained to demonstrate all walls of the lesion. Benign cysts yield a clear, slightly straw-colored fluid with low fat, protein and LDH content. Glucose content is

often elevated. Cystic or necrotic tumors generally yield a bloody or murky aspirate with high fat and protein content, low LDH and normal glucose. Inflammatory cysts may yield clear or murky and bloody aspirate with mildly elevated fat and protein, but the LDH content is always markedly elevated. The incidence of criteria derived from cyst puncture and aspiration test complex is presented in tabular form.

If drip infusion nephrotomography indicates neoplastic, inflammatory or indeterminate lesions, or if a solid mass is suggested on the ultrasound study, renal arteriography and adrenalin arteriography are advocated as the next examination. The arteriographic findings in various entities are discussed and presented in tabular form. Included are hypervascular and hypovascular tumors, acute inflammatory conditions, hematoma, pyelonephritis, cysts, benign renal tumors, and pseudotumors.

The diagnostic accuracy of the author's sequential protocol is both remarkable and encouraging. The roentgenographic diagnosis was correlated with the histopathologic diagnosis in 169 patients for an accuracy rate of 97 per cent in more than 96 per cent of the patients. The complication and morbidity were quite low, especially as compared with surgical exploration.

Because of the detailed information and discussion of diagnostic subtleties, reading of the original article is recommended.—*E. B. Best, M.D.*

Tavernier, J., Lemaitre, G., Michel, J. R., Padovani, J., Pinet, A., and Plainfossé, M. C. Les pseudo-tumeurs rénales. (Renal pseudotumors.) *J. de radiol., d'électrol. et de méd. nucléaire*, June–July, 1973, *54*, 457–464. (Address: Pr agr. J. Tavernier, Service de Radiologie, Hôpital des Enfants, Cours de l'Argonne, F 33000, Bordeaux, France.)

A renal pseudotumor is defined as a circumscribed hypertrophy of the normal renal parenchyma, presenting the appearance of a space-occupying lesion on urograms. These pseudotumors should be properly identified by urography, or if necessary by angiography, in order to avoid a useless exploratory lumbotomy, especially when the clinical symptoms, such as hematuria, point to a possible tumor. They may be divided into 2 main types:

(1) Pseudotumor occurring in a *normal kidney*, and resulting from:

(a) Atypical fetal lobulation, most often located on the lateral border of the left kidney, at the junction of the upper and middle third. The middle calyx is frequently elongated, with a normal cortical index.

(b) Protrusion of the upper lip of the renal hilum. The authors observed this variant in 7 of their cases. The left kidney is more often involved, with enlargement of its upper pole, more pronounced in the suprahilar region. The upper calyx is usually stretched and curved, with a lateral convexity. The suprahilar cortical index is normal or slightly increased. Arterial studies are normal or may show a prominent upper pole artery branching off more proximally from the main renal artery.

(c) Hypertrophy of Bertin's columns (focal cortical interpapillary hyperplasia). This anomaly is mostly found in association with renal duplication or with a bifid renal pelvis, and localized between the upper and middle calyces. They are seen elongated and curved on urograms. Angiography reveals a narrowing of the interlobar arteries and their compression by the cortical mass. The latter is vascularized by few arterial collaterals. The arciform arteries are normal. On the films obtained during the nephrographic phase, the hypertrophied area appears as a round or oval opacity, surrounded in one-third of the cases by a sharp radiolucent line of perisinusal fat. Scintigraphy demonstrates a normal concentration in the suspicious area. The authors have selected 14 such cases, out of which 3 were histologically corroborated. A cortical adenoma, a very rare lesion, is to be considered in the differential diagnosis as it may simulate the same angiographic pattern.

(d) Vascular anomaly. In such a case, the lower pole of the kidney, sometimes bilaterally, is greatly enlarged with an increased cortical index. Arteriography reveals ectopic or accessory polar arteries.

(e) Aberrant papilla. This produces a constant filling defect in a calyx or the renal pelvis.

(2) Pseudotumor occurring in a *diseased kidney:* regenerative nodule. The nodular formation is composed of parenchyma which is normal or modified by compensatory hypertrophy. It may be congenital, as in the case of a segmental hypoplasia, or it may be acquired. In the latter instance, it may accompany chronic pyelonephritis or it may be a sequela of a previous tumor, an infarct or a prolonged stasis. The hypertrophied zone bulges in relation to the surrounding atrophic tissue.

Urography reveals an evident mass compressing the renal pelvis or the calyces. No calyceal formation is ever present within the mass. The remainder of the kidney shows atrophy. Angiographic studies demonstrate displacement of the interlobar arteries, which appear elongated or arciform. The pseudotumor is vascularized by arteriolar collaterals. Nephrographic films show a homogeneous density within the round mass, without the radiolucent rim sometimes seen in focal cortical hyperplasia. Scintigraphy reveals normal uptake in the nodular area.

The authors have collected 6 cases of pseudotumors, 2 of which were accompanied by chronic pyelonephritis and 4 by segmental hypoplasia.—*H. P. Lévesque, M.D.*

Capdeville, R., Fortier-Beaulieu, M., Gauthier, N., and Mareschal, J. L. Les images striées du haut appareil urinaire: à propos de 58 observations. (Mucosal stria-

tions of the upper urinary system: review of 58 cases.) *J. de radiol., d'électrol. et de méd. nucléaire*, June–July, 1973, *54*, 509–513. (Address: R. Capdeville, Hôpital International de l'Université de Paris, Service de Radiologie, 42, boulevard Jourdan, F 75014, Paris, France.)

Mucosal striations of the urinary system were observed by the authors in 58 cases, comprising 43 adults and 15 children. These changes were detected on excretory urography in 54 cases, on retrograde pyelography in 3 cases, and on retrograde cystography displaying ureteral reflux in 1 case.

Radiologic appearance. These striations are composed of radiolucent lines, sometimes barely visible, sometimes thicker. The lines run parallel to the long axis of the excretory cavities: longitudinal in the ureter and infundibula, and fanlike in the pelvis. They may be restricted to a small segment of the urinary tract, or diffusely distributed. They are usually seen on 1 or 2 films of the urographic examination during the first 20 minutes. When apparent in cavities which are not too expanded and not well filled, they are rather localized. When evidenced within enlarged cavities, the involvement tends to be more extensive. On the edge of the cavities, they may produce a delicate notching due to their *en face* presentation.

Clinical significance. The importance of these mucosal folds is controversial. Some authors have thought that the simple constrictive effect of a contraction could create them and that they would be of negligible interest. Poole *et al.*, on the contrary, reported 43 cases, all corresponding to pathologic conditions, and the authors with their present series of 58 cases come to the same conclusion.

Histopathologic studies. In the series of Poole *et al.* a specimen could be obtained in 25 cases. The anatomopathologic analysis revealed submucosal edema with, at times, inflammatory cellular infiltration of the muscularis. Of this, the authors were able to obtain a specimen in 3 cases. The pathologic study gave comparable findings: thickening of the submucosa by fibrosis, inflammatory infiltration and slight edema, and enlargement of the folds with deep intervening incisurae.

Associated pathologic findings. The presence of these mucosal folds was always accompanied by urinary infection. In 43 cases various infectious agents were demonstrated, associated with ureteral reflux in 11 cases, including 9 children, and with lithiasis in 6 cases. Out of the total of 58 cases, there were 14 cases of lithiasis, 4 cases presenting hematuria, 1 case of renal trauma, 1 case of bilharziasis and 1 case of renal tumor.

Clinical evolution. In 8 cases, the abnormal folds disappeared within 6 months after the infection was controlled and after proper treatment of the associated condition. Such a short period would favor edema as the causative factor. The authors also observed the persistence of these folds for periods of between 4 months and 3½ years after treatment. Their irreversibility may be explained by the sclerous infiltration and the fibrosis.

The authors conclude that the presence of a striated mucosa should be considered abnormal, because in their 58 cases a pathologic association was established. In 18 of their cases, it was the only abnormal sign, which prompted further investigation that brought to light an infection or a ureteral reflux. —*H. P. Lévesque, M.D.*

NERVOUS SYSTEM

CHYNN, K. YORK. Painless myelography: introduction of a new aspiration cannula and review of 541 consecutive studies. *Radiology*, Nov., 1973, *109*, 361–367. (From: The Department of Radiology, St. Luke's Hospital Center and College of Physicians and Surgeons, Columbia University, New York, N. Y.)

The author uses a selected consecutive group of 54 cases out of a total of 541 cases to document the ease, practicality, and low-patient morbidity using a modified special aspiration cannula designed after the Cuatico needle set. The technique for performance of proper myelography from beginning to end is considered at some length.

The technique for removal of pantopaque is also described in some detail. The author indicates that his goals in the removal of the pantopaque are: 1. remove pantopaque with as little patient discomfort as possible; 2. recover the total volume of injected pantopaque; 3. remove as little cerebrospinal fluid as possible; and 4. perform the procedure from start to finish with greatest expediency.

The aspiration cannula is inserted through the thin walled puncture needle. There are 2 side holes in the cannula which are 0.15 mm. from the open distal end. This allows a great area of aspiration space to come in contact with the dependent pantopaque. This single modification is apparently the key point in the successful removal. This in turn prevents sucking a nerve root into the tip of the needle by the sudden vacuum effect exerted on the withdrawal attempt. (The author has used the modified cannula on 120 cases and is convinced that this technique is always successful in removal of pantopaque except in cases of blockage.)

In résumé, the results on the specific 54 patients are: 1. painless withdrawal of the contrast medium; 2. recovery time less than 10 minutes and usually less than 8 cc. of cerebrospinal fluid removed; 3. total recovery of the contrast medium except for tiny droplets in 98 per cent; and 4. retap of subarachnoid space unnecessary with uninterrupted flow of the contrast medium.—*J. P. Eberts, M.D.*

Skeletal System

Brown, C. Selby, and Knickerbocker, W. J. Radiologic studies in the investigation of the causes of total hip replacement failures. *J. Canad. A. Radiologists*, Sept., 1973, *24*, 245–253. (Address: C. Selby Brown, M.B., Macquarie Street, Sydney, N.S.W. 2000, Australia.)

The value of arthrographic examinations is discussed and a series of 20 examinations is reviewed; 16 of these patients had surgery following examination. After the preliminary anteroposterior and lateral roentgenograms, the joint space was aspirated; if no aspirate was obtained, 5–10 cc. of sterile saline was injected and withdrawn for laboratory studies. Under fluoroscopic control, 15 cc. or more of renografin 60% was injected. The joint should be manipulated and/or longitudinal traction applied, so that the contrast material can penetrate more completely.

A normal postoperative hip is illustrated as well as the roentgenograms of 13 abnormal examinations.

With a loose cement-bone junction, the contrast medium extends between the bone and the cement. There may be penetration of the shaft cortex by the stem or of the cup through the medial acetabular floor. There may be septic loosening, in which case the aspirated fluid will be turbid and there may be associated marginal bone erosion.

A pocket of contrast material projecting from the joint indicates infection particularly if the margin is irregular: this represents membrane formation. The electron microscope showed that this distinctive gray-white membrane lining the bone-cement junction and capsule, was formed from disintegrated cellular and collagenous material.

If component loosening is demonstrated with clear joint fluid, negative growth on culture and normal sedimentation rate, hip revision is indicated.

With turbid fluid, positive culture and elevated sedimentation rate, Girdlestone arthroplasty is the operation of choice.

In only 1 case was a false negative proven at surgery and there was 1 false positive result.—*Martha Mottram, M.D.*

Lequesne, M., and Cassan, P. La nécrose de la tête fémorale par contusion. (Necrosis of the femoral head due to contusion.) *Presse méd.*, Oct., 1973, *2*, 2513–2516. (Address: Dr. M. Lequesne, Service de Rhumatologie de l'Hôpital Léopold-Bellan, 7, rue du Texel, F 75014 Paris, France.)

The authors describe necrosis of the femoral head due to contusion, without the occurrence of fracture or dislocation.

They stress the importance of recognizing this condition and its implications, especially from a medico-legal point of view.

Eight cases illustrating this condition are presented.

Trauma resulting in contusion may be: (1) direct to the groin (at times the trauma may be violent)—1 case; (2) indirect, axial or cephalad transmission to the femoral head from trauma to the knee or the heel—2 cases; (3) forced movement resulting in sudden marked abduction—2 cases; and (4) lateral contusion to the greater trochanter—3 cases.

Pain in the hip may appear immediately after trauma, or may become manifest later, upon attempted ambulation following confinement or bed rest of a few days' duration.

The radiologic appearance of necrosis becomes manifest in 4 to 8 months; occasionally, after a longer lapse of time, up to 2 years.

Bone tissue obtained by biopsy shows histologic changes due to necrosis.

There were 7 males and 1 female in the authors' series. Three patients sustained the injury during work.

When immediate pain and swelling develop following trauma, aspiration of the hemarthrosis should be performed to eliminate pressure as a cause of necrosis. This is not possible, if the occurrence of pain is delayed. Weight bearing should be avoided and ambulation, with the patient gaining support by the use of 2 canes, should be encouraged.

A review of the literature by the authors fails to reveal any definite reference to contusion as a cause of necrosis which would be of definitive or indisputable medicolegal value.

Reproduction of 2 roentgenograms, 1 slide from bone tissue biopsy, and 2 tables accompany this article.—*William H. Shehadi, M.D.*

Kolár, J., and Penn, W. Progressive post-traumatic pelvic destructions. *Radiol. clin. et biol.*, 1973, *42*, 373–384. (Address: Ass. Prof. Jaromír Kolár, M.D., Radiological Clinic, Charles University, U nemocnice 2, Praha 2, Czechoslovakia.)

The authors describe 6 cases of post-traumatic osteolysis in the pelvic bones which on biopsy or autopsy were subsequently shown to represent benign slowly expanding hematomata. Considerable microscopic and roentgenologic confusion resulted in false diagnosis of tumor in all cases, and considerable loss of time was encountered in establishing the correct diagnosis.

Among the differential diagnoses which were included were chondrosarcoma, nonosteogenic fibroma, osteoclastoma, aneurysmal bone cyst, Gorham's angiomatosis, benign osteoblastoma, eosinophilic granuloma and metastatic malignancy.

The authors discuss possible differential points in the pathologic appearances, but are not able to pick

out any pathognomonic changes in the roentgenographic appearance which might enable a positive diagnosis of hematoma without biopsy. They urge that the possibility of post-traumatic osteolysis on the basis of hematoma be kept in mind when evaluating lesions of this type.—*Robert P. Andrews, M.D.*

AMERATUNGA, BERTIE. Melorheostosis. *Australasian Radiol.*, June, 1972, *16*, 148–151. (From: The General Hospital, Badulla, Ceylon.)

Leri's flowing periostitis, melorheostosis, was first described by André Leri in 1922, and only a few more than a hundred cases have been described since then. In this report 2 cases are presented, one involving the hand and the other, an extremely rare involvement of the ribs.

The etiology of the condition remains unknown. Numerous theories have been advanced including congenital, vasomotor imbalance, chronic smouldering osteomyelitis, endocrine disturbance, and subperiosteal telangiectases; however, none are fully acceptable.

The condition may be asymptomatic, but 50 per cent of cases present with pain. The hyperostosis tends to follow the distribution of a main nerve or artery. The lesions flow from the shoulder to the hand and from the hip to the foot—the outline wavy like a guttering candle. Characteristically, only one side of the bone is involved, and the disease may be monostotic or polyostotic. Associated soft tissue calcifications are common. The marrow cavity is often obliterated by the new endosteal bone, and cyst-like lesions may be present early.

Macroscopically, there is dense sclerotic eburnaceous bone, almost of tumor-like quality. Microscopically, the haversian system is usually preserved, but there is fibrotic infiltration of the trabeculae. There is no evidence of chronic inflammation. Melorheostosis has been associated with cases of scleroderma, tuberous sclerosis, osteopoikilosis, and Felty's syndrome.

The disease is slow of onset and insidious in progress with periods of remission and exacerbation. Surgical excision of eburnaceous bone usually produces relief of pain. Sympathectomy with ganglion block has also been helpful. There is no known drug therapy, and radiotherapy has no effect on the course of the disease.—*Arch H. Hall, M.D.*

GUPTA, S. K., and SRIVASTAVA, T. P. Roentgen features of skeletal involvement in small pox. *Australasian Radiol.*, June, 1973, *17*, 205–211. (From: Department of Radiology and Orthopedics, Institute of Medical Sciences, Banaras Hindu University, Varanasi-5, India.)

The authors tabulate 20 cases as regards the skeletal manifestations of small pox. There are both arthritis and osteomyelitis. Bones and joints are affected at the same time as the cutaneous signs are present. Viral inclusion bodies have been found in the sterile joint fluid and in the bone marrow.

There were 6 early cases and they all showed soft tissue swelling around the joints, osteoporosis and destruction of the adjacent bones. There was periosteal reaction often extending along the entire shaft. The epiphyseal lines were irregular and eroded. The appearance is definitely different from that seen in pyogenic osteomyelitis. The elbows are most commonly involved (75 per cent). Diagnosis at this stage is important to prevent unnecessary interference in a self-limited lesion.

In advanced cases, there were disorganized joints, fragmentation of epiphyses and irregular articular surfaces. Secondary infection causes purulent arthritis and osteomyelitis with sequestration. Flail joints and gross deformities were often present. Without secondary infection, a good range of motion is usually maintained. One case had spondylitis. Most patients had bilateral symmetric involvement, 12 out of 20, and multiple sites were involved in 15 out of 20.

Late recognition of the skeletal changes of small pox may be made years after the original infection. In a few cases, periosteal bone is resorbed and the shafts are remodeled. There may be shortening of bones, flail joints, subluxations, dislocations, gross deformities, precocious osteoarthritis and ankylosis. The appearance may superficially resemble achondroplasia or pseudohypoparathyroidism.—*Martha Mottram, M.D.*

LACHMAN, RALPH, CROCKER, ALLEN, SCHULMAN, JOSEPH, and STRAND, ROY. Radiological findings in Niemann-Pick disease. *Radiology*, Sept., 1973, *108*, 659–664. (Address: Ralph Lachman, M.D., Department of Radiology, and Department of Pediatrics, UCLA, Harbor General Hospital, 1000 West Carson Street, Torrance, Calif. 90509.)

Niemann-Pick disease is a syndrome with an inborn error of phospholipid metabolism inherited by autosomal recessive transmission. Thirty cases are grouped according to their clinical manifestations.

Twenty-eight of the cases fit into 1 of 4 clinical categories previously described by Crocker and 2 patients with an unusual phenotype are included. The roentgenographic features are reviewed and compared.

Group A (classical) patients with gross hepatosplenomegaly and onset of central nervous system (CNS) symptoms in early infancy died at the age of 1–2 years. All of the 8 patients in this group showed reticulonodular interstitial pulmonary infiltrates and 5 of the 8 showed osteoporosis and coxa valga. Long bone marrow cavity expansion with modeling defects and metacarpal widening were each seen in 1 patient.

Group B patients with heavy visceral involvement

and normal CNS could be followed for the longest period since there is no fatal outcome. These patients showed the most striking involvement in all roentgenographic aspects. All 4 of the patients showed marked reticulonodular pulmonary infiltrates. One of 4 had osteoporosis and coxa valga. Two had progressive long bone marrow cavity expansion with modeling defects and metacarpal widening. The long survival of patients in this group permits bone changes in some patients that are reminiscent of those seen in chronic Gaucher's disease, including lack of normal modeling of the distal femora.

Group C patients had a moderate course with moderate hepatosplenomegaly, onset of CNS involvement in late infancy and death at 3–7 years. Six of the 9 patients in this group had moderate reticulonodular pulmonary infiltrates, and all showed osteoporosis and coxa valga. None showed long-bone medullary cavity expansion, while 4 showed metacarpal widening. One patient was noted to have bilateral notch defects at the medial aspects of the proximal humeri.

Group D patients (Nova Scotian form) showed prominent hepatosplenomegaly and onset of CNS symptoms in early to middle childhood and death at 12–20 years. None of these patients were found to have bone or pulmonary involvement.

The atypical phenotype patients with white hair and diarrhea died at ages 5 and 8 months. Both patients showed punctate calcific deposits just inferior to the sacrum and coccyx and 1 showed similar calcifications in relation to the hips and feet.

The long bone changes are those of medullary cavity widening produced by the space-occupying effects of accumulation of Niemann-Pick cells in the marrow. Thinning of the cortices and some failure of tubulation may be seen. The etiology of metacarpal widening was not studied histologically. The coxa valga deformity is undoubtedly related to decreased weight bearing. In the 3 groups with bone abnormalities, these changes were seen on the initial examination.

The advanced pulmonary lesions are those of diffuse 2 mm. nodules associated with linear strands producing a "honeycombing" effect seen best in the lower lobes. Multiple Kerley B lines are present in the bases bilaterally. Very little progression is seen on serial roentgenograms and there are no differentiating features from other interstitial patterns.—*J. F. Bergstrom, M.D.*

Blood and Lymph System

Syme, James. Branch involvement at aortography in dissecting aneurysm, with particular reference to the lower intercostal and renal arteries. *Australasian Radiol.*, June, 1972, *16*, 140–147. (From: University of Melbourne, and The Royal Melbourne Hospital, Melbourne, Australia.)

The transfemoral catheter approach for aortography in dissecting aneurysm facilitates recognition of major branch involvement in both the thorax and abdomen. In 17 cases reviewed it was emphasized that apparent compromise of the blood supply of the kidneys and other viscera need not be associated with functional impairment. A frequently encountered pattern of involvement of the lower intercostal and renal arteries results from a tendency for most dissections to pursue a similar course.

With contrast medium injected into the true lumen, the only abnormality detected with certainty in the relatively small intercostal arteries and also in the coronaries was failure to fill. This could also occur in the large visceral abdominal or renal arteries, with often no trace of the affected vessels. Occasionally, the artery was smoothly constricted at or near its origin. A second channel, an extension of the sac, might be opacified for a varying distance along a branch.

Constriction of the origin of a branch is similar to the narrowing of the true aortic lumen. It is due to pressure from the dissection sac. A longitudinal radiolucent line in the opacified aorta or branch artery represents dissected intima and media. Normal filling of a branch at aortography from injection in the false channel indicates exclusive supply from the sac following circumferential tear which has detached the origin completely from the true aortic lumen. The maintenance of organ or visceral function in the face of contradictory aortographic findings should suggest the diagnosis of aortic dissection.

In most cases of aortic dissections the sac lies in the lower left posterior thorax. The left renal artery is more often involved than the right, and the right is seldom involved by itself.—*Arch H. Hall, M.D.*

Putman, Charles E., Minagi, Hideyo, and Blaisdell, F. William. The roentgen appearance of disseminated intravascular coagulation (DIC). *Radiology*, Oct., 1973, *109*, 13–18. (From: The Departments of Radiology and Surgery of the University of California School of Medicine at San Francisco General Hospital, San Francisco, Calif.)

Disseminated intravascular coagulation (DIC) is a phenomenon in which there is diffuse clotting and platelet aggregation in the peripheral capillary bed. It is a cause of pulmonary insufficiency which may follow severe trauma and shock, but may also occur with drug reactions, Gram-negative sepsis, transfusion with incompatible blood and the use of pump oxygenator in cardiopulmonary bypass.

The pulmonary insufficiency syndrome which follows severe shock has been termed variously as "hemorrhagic lung syndrome," "DaNang lung," "traumatic wet lung," and "shock lung." It is the result of a complex series of events following the clotting and forming of aggregates in the pulmonary

microcirculation. This causes vascular obstruction and subsequently, interstitial edema, alveolar hemorrhage, atelectasis and hyaline membrane formation. The coagulopathy causes a depletion of clotting factors which paradoxically results in a bleeding diathesis.

The authors report on 2 patients with DIC and review the pathologic changes and roentgenographic manifestations. In the early stages of DIC, there may be little in the way of roentgen findings, while histologically there may be fibrin and platelet microemboli in the pulmonary arterioles. Pathologic changes progress rapidly with venous congestion, interstitial edema, and peribronchial and perivascular hemorrhage occurring between 12–24 hours. This is followed by intraalveolar hemorrhage and edema at 24–48 hours and hemorrhagic consolidation at 48–72 hours. The roentgenographic pattern may begin as localized infiltrates followed by progressive consolidation. A picture of complete consolidation may appear all at once within 24 hours after a relatively normal chest roentgenogram.

Roentgenographically, the condition is most often misdiagnosed as pulmonary edema, which in fact, much of the pattern is due to. Other processes to consider in the post-traumatic patient with nearly identical roentgenographic patterns are: fat embolism; massive aspiration; oxygen toxicity; widespread pneumonia; and lung contusion.

If prolonged survival is accomplished, a pattern of pulmonary fibrosis may develop.

The roentgenographic pattern for the condition is nonspecific, but DIC is one of the conditions the radiologist should consider in his differential diagnosis when a pulmonary edema-like pattern is seen in a patient with severe trauma.—*J. M. Stoebner, M.D.*

GENERAL

FLEURQUIN, A. G. Pelvic lipomatosis, report of three cases. *Rev. Méd. de Córdoba, Argentina*, Jan.–June, 1973, *61*, 85–91. (From: Department of Radiology, Pontiac General Hospital, Pontiac, Mich.)

Pelvic lipomatosis is due to abnormal accumulation of fatty tissue in the pelvic cavity. It is not etiologically related to obesity, although many of these patients are overweight. It has been suggested that it is due to a hormonal disturbance.

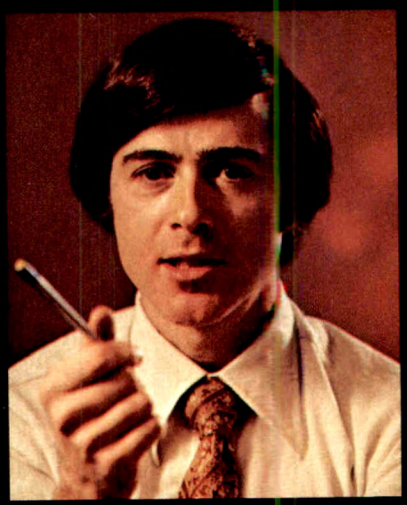
Three cases are reported, and the pertinent findings reviewed.

The roentgenographic changes, which simulate a diffuse pelvic neoplasm, are: displacement of a peculiarly shaped (inverted pear) urinary bladder; straightening of the rectosigmoid colon; displacement and/or compression of the ureters; upward displacement of the pelvic small bowel; and a characteristic increased radiolucency of the pelvic soft tissues.

Presenting symptoms include: backache; constipation; occasional rectal bleeding; bladder discomfort; difficulty in emptying the bladder; hematuria; pyuria; and low abdominal mass.

Awareness of this entity would prevent unnecessary surgery for a suspected nonexistent pelvic neoplasm, as occurred in the first case encountered in this series.—*Julian B. Alvarez, M.D.*

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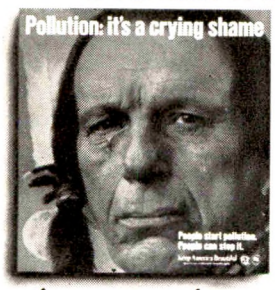
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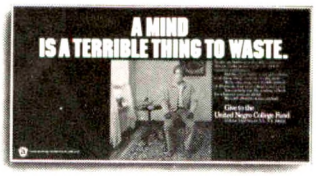




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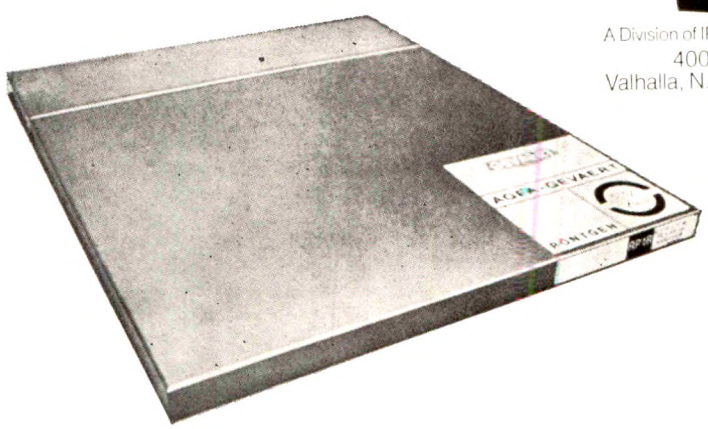
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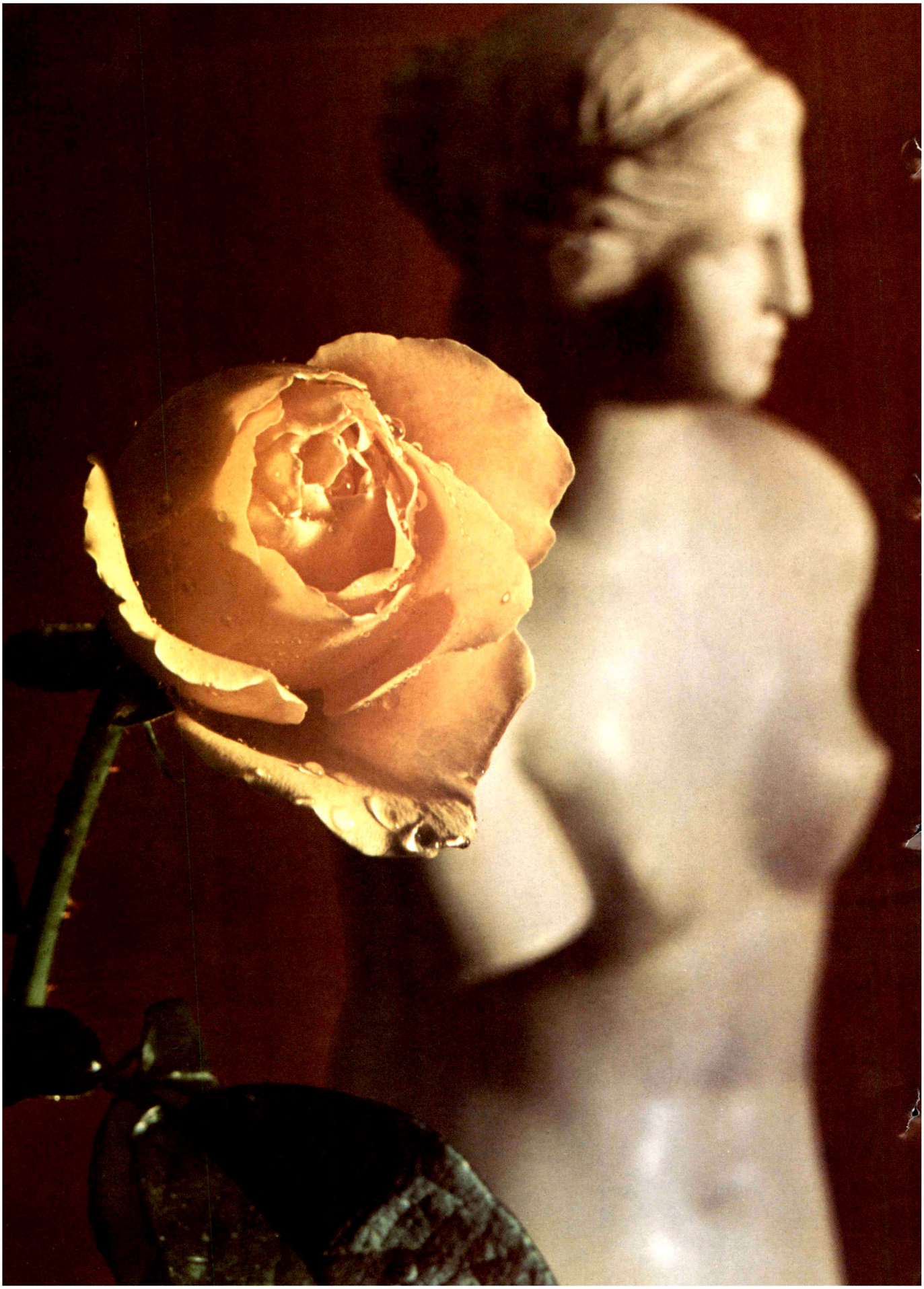
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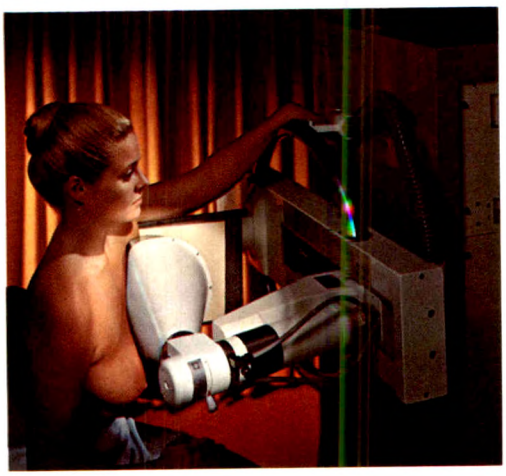
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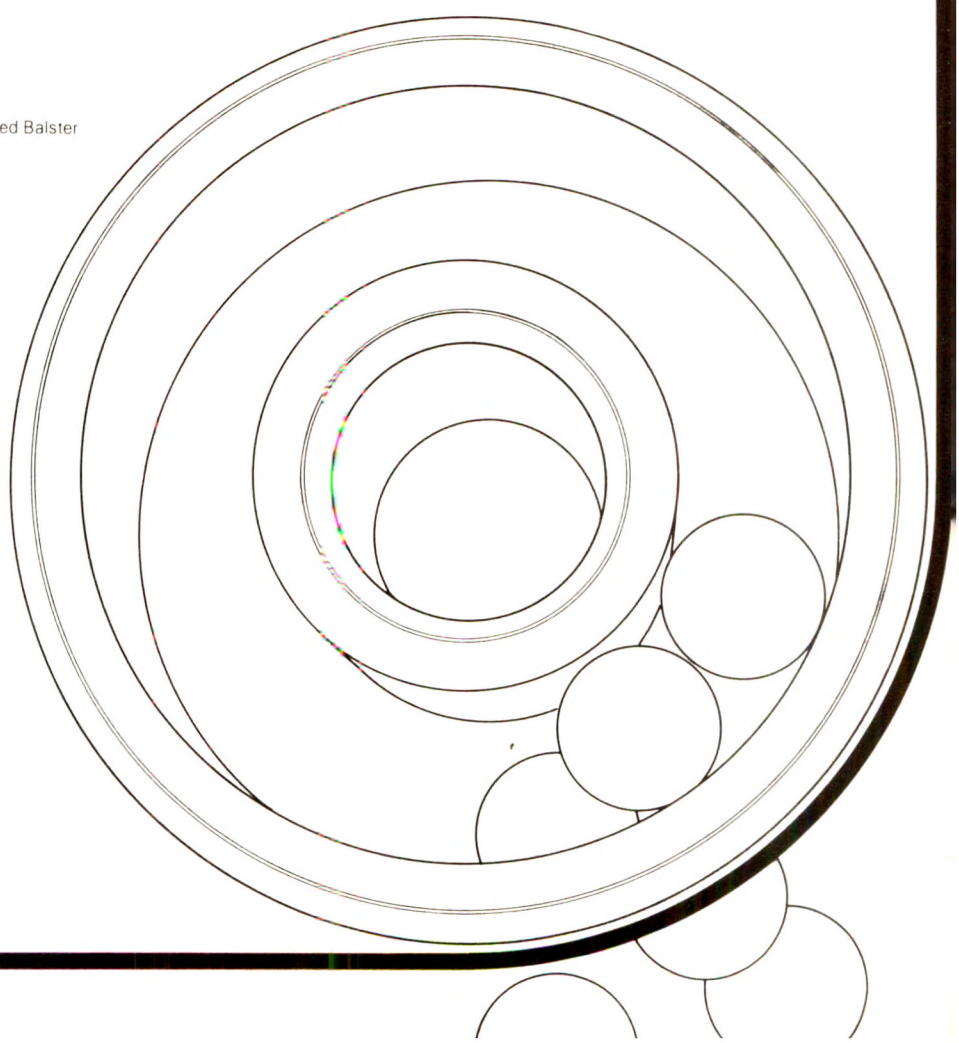
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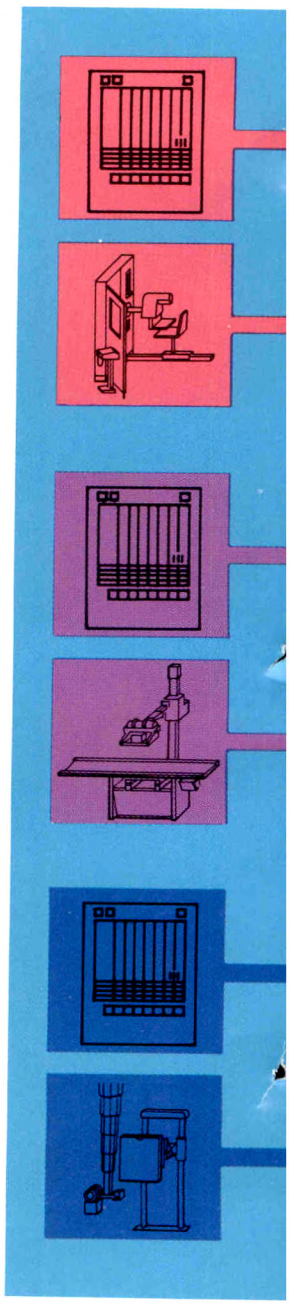


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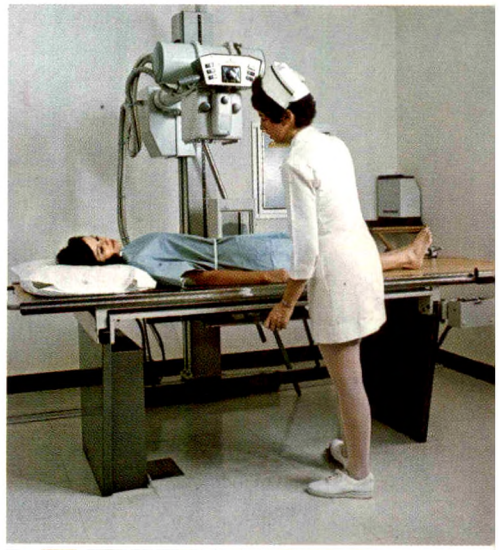
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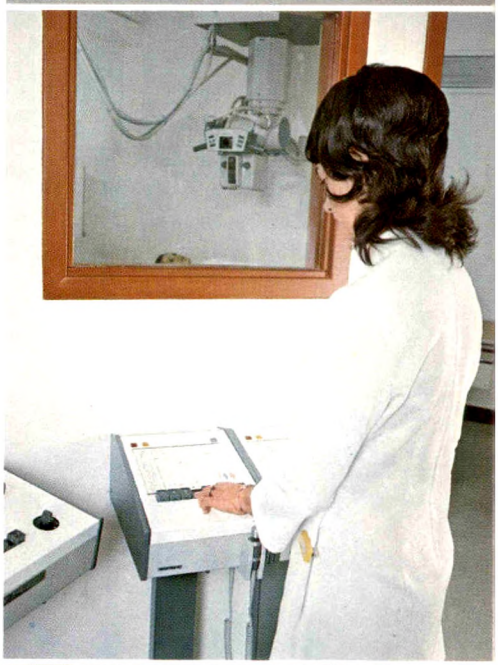
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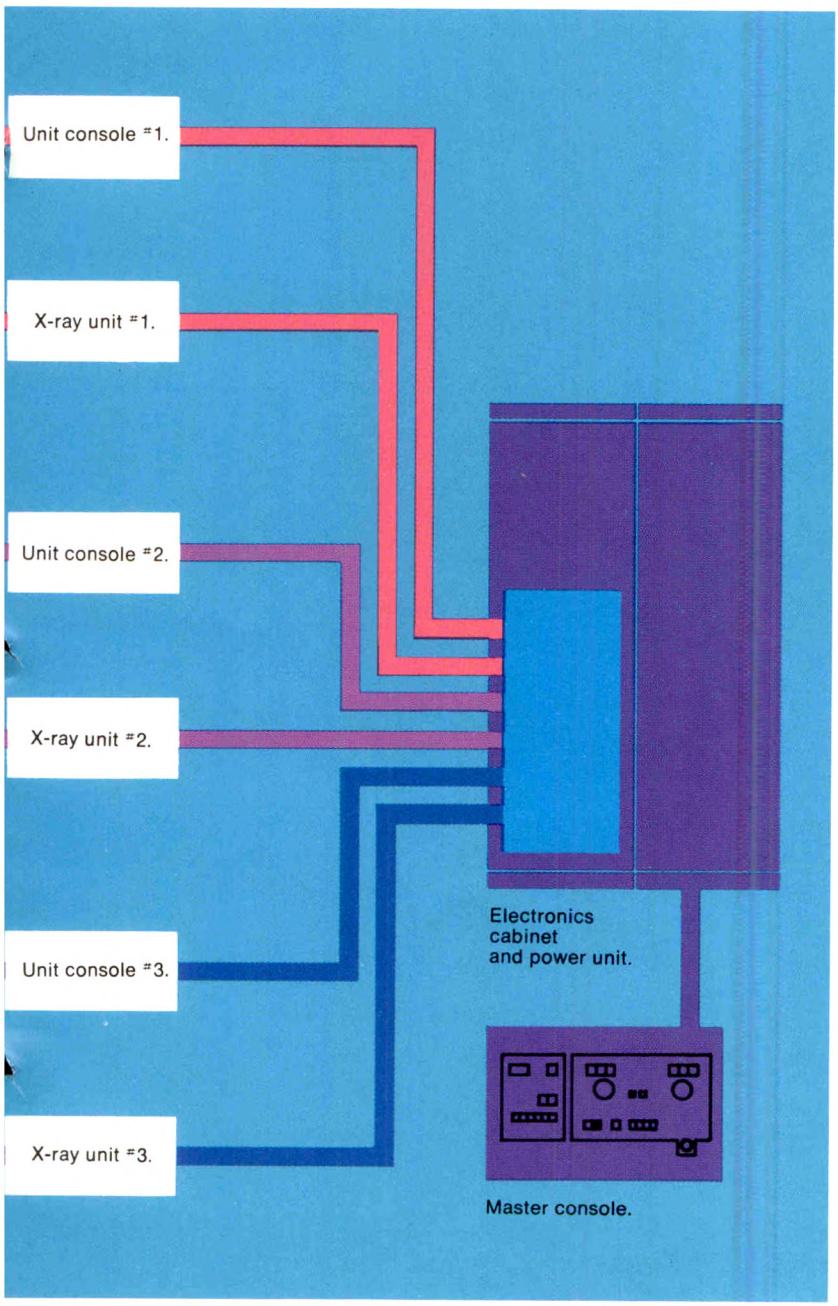
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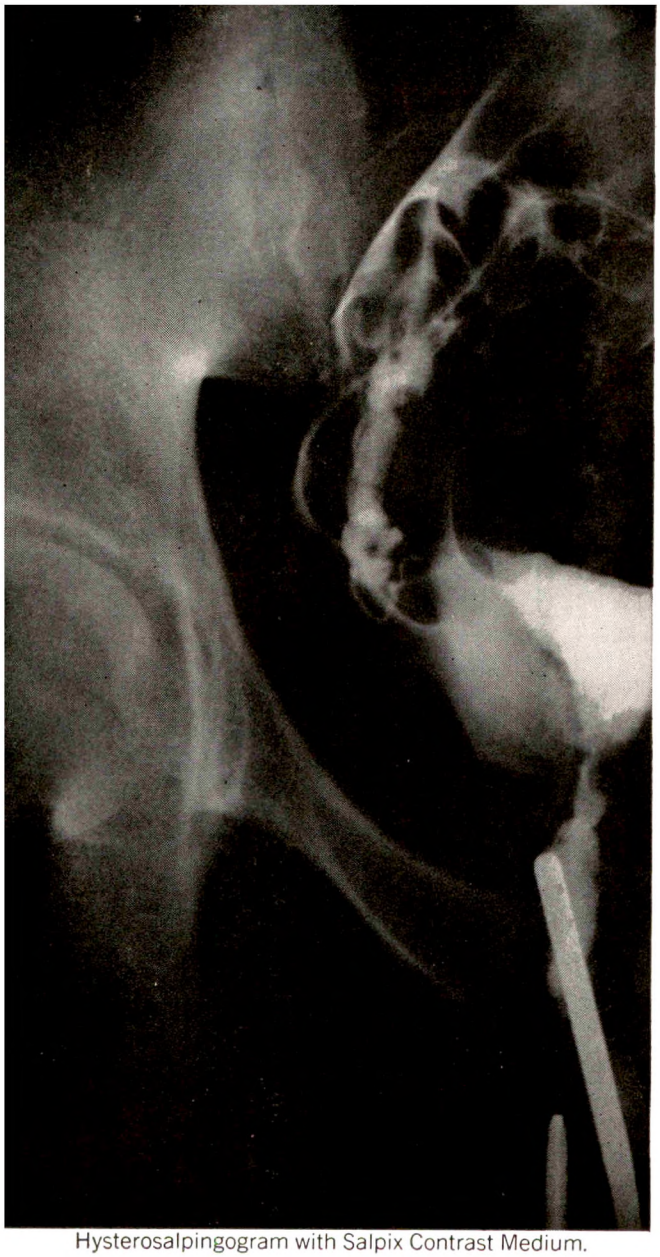
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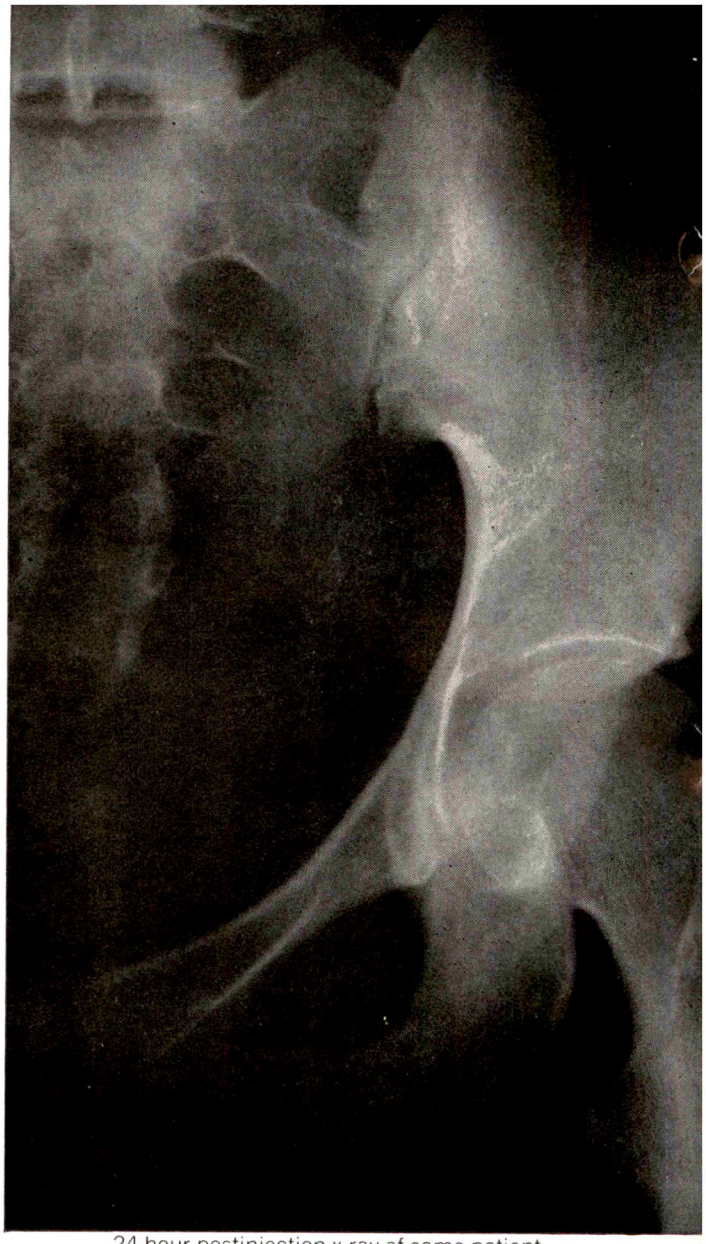





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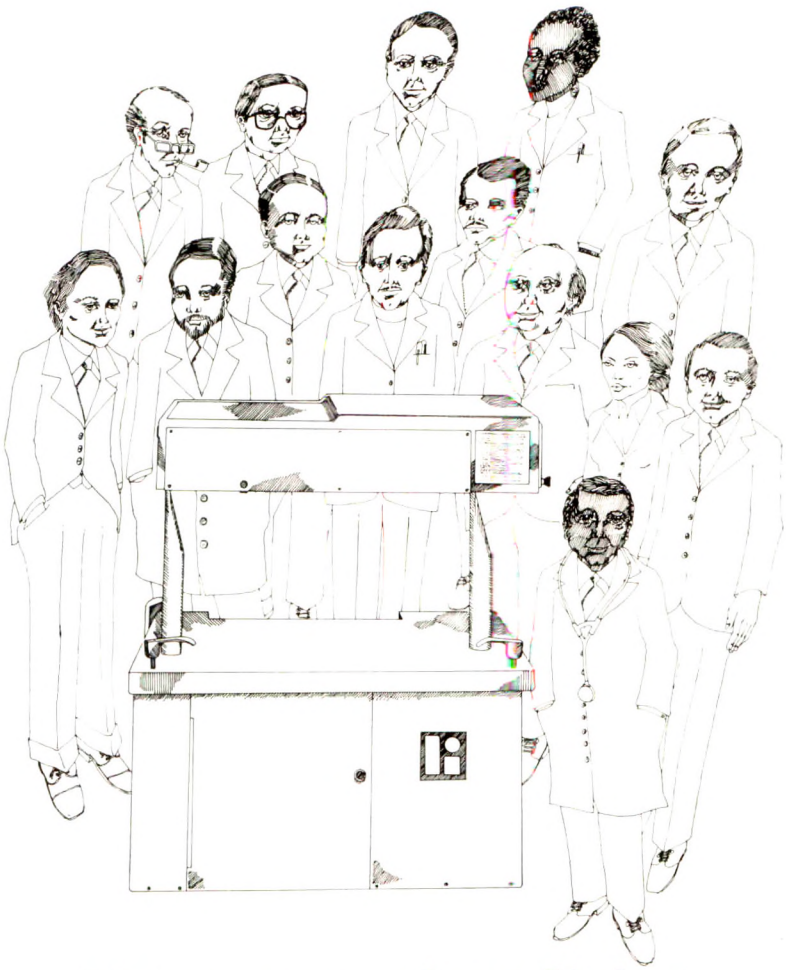
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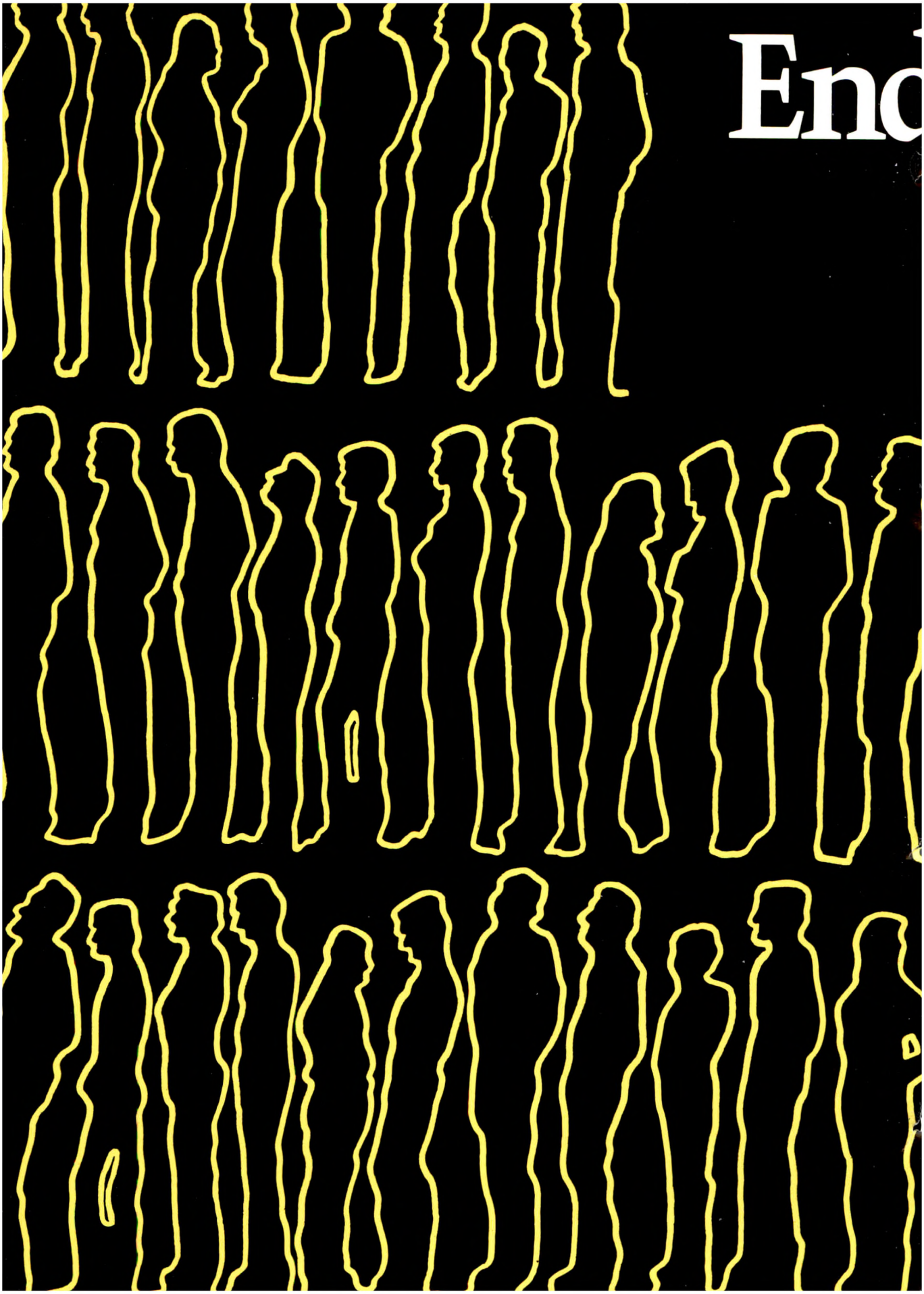
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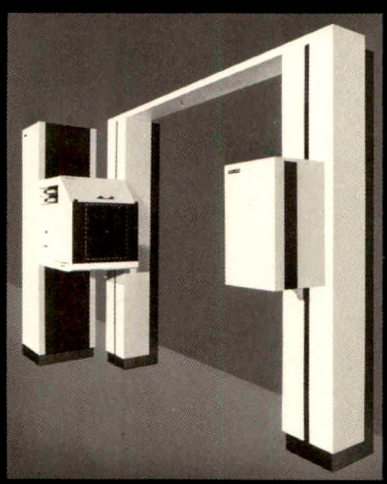
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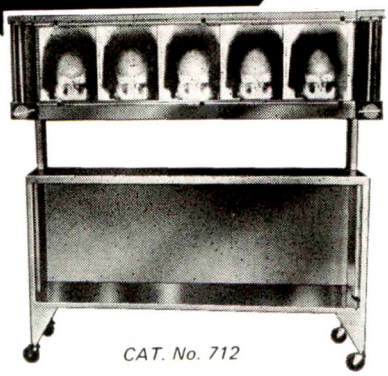
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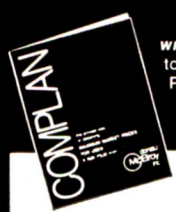
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THE SPLEEN *by* **A. I. S. Macpherson, John Richmond** *and* **A. E. Stuart**, *all of Univ. of Edinburgh.* Long accepted descriptions of the morbid anatomy and clinical features of splenic disorders are briefly described with the greater part of the book devoted to areas in which there seems to be interest, dubiety, controversy or actual advances. The histological anatomy of the spleen is examined afresh, and the splenic circulation is discussed with particular reference to electron-microscopic findings. *'73, 290 pp., 97 il. (6 in full color), 7 tables, $15.75*

FINANCIAL ADVICE FOR PHYSICIANS *by* **John N. Sheagren.** *Foreword by* **Norman F. Dacey.** Each unique financial problem of the physician is outlined. The medical student and house officer will particularly benefit from the descriptions of life insurance and the best type to purchase; other types of insurance coverages and how they represent the reserve for contingency; investments — which are best and how to get started; basic principles of estate planning and a sketch of a reasonable life-long plan for the average physician. The established physician will find much value in discussions of investment media and investing techniques; wills, trusts and the problem of probate; and taxes — death, gift and income. *'72, 132 pp., 1 table, $7.75*

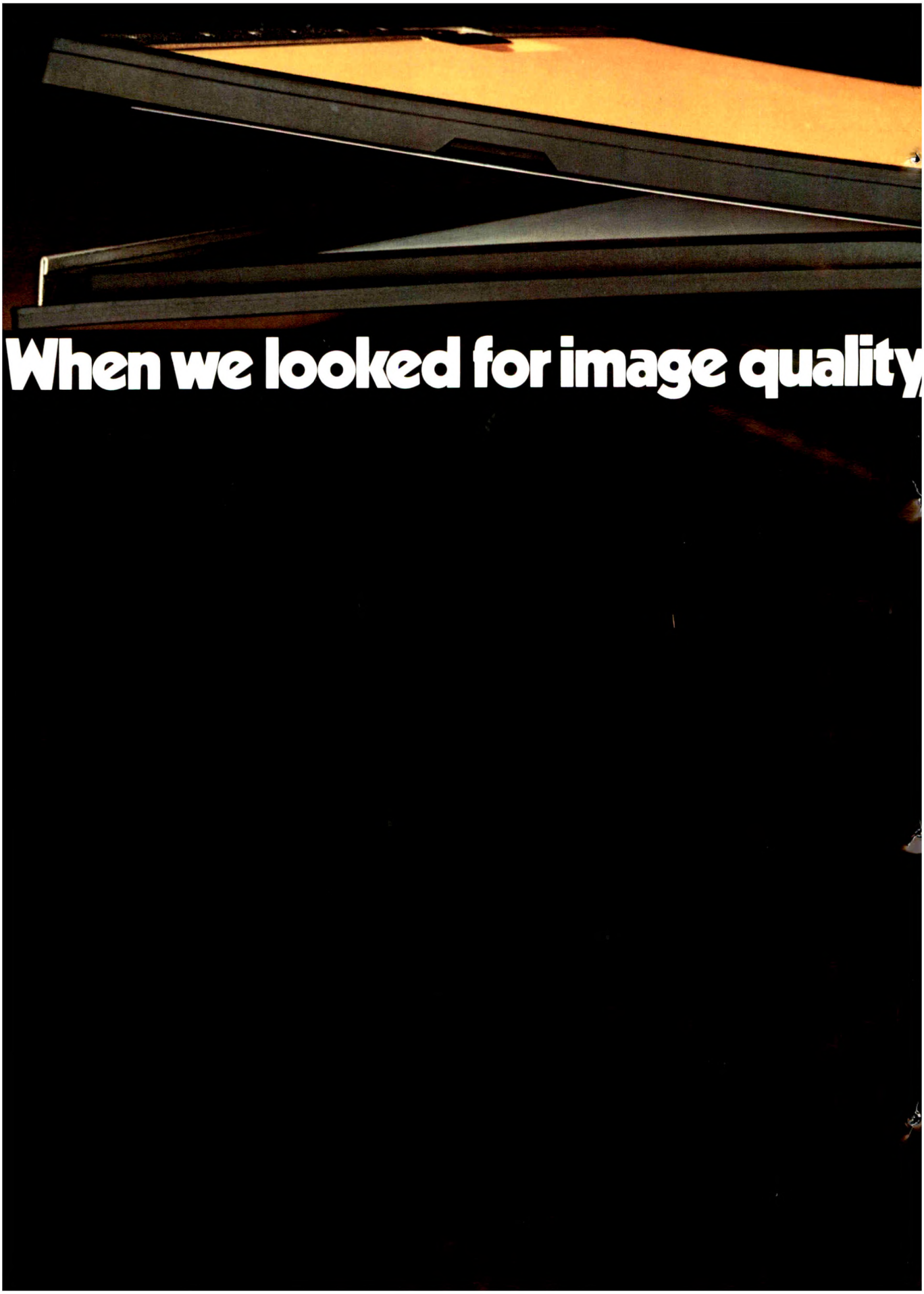
THE FUNDAMENTALS OF RADIOLOGICAL SCIENCE *by* **John Hale**, *Hospital of the Univ. of Pennsylvania, Philadelphia.* This is a textbook of basic physics for radiotherapy, clinical nuclear medicine, diagnostic radiology and radiation safety physics students. It is directed toward first-year physicians as medical doctors as well as beginning physicists and engineers in the radiological or health physics. Discussions include such topics as basic mathematics, machines for producing radiation and the radiation field, and dose kerma and exposures. *'74, about 328 pp., 172 il., 3 tables*

ROENTGEN EXAMINATIONS IN ACUTE ABDOMINAL DISEASES (3rd Ed.) *by* **J. Frimann-Dahl**, *Ulleval Hospital, Oslo, Norway.* This new, completely revised third edition contains one hundred thirty-three new black and white illustrations and two new color illustrations. Concerned principally with roentgen anatomy of the abdomen and its unusual and normal variations. The diagnosis of the acute abdominal diseases is covered including such cases as: the situation in which the actual lesion can be diagnosed on plain films only; the lesions which may be observed by repeat examinations; and emergency cases in which diagnosis must be made immediately. Acute abdominal diseases, indeed a challenge to surgeons and radiologists when they are so often encountered in these acute cases, are also included. This book is, except for Epstein's book published in 1959, the only book in English covering this field for adults. *'74, about 580 pp., 614 il. (4 in full color)*

A PRACTICAL MEDICO-LEGAL GUIDE FOR THE PHYSICIAN *by* **Everett J. Gordon**, *Georgetown Univ., Washington, D.C. Foreword by* **The Honorable William E. Stewart, Jr.** Written by a practicing physician to aid the doctor in his management of personal injury cases which involve litigation, it fills the need for a text that relates actual clinical experiences. Both physicians and attorneys will find this practical guide valuable in medico-legal practice. *'73, 360 pp., 53 il., $10.95*

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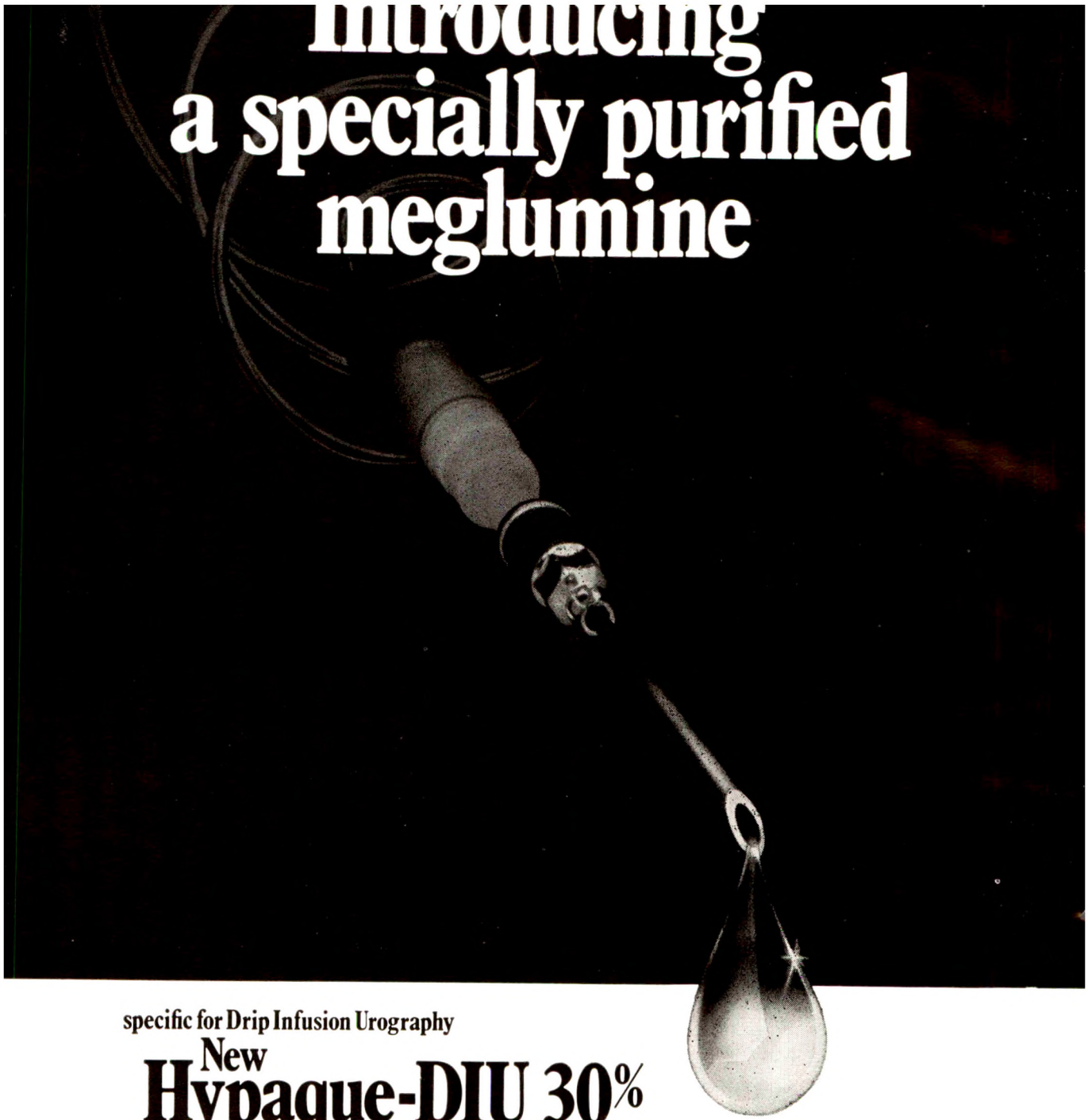
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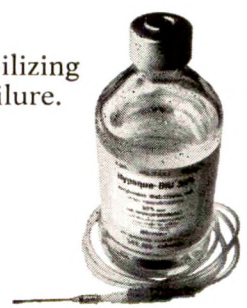
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Action: Hypaque-DIU 30 per cent is a diagnostic radiopaque medium which provides visualization of internal structures.

Indication: Hypaque-DIU 30 per cent is indicated for infusion urography.

Warnings: *Usage in Pregnancy.* No teratogenic effects attributable to a 60 per cent solution of Hypaque meglumine have been observed in reproduction studies in animals. However, before administration of Hypaque-DIU 30 per cent to women of child-bearing potential, the benefit to the patient should be carefully weighed against the possible risk to the fetus. In addition, most authorities consider elective contrast radiography of the abdomen contraindicated during pregnancy.

Serious or fatal reactions have been associated with the administration of radiopaque media. It is of utmost importance that a course of action be carefully planned in advance for the immediate treatment of serious reactions, and that adequate and appropriate facilities be readily available in case a severe reaction occurs.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravascularly.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake and direct thyroxine assays, are not affected.

Precautions: Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered. **Diphenhydramine hydrochloride (i.e., Benadryl®), however, should not be mixed with Hypaque-DIU 30 per cent since it may cause precipitation.** The intravenous injection of a test dose of 0.5 to 1 ml. of the contrast agent before infusion of the full dose has been employed in an attempt to predict severe or fatal adverse reactions. The preponderance of recent scientific literature, however, now demonstrates that this provocative test procedure is not reliably predictive of serious or fatal reactions.

Severe reactions and fatalities have occurred with the test dose alone, with the full dose after a nonreactive test dose, and with or without a history of allergy. No conclusive relationship between severe or fatal reactions and antigen-antibody reactions or other manifestations of allergy has been established. A history of allergy may be useful in predicting reactions of a mild or intermediate nature.

Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations, or of sensitivity to iodine.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure, it is essential to omit preparatory dehydration, to administer fluids, and attempt to alkalinize the urine.

Immediate adverse reactions to infusion urography have not been reported to occur at a higher frequency or with greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Infusion urography imposes a sudden osmotic load. This should be considered in patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, there is a possibility of the development of congestive heart failure hours after the procedure.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating infusion urography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion urography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

Adverse Reactions: Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

The following have been reported after administration of diatrizoates and other iodinated contrast media. *Cardiovascular reactions:* vein cramp and thrombophlebitis following intravenous injection, rare cases of cardiac arrhythmias, hypertension, hypotension and shock, and cardiac arrest. *Allergic reactions:* asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria, and rarely, anaphylactic reactions. Signs and symptoms related to the *respiratory system:* pulmonary or laryngeal edema, bronchospasm; or to the *nervous system:* restlessness, convulsions. *Other reactions:* flushing, pain, warmth, nausea, vomiting, anxiety, headache, dizziness, hematoma, and ecchymosis. Occasionally, transient proteinuria, and rarely, oliguria or anuria. Rarely, immediate or delayed rigors can occur, sometimes accompanied by hyperpyrexia. Infrequently, "iodism" (salivary gland swelling) from organic iodinated compounds appears two days after exposure and subsides by the sixth day.

Dosage and Administration: The recommended dose for infusion urography is calculated on the basis of 2 ml. of Hypaque-DIU 30 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimal results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

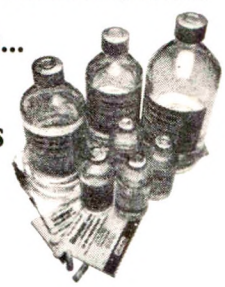
The solution is infused over a period of three to ten minutes. Radiographs are usually taken at 10, 20, and 30 minutes from the beginning of the infusion. Earlier films may be obtained for the evaluation of hypertension. Nephrograms are usually obtained at the end of the infusion, and voiding cystourethrograms are usually obtained 30 minutes after the beginning of the infusion.

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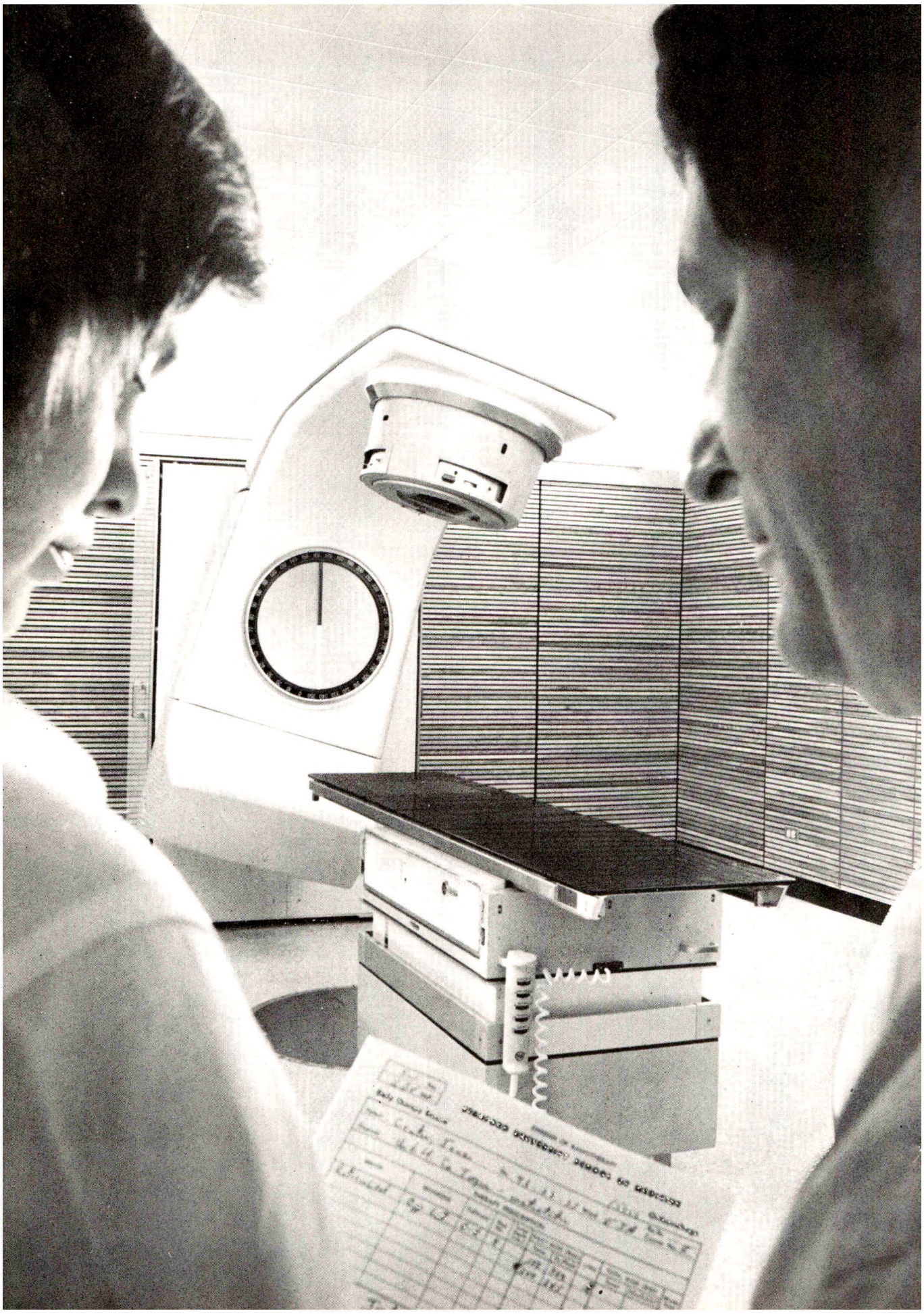
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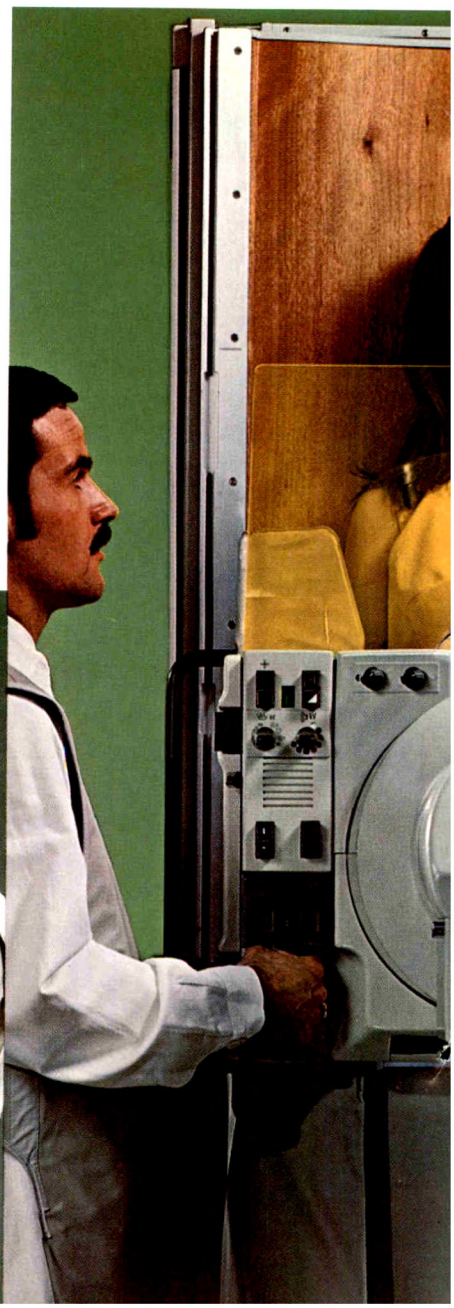
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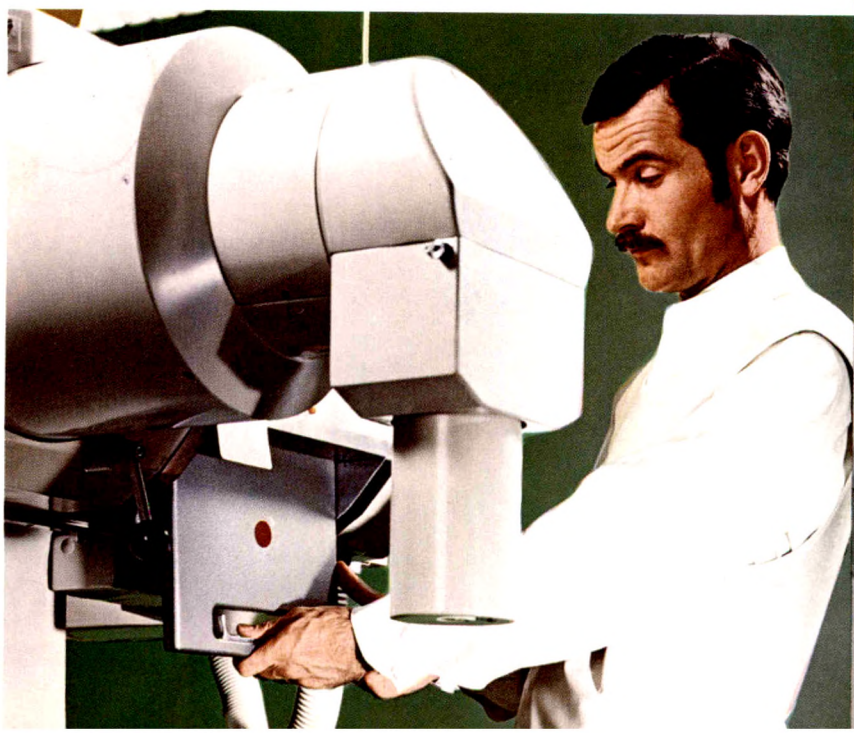
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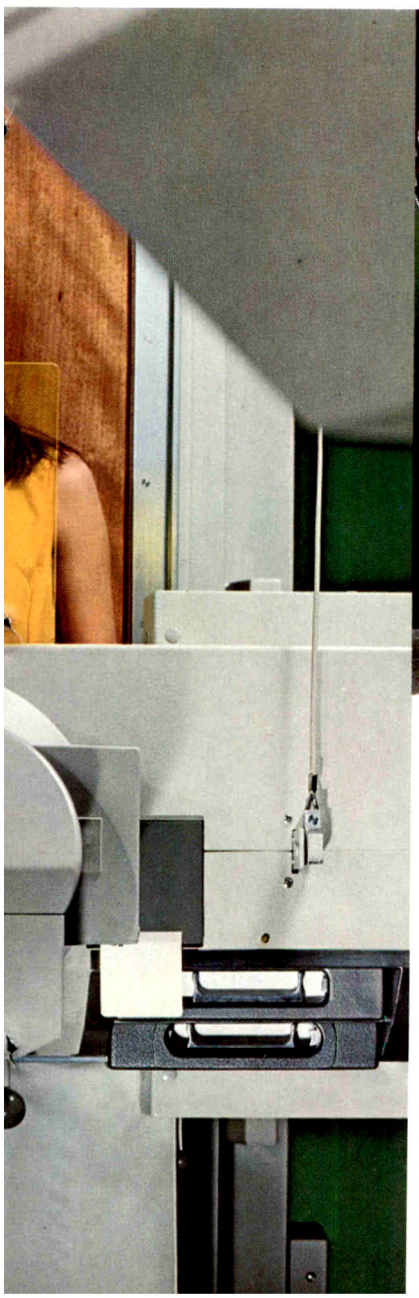
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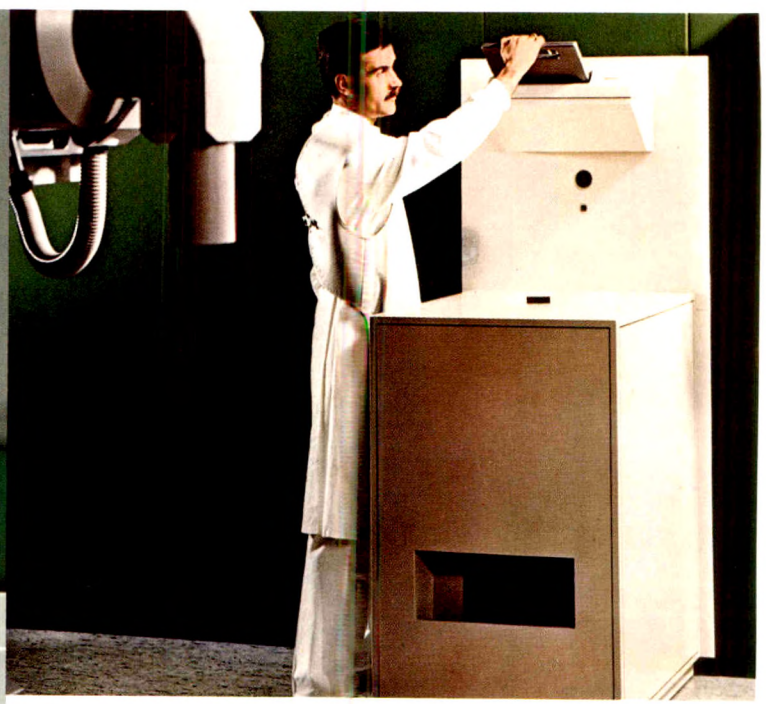
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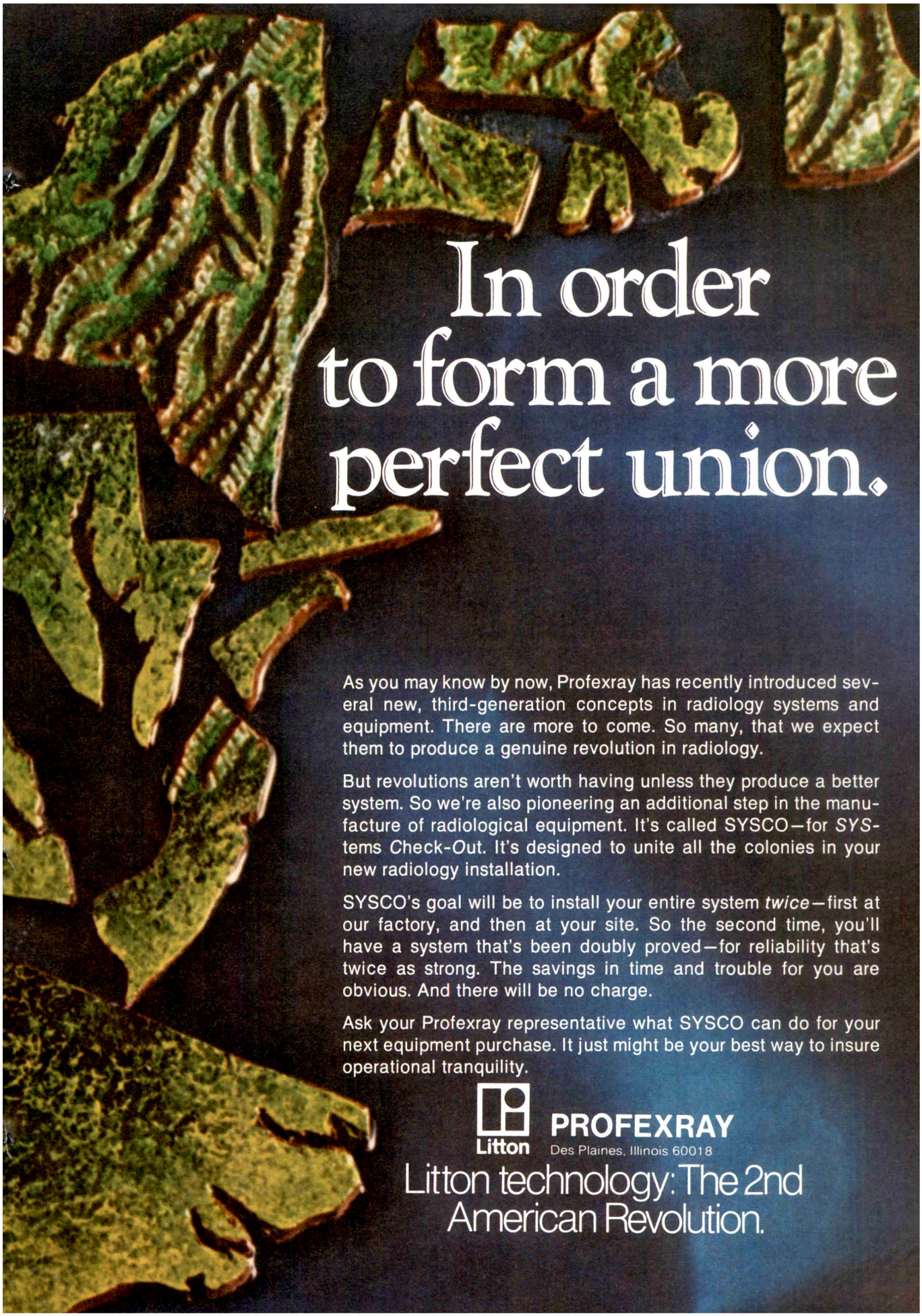
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INDEX TO ADVERTISERS

Amersham/Searle ..... Back Cover
Atomic Energy of Canada, Ltd. xi
Automatic Seriograph xxxix
CGR Medical Corporation ..... xxxii, xxxiii
Eastman-Kodak ..... xliv, xlv
Elema-Schonander xxv
Flow Pharmaceuticals ..... Inside Front Cover
General Electric ..... xlviii, xlix
Grune & Stratton i
Ilford X-Ray ..... xx, xxi
Low X-Ray xxxi
Machlett Laboratories liv
Donald McElroy xlii
Ortho Pharmaceuticals xxxviii
Philips Medical Systems (England) ..... xvi, xvii
Philips Medical Systems ..... xl, xli
Picker Corporation xxix
Procter & Gamble ..... xxvi, xxvii, xxviii
Profexray ..... xviii, xix, xxxv, lvi, lvii, Inside Back Cover
Purdue-Frederick v
Radx xxxiv
Siemens Corporation ..... xxxvi, xxxvii, lii, liii
Squibb, E. R. & Sons ..... viii, ix, x, xxii, xxiii, xxiv
S & S X-Ray xliii
Thomas, Charles C, Publisher ..... xxviii, xliii
3-M Company ..... xii, xiii
Toshiba vii
Varian Radiation Division ..... l, li
Winthrop Laboratories ..... xiv, xv, xlvi, xlvii

•

We try to present an accurate index. Occasionally this may not be possible because of a last-minute change or an omission.

From the beginning, fluoroscopy procedures have required a compromise from x-ray equipment designers. Brightness, contrast, total available information must be tempered by dosage limitations. Some designers put an iris (mechanical or auto-gain) on the camera. But that merely reduces exposure to the camera, not to the patient. Others yo-yo the MA. But that adds a sluggishness to the entire system.

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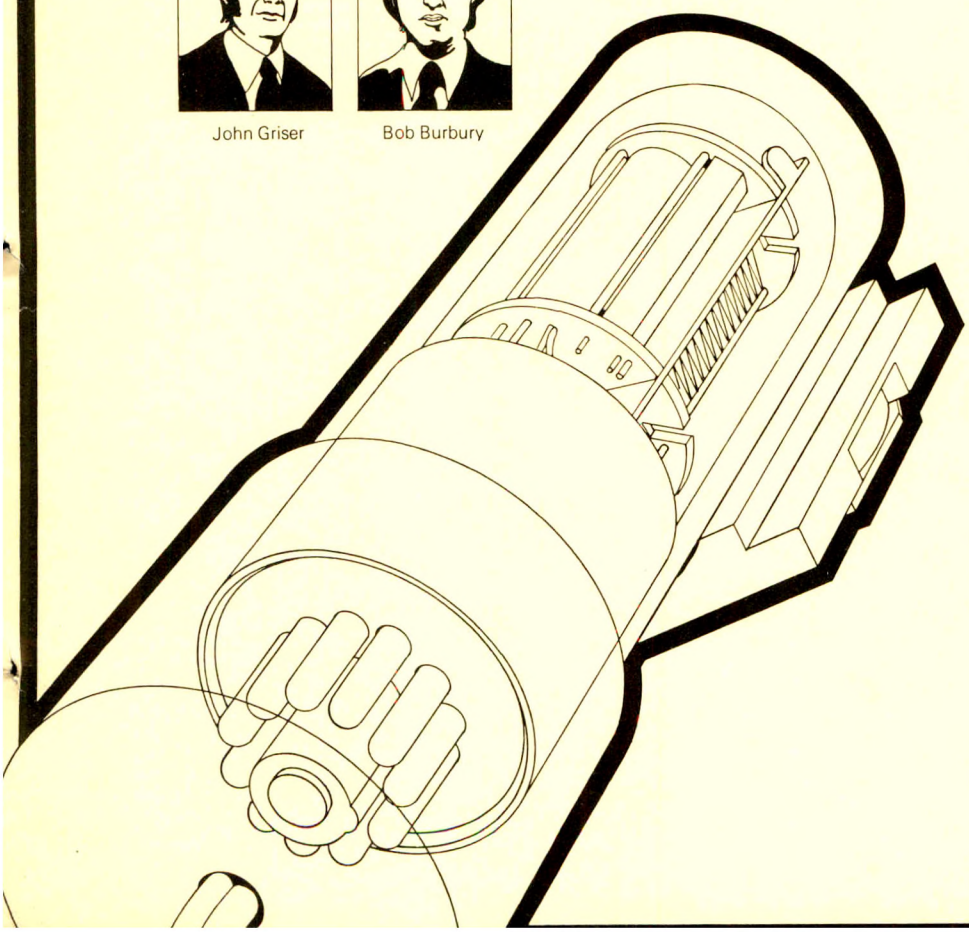
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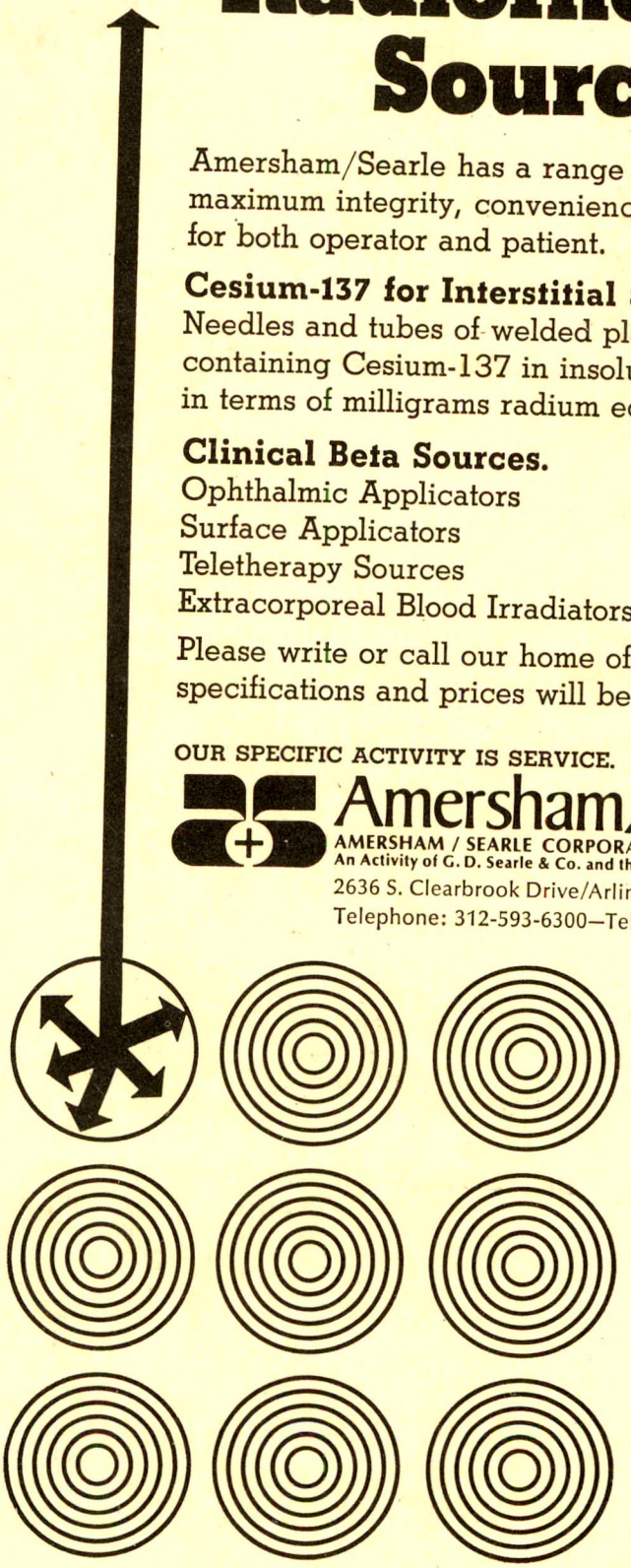
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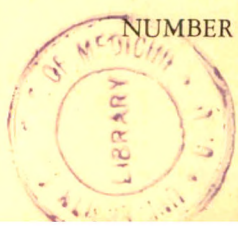
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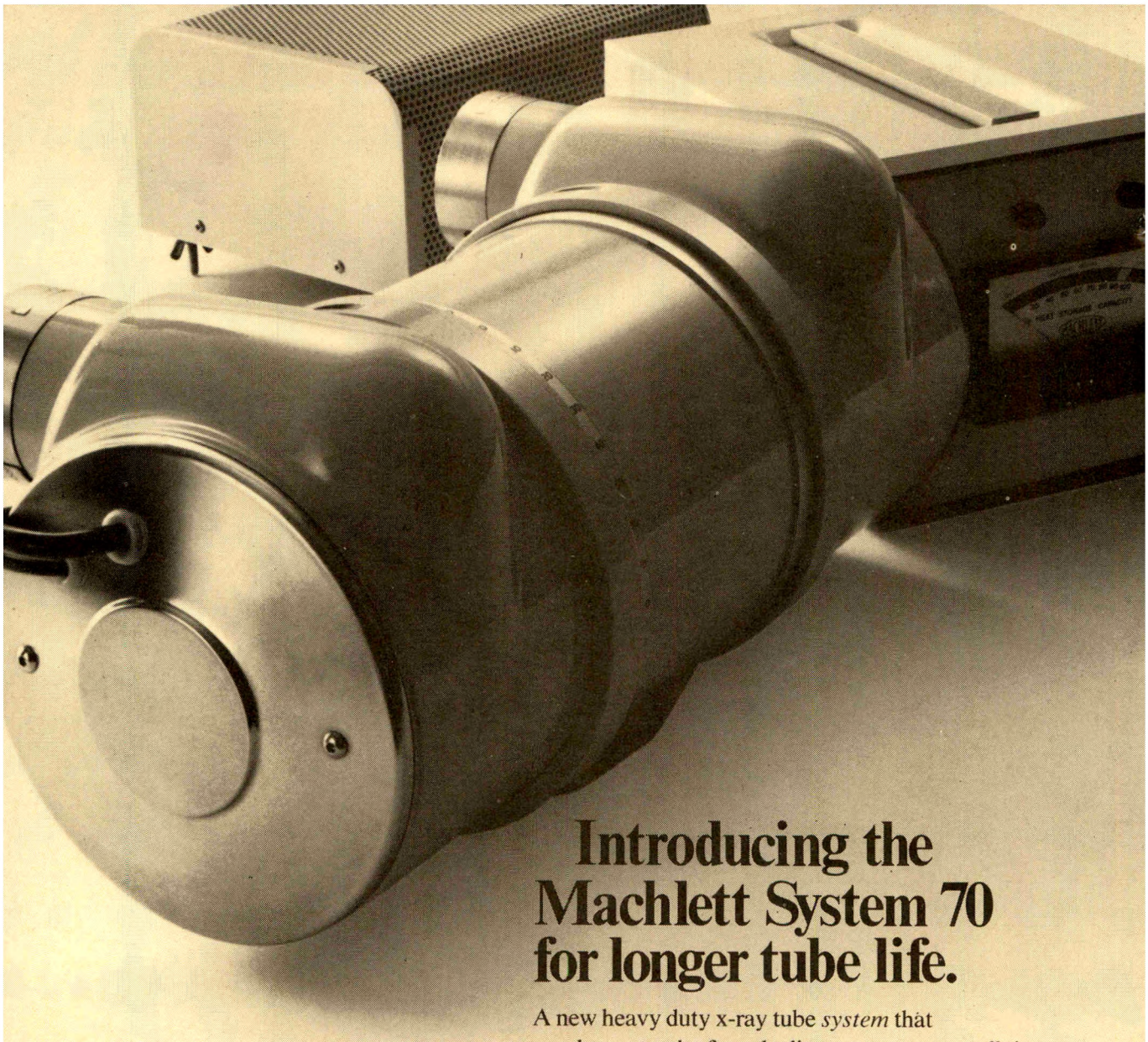
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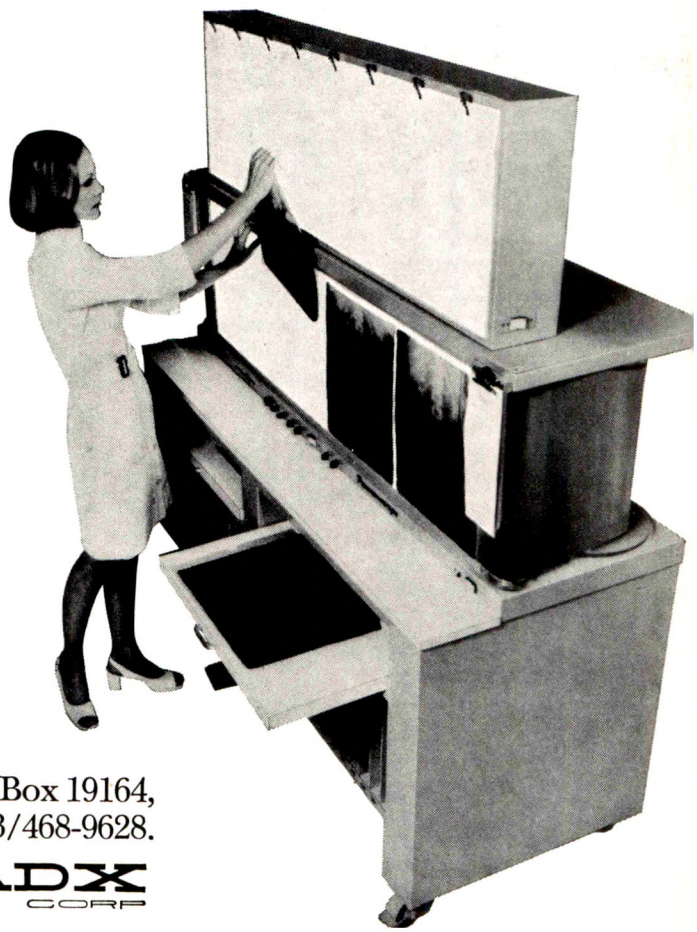
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VOL. 121 JUNE, 1974 NO. 2

TABLE OF CONTENTS

Cervical Esophageal Webs—A Roentgen-anatomic Correlation. JAMES L. CLEMENTS, JR., M.D., GEORGE W. COX, M.D., WILLIAM E. TORRES, M.D., AND H. STEPHEN WEENS, M.D. 221

Enhanced Visualization of Esophageal Varices by Buscopan. CHUNG-I. LIU, M.D. 232

Nonobstructive Mucosal Diaphragms or Rings of the Gastric Antrum in Adults. GARY G. GHAHREMANI, M.D. 236

Acquired Paraesophageal and Disparaesophageal Hernias: Complications of Hiatal Hernia Repair. TERRY HOYT, M.D., AND MYO M. KYAW, M.D. 248

Spontaneous Reduction of Hernia "En Masse." LOUIS S. PARVEY, M.D., ELLIOT HIMMELFARB, M.D., AND JACK RABINOWITZ, M.D. . . . 252

Diagnostic Value of Hypotonic Duodenography. CHARLES L. RENNELL, M.D. 256

Hypotonic Roentgenography with Glucagon. ROSCOE E. MILLER, M.D., STANLEY M. CHERNISH, M.D., JOVITAS SKUCAS, M.D., BERNARD D. ROSENAK, M.D., AND BRUCE E. RODDA, PH.D. 264

Collagenous Sprue. H. WALTER PEPPER, M.D., LLOYD L. BRANDBORG, M.D., JON D. SHANSER, M.D., HENRY I. GOLDBERG, M.D., AND ALBERT A. MOSS, M.D. 275

Roentgenographic Classification of Diffuse Diseases of the Small Intestine Presenting with Malabsorption. TIMOTHY E. TULLY, M.D., AND SAMUEL B. FEINBERG, M.D. 283

Those Other Types of Enterocolitis. TIMOTHY E. TULLY, M.D., AND SAMUEL B. FEINBERG, M.D. 291

Clindamycin (Cleocin) Colitis. LEROY E. COHEN, M.D., MAJOR, MC, CHARLES J. SMITH, M.D., MAJOR, MC, JAMES D. PISTER, M.D., CAPTAIN, MC, AND RALPH F. WELLS, M.D., COLONEL, MC . . . 301

Inflammatory Pseudotumor of the Small Intestine. WILLIAM L. CAMPBELL, M.D., WILLIAM M. GREEN, M.D., AND WILLIAM B. SEAMAN, M.D. . 305

Frequency of Visualization of the Normal Appendix with the Barium Enema Examination. RAYMOND P. SAKOVER, M.D., AND RAYMOND L. DEL FAVA, M.D. 312

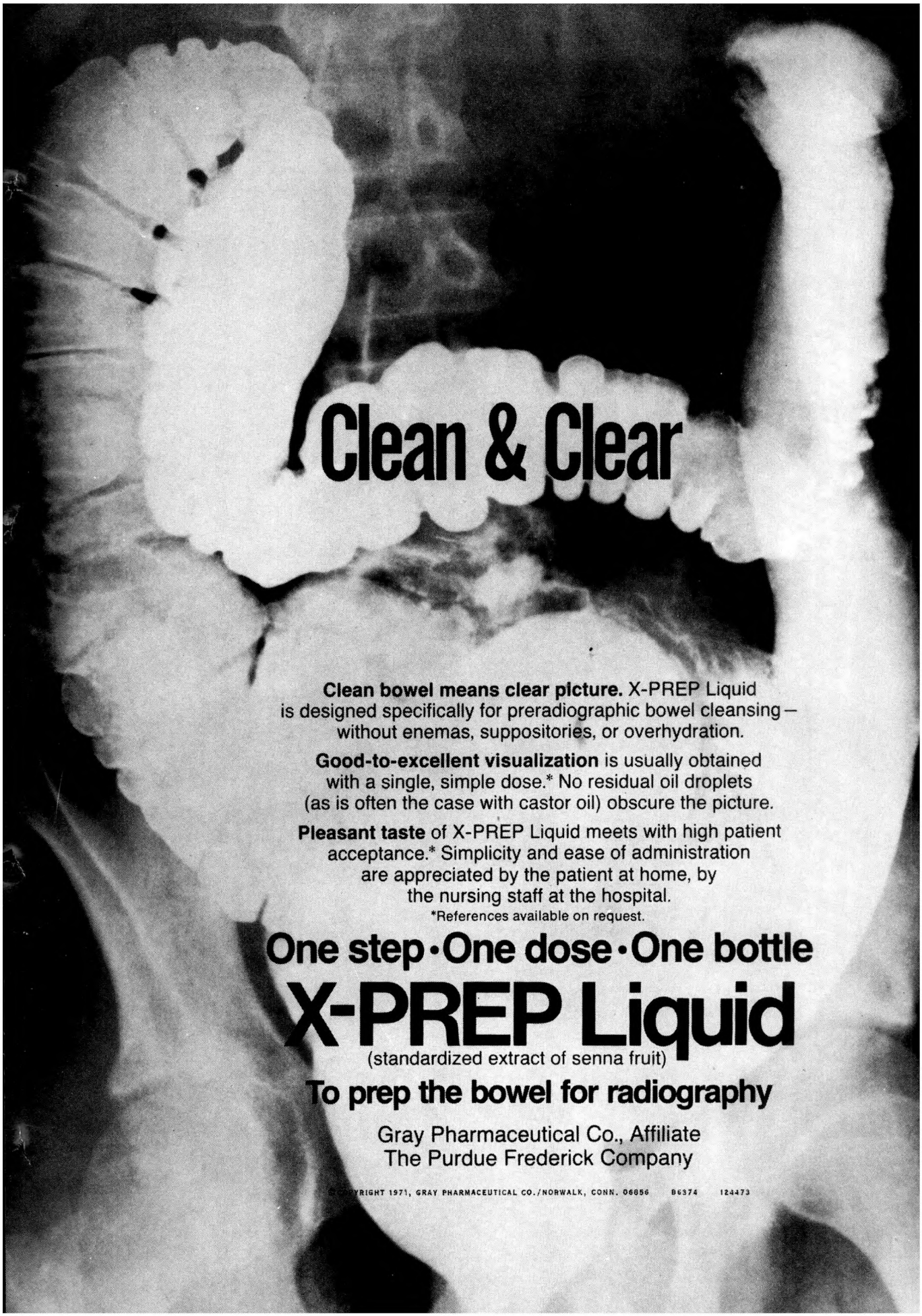
Oral Examination of the Colon: "The Colonic Cocktail." RUBEM POCHACZEVSKY, M.D. 318

The Water Double Contrast Barium Enema Study: A New Method of Roentgen Examination of the Colon. RUBEM POCHACZEVSKY, M.D. 326

(*Continued on page iv*)

LIBRARY

Giant Duodenal Diverticula. Jack R. Millard, M.D., Fred M. H. Ziter, Jr., M.D., and William P. Slover, M.D. 334
Giant Sigmoid Diverticulum. Jack G. Rabinowitz, M.D., Jack Farman, M.D., Ch.B., M.Med., Serge Dallemand, M.D., Jack Twerskey, M.D., and Yale Rosen, M.D. 338
Large Diverticulum of the Anal Canal: Case Report and Review of the Literature on Anal Canal and Rectal Diverticula. A. F. Govoni, M.D., C.R.C.P.(C), and Jan J. Smulewicz, M.D. 344
Evaluation of the Retrogastric Space: Normal Appearance and Variation. Joseph P. Whalen, M.D., Lewis M. Bader, M.D., and Richard Wolfman, M.D. 348
"Lower" Gastrointestinal Tract Hemorrhage: New Concepts Based on Arteriography. William J. Casarella, M.D., Shelby J. Galloway, M.D., Richard N. Taxin, M.D., David A. Follett, M.D., Edward J. Pollock, M.D., and William B. Seaman, M.D. 357
Ventral Portal Vein. George Harell, M.D. 369
Contrast Enhancement of the Mesenteric and Portal Veins Using Intra-arterial Papaverine. Warren C. Widrich, M.D., David L. Nordahl, M.D., and Alan H. Robbins, M.D. 374
Use of C-Terminal Octapeptide of Cholecystokinin in Cholecystography. Jonathan A. Levant, M.D., and Richard A. L. Sturdevant, M.D. . 380
Intrahepatic Arterial Portal Communications: Angiographic Study. Yacov Itzchak, M.D., Rafael Adar, M.D., Harry Bogokowski, M.D., Mark Mozes, M.D., and Victor Deutsch, M.D. 384
Pyogenic Liver Abscess: Angiographic Diagnosis and Treatment by Closed Aspiration. Stanley B. Novy, M.D., Sidney Wallace, M.D., Arnold M. Goldman, M.D., and Yoram Ben-Menachem, M.D. . 388
Gallbladder Ectopia: A Review of Anomalies of Position. David E. Blanton, M.D., Charles A. Bream, M.D., and Stanley R. Mandel, M.D., F.A.C.S. 396
Macro-Regenerating Nodule in the Cirrhotic Liver: Radiologic Features and Differential Diagnosis. Jack G. Rabinowitz, M.D., Mahesh Kinkabwala, M.D., and Sidney Ulreich, M.D. 401
Localized Deformity of the Gallbladder Secondary to Hepatic Mass Lesions. Norman Joffe, M.D., and George O. Babenco, M.D. 412
Pitfalls in the Arteriographic Differentiation of Intrahepatic and Extrahepatic Masses. Jon D. Shanser, M.D., Morton G. Glickman, M.D., and Alphonse J. Palubinskas, M.D. 420
Officers 430
Editorial
Seventy-fifth Annual Meeting of the American Roentgen Ray Society. Colin B. Holman, M.D. 431
Advance Registration, American Roentgen Ray Society 433
Preliminary Program, American Roentgen Ray Society 436
Workshops on Federal Diagnostic X-Ray Standard. Department of Health, Education, and Welfare 441
News Items 443
Book Review 446
Books Received 446
Society Proceedings 448
Abstracts of Radiologic Literature 455



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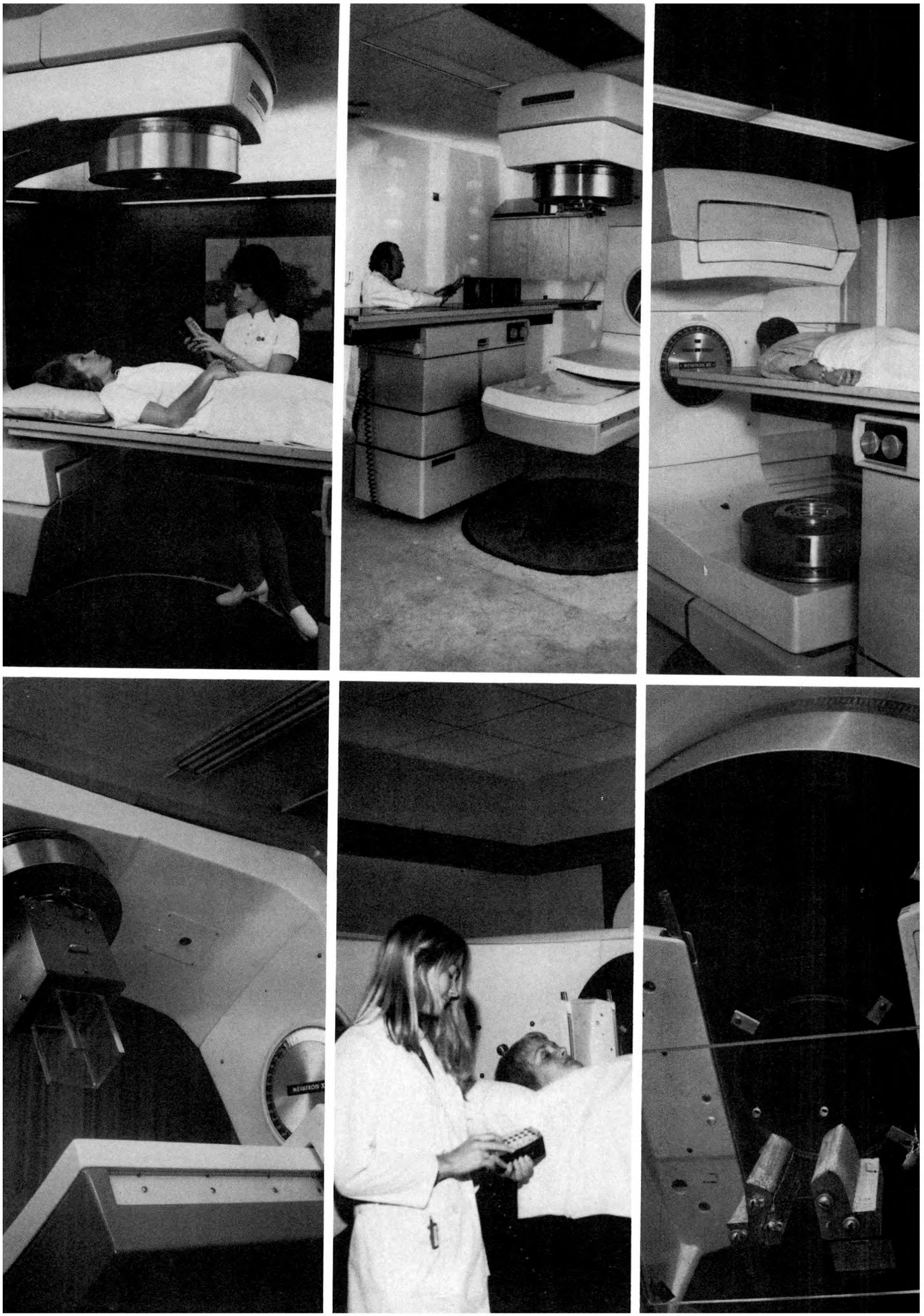
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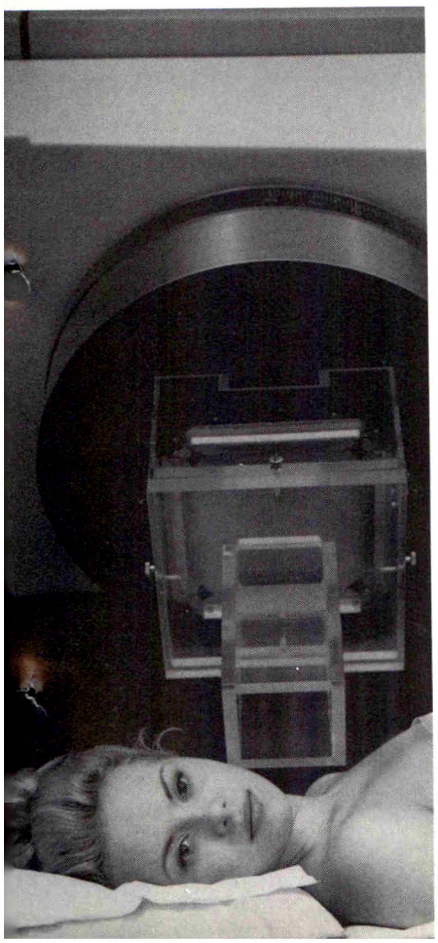
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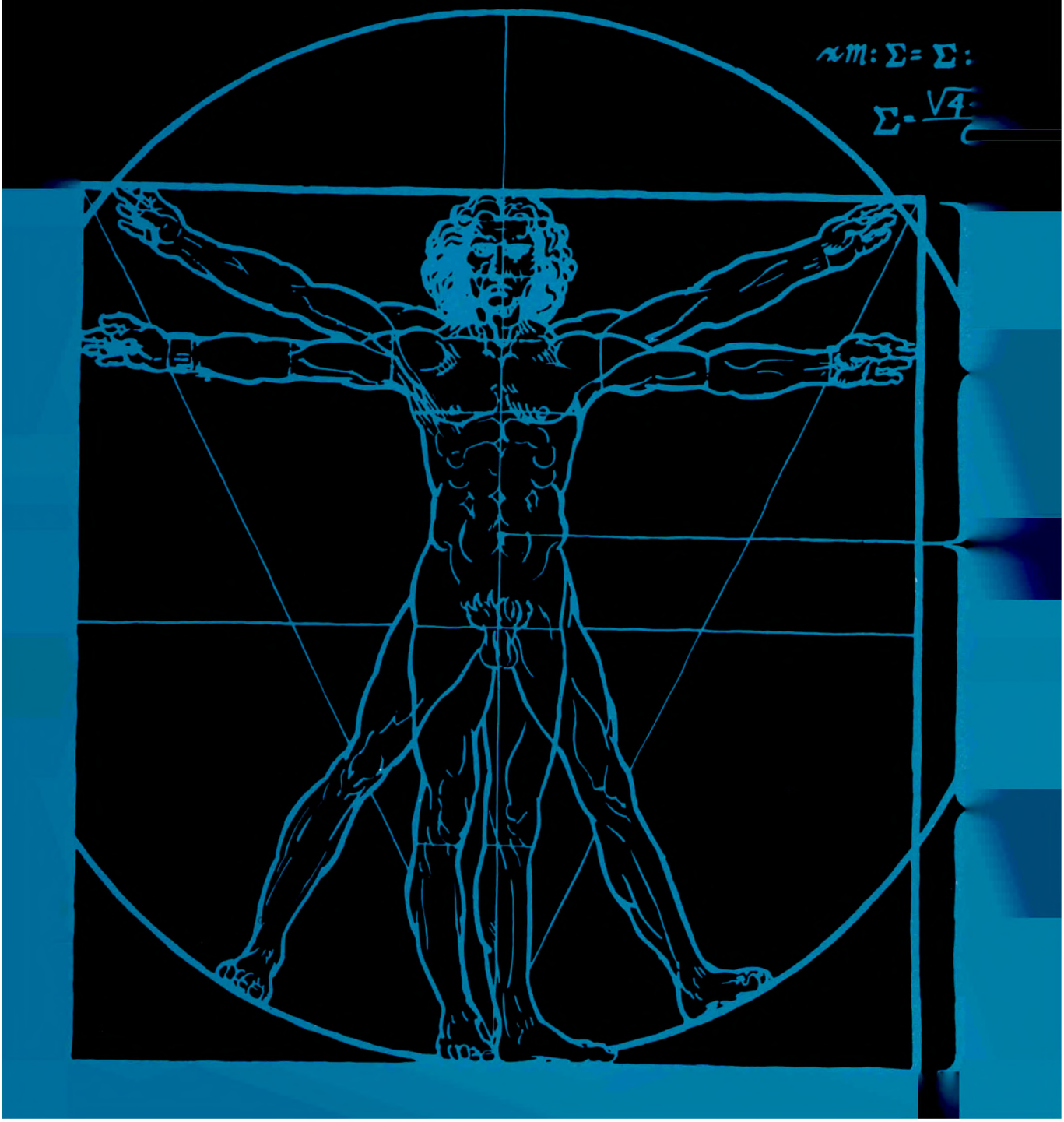
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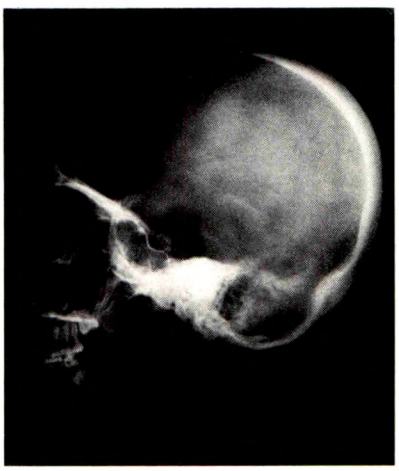
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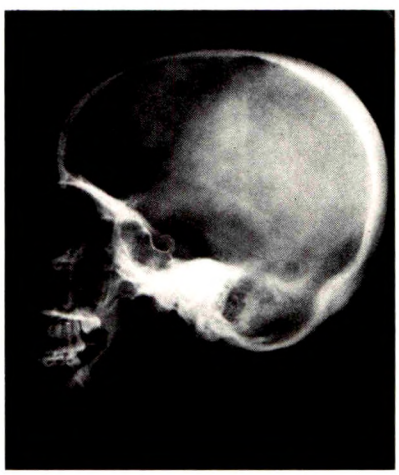
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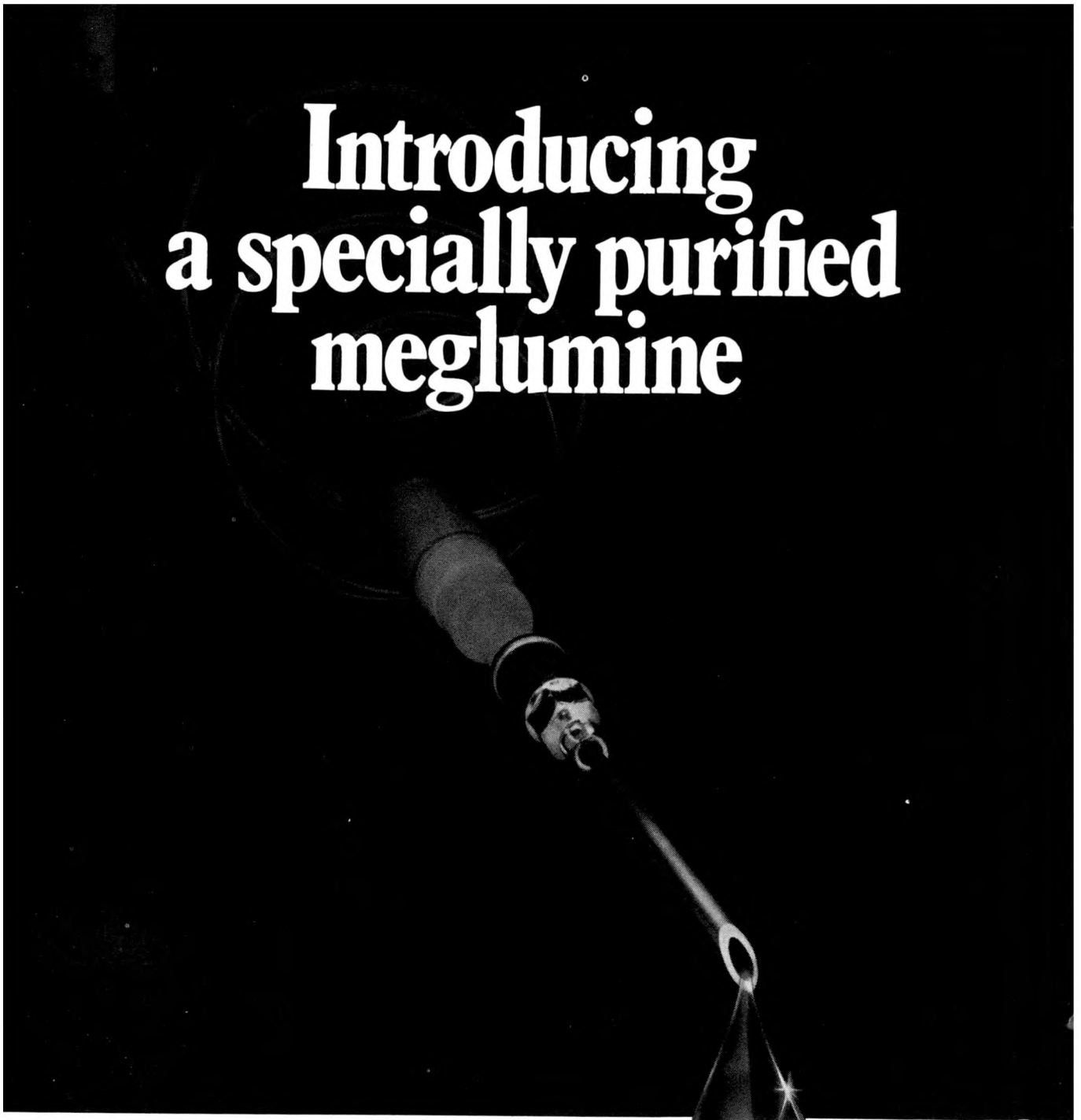
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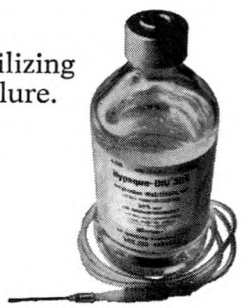
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Action: Hypaque-DIU 30 per cent is a diagnostic radiopaque medium which provides visualization of internal structures.

Indication: Hypaque-DIU 30 per cent is indicated for infusion urography.

Warnings: *Usage in Pregnancy.* No teratogenic effects attributable to a 60 per cent solution of Hypaque meglumine have been observed in reproduction studies in animals. However, before administration of Hypaque-DIU 30 per cent to women of child-bearing potential, the benefit to the patient should be carefully weighed against the possible risk to the fetus. In addition, most authorities consider elective contrast radiography of the abdomen contraindicated during pregnancy.

Serious or fatal reactions have been associated with the administration of radiopaque media. It is of utmost importance that a course of action be carefully planned in advance for the immediate treatment of serious reactions, and that adequate and appropriate facilities be readily available in case a severe reaction occurs.

Contrast media have been shown to promote the phenomenon of sickling in individuals who are homozygous for sickle cell disease when the material is injected intravascularly.

The results of PBI and RAI uptake studies, which depend on iodine estimations, will not reflect thyroid function for up to 16 days following administration of iodinated urographic media. However, function tests not depending on iodine estimations, e.g., T_3 resin uptake and direct thyroxine assays, are not affected.

Precautions: Before a contrast medium is injected, the patient should be questioned for a history of allergy. Although a history of allergy may imply a greater than usual risk, it does not arbitrarily contraindicate the use of the medium. Premedication with antihistamines to avoid or minimize possible allergic reactions may be considered.

Diphenhydramine hydrochloride (i.e., Benadryl®), however, should not be mixed with Hypaque-DIU 30 per cent since it may cause precipitation. The intravenous injection of a test dose of 0.5 to 1 ml. of the contrast agent before infusion of the full dose has been employed in an attempt to predict severe or fatal adverse reactions. The preponderance of recent scientific literature, however, now demonstrates that this provocative test procedure is not reliably predictive of serious or fatal reactions.

Severe reactions and fatalities have occurred with the test dose alone, with the full dose after a nonreactive test dose, and with or without a history of allergy. No conclusive relationship between severe or fatal reactions and antigen-antibody reactions or other manifestations of allergy has been established. A history of allergy may be useful in predicting reactions of a mild or intermediate nature.

Caution is advised in patients with severe cardiovascular disease and in patients with a history of bronchial asthma or other allergic manifestations, or of sensitivity to iodine.

In myelomatosis, urography should only be performed with caution. If a weak protein-binding agent such as a diatrizoate is used for the procedure, it is essential to omit preparatory dehydration, to administer fluids, and attempt to alkalinize the urine.

Immediate adverse reactions to infusion urography have not been reported to occur at a higher frequency or with greater severity than with routine excretory urography. However, sequelae of this procedure are possible some hours after the examination. Infusion urography imposes a sudden osmotic load. This should be considered in patients with established, decreased glomerular filtration and renal tubular damage. In addition, these patients may also have coexisting or associated cardiovascular disease. Therefore, there is a possibility of the development of congestive heart failure hours after the procedure.

Because of the possibility of inducing temporary suppression of urine, it is wise to allow an interval of at least 48 hours to pass before repeating infusion urography in patients with unilateral or bilateral reduction of normal renal function.

Preparatory dehydration or abdominal compression may not be necessary in infusion urography. In infants and young children and in azotemic patients, especially those with oliguria, dehydration is considered dangerous.

Adverse Reactions: Reactions accompanying the use of contrast media may vary directly with the concentration of the substance, the amount used, the technique used, and the underlying pathology.

The following have been reported after administration of diatrizoates and other iodinated contrast media. *Cardiovascular reactions:* vein cramp and thrombophlebitis following intravenous injection, rare cases of cardiac arrhythmias, hypertension, hypotension and shock, and cardiac arrest. *Allergic reactions:* asthmatic attacks, nasal and conjunctival symptoms, dermal reactions such as urticaria, and rarely, anaphylactic reactions. Signs and symptoms related to the *respiratory system:* pulmonary or laryngeal edema, bronchospasm; or to the *nervous system:* restlessness, convulsions. *Other reactions:* flushing, pain, warmth, nausea, vomiting, anxiety, headache, dizziness, hematoma, and ecchymosis. Occasionally, transient proteinuria, and rarely, oliguria or anuria. Rarely, immediate or delayed rigors can occur, sometimes accompanied by hyperpyrexia. Infrequently, "iodism" (salivary gland swelling) from organic iodinated compounds appears two days after exposure and subsides by the sixth day.

Dosage and Administration: The recommended dose for infusion urography is calculated on the basis of 2 ml. of Hypaque-DIU 30 per cent per pound of body weight. The average dose of the solution for adults is 300 ml.; for optimal results, a minimum dose of 250 ml. should be used. A maximum dose of 400 ml. is generally sufficient for the largest of subjects.

The solution is infused over a period of three to ten minutes. Radiographs are usually taken at 10, 20, and 30 minutes from the beginning of the infusion. Earlier films may be obtained for the evaluation of hypertension. Nephrograms are usually obtained at the end of the infusion, and voiding cystourethrograms are usually obtained 30 minutes after the beginning of the infusion.

How Supplied: Calibrated bottles of 300 ml., rubber stoppered, packed with and without sterile disposable intravenous infusion sets.

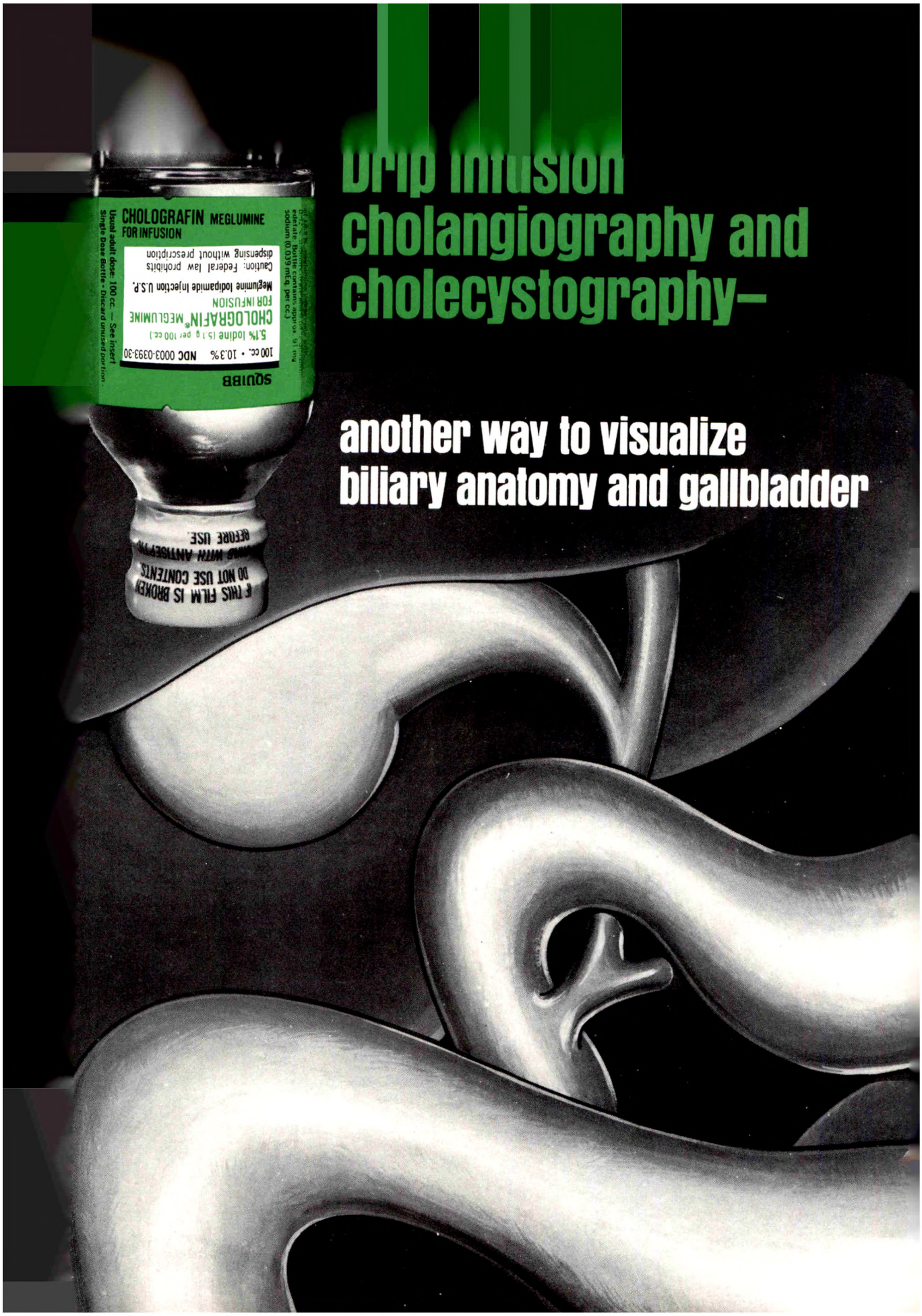
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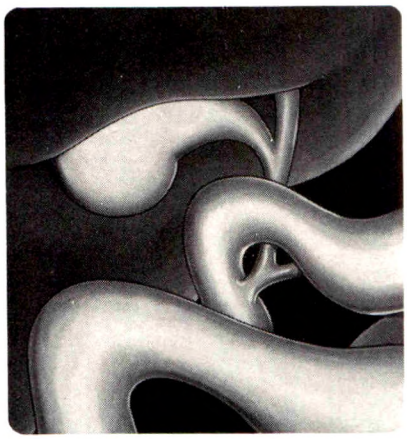


(1621M)



Drip infusion cholangiography and cholecystography—

another way to visualize biliary anatomy and gallbladder



Clinical Comparison of Drip-infused and Injected Cholografin® Meglumine (Meglumine Iodipamide Injection U.S.P.) in a total of 198 Patients*

- **Lower incidence of adverse reactions with drip infusion**
  Drip infusion—7.4% (10 of 135 patients) had reactions
  Injection—19% (12 of 63 patients) had reactions
  See brief summary on next page for reactions which can occur.
- **Good visualization with both techniques**
  Drip infusion—84.4% were of excellent to fair quality
  Injection—75.4% were of excellent to fair quality
- **Methods of administration**
  Drip infusion—100 ml. of 10.3% meglumine iodipamide infused over a 30-minute period
  Injection—20 ml. of 52% meglumine iodipamide injected over a 10-minute period

CHOLOGRAFIN® MEGLUMINE FOR INFUSION

(Meglumine Iodipamide Injection U.S.P.)

in 100 ml. single-dose bottles
ready-to-use for drip infusion cholangiography and cholecystography

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*Information on file at Squibb Institute for Medical Research.

For brief summary, see next page.

for patients undergoing cholangiography and cholecystography

CHOLOGRAFIN® MEGLUMINE FOR INFUSION

(Meglumine Iodipamide Injection U.S.P.)

CHOLOGRAFIN® MEGLUMINE

(Meglumine Iodipamide Injection U.S.P.)

Cholografin Meglumine for Infusion (Meglumine Iodipamide Injection U.S.P.) is a sterile aqueous solution providing 10.3% meglumine iodipamide with 0.32% sodium citrate as a buffer and 0.04% disodium edetate as a sequestering agent. The bound iodine content is about 5.1% (5.1 g./100 ml.). *The solution contains approximately 0.91 mg. (0.039 mEq.) sodium per ml. (91 mg./100 ml.).*

Cholografin Meglumine (Meglumine Iodipamide Injection U.S.P.) is a sterile aqueous solution providing 52% meglumine iodipamide with 0.32% sodium citrate as a buffer and 0.04% disodium edetate as a sequestering agent. The bound iodine content is about 26% (5.2 g./20 ml.). *The solution contains approximately 0.91 mg. (0.039 mEq.) sodium per ml. (18.2 mg./20 ml.).*

CONTRAINDICATIONS: Contraindicated in patients hypersensitive to salts of iodipamide; in patients exhibiting sensitivity reactions to the test dose; in the presence of concomitant severe renal and liver function impairment.

WARNINGS: Intravenous or intra-arterial injections of contrast media have been shown to promote the phenomenon of sickling in persons homozygous for sickle cell disease. Perform thyroid function tests, if indicated, before administration of iodine-containing contrast media may alter results of such tests. A history of sensitivity to iodine *per se* or to other contrast agents is not an absolute contraindication to the use of meglumine iodipamide but calls for extreme caution in administration.

Usage in Pregnancy: The safety of meglumine iodipamide for use during pregnancy has not been established; therefore, it should be used in pregnant patients only when, in the judgment of the physician, its use is deemed essential to the welfare of the patient.

PRECAUTIONS: Diagnostic procedures involving use of contrast agents should be performed under the direction of personnel with prerequisite training and a thorough knowledge of the particular procedure. Appropriate facilities should be available for coping with situations which may arise as a result of the procedure and for emergency treatment of severe reactions to the contrast agent itself; competent personnel and emergency facilities should be available for at least 30 to 60 minutes after I.V. administration since severe delayed reactions have been known to occur. These severe life-threatening reactions suggest hypersensitivity to the contrast agent. A personal or family history of asthma or allergy or a history of a previous reaction to a contrast agent warrants special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 ml. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated. When the 52% meglumine iodipamide preparation is used, some clinicians feel it may be advisable to have a continuous I.V. infusion running prior to administration of the drug.

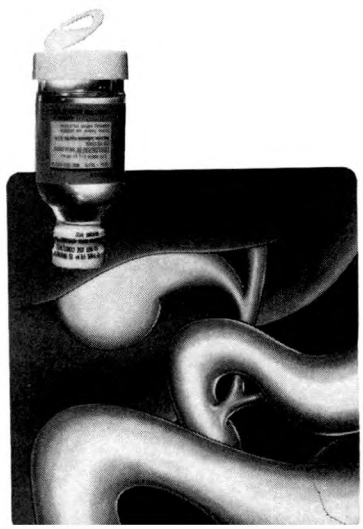
ADVERSE REACTIONS: Local reactions at the site of infusion or injection are not observed unless excessive amounts are extravasated during the infusion or injection. After too rapid administration, mild transient symptoms such as restlessness, sensations of warmth, sneezing, perspiration, salivation, flushing, pressure in the upper abdomen, dizziness, nausea, and vomiting may occur. These symptoms disappear when the infusion or injection has been completed. Hypersensitivity reactions may occur. In rare instances, despite the most careful sensitivity testing, anaphylactoid reactions may occur. Renal function tests may be altered and renal failure may occur.

For full information, consult package inserts.

HOW SUPPLIED: Cholografin Meglumine for Infusion (Meglumine Iodipamide Injection U.S.P.)—10.3%—is available in single-dose bottles of 100 ml. Cholografin Meglumine (Meglumine Iodipamide Injection U.S.P.)—52%—is available in single-dose vials and ampuls of 20 ml.

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TECHNOLOGY UTILIZATION
PROGRAM AND INSTRU-
MENT DEMONSTRATION

Application Of Optical Instrumentation In Medicine III

AUGUST 1-2, 1974
Muehlebach Hotel,
Kansas City, Missouri

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THE SOCIETY OF PHOTO-OPTICAL INSTRUMENTATION ENGINEERS

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Bureau of Radiological Health, Dept. of Health, Education and Welfare

COOPERATING SOCIETY
American Association of Physicists in Medicine



PROGRAM CHAIRMEN: Paul L. Carson, Edward L. Chaney, William R. Hendee, University of Colorado Medical Center

SPIE Technology Utilization Programs on "APPLICATION OF OPTICAL INSTRUMENTATION IN MEDICINE" held during the last two years have demonstrated the need for a forum for ongoing interchange of ideas and capabilities among physicians, physicists, engineers and manufacturers involved in optical instrumentation and its applications to medicine. In response to that need, a third Technology Utilization Program is planned in conjunction with the 16th Annual Meeting of the American Association of Physicists in Medicine in Kansas City.

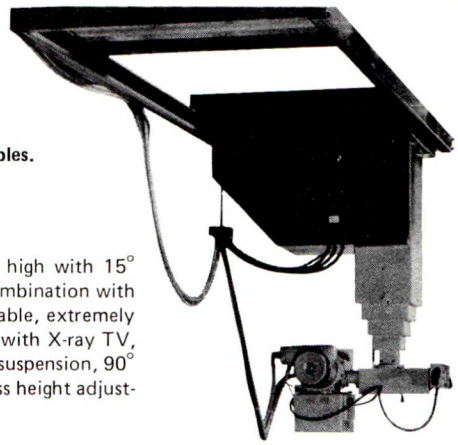
This program will be devoted to an in-depth exploration of topics chosen for their current interest and for their potential for improvement of selected diagnostic medical procedures.

Scheduled Sessions:

- **Computerized Axial Tomography***
- **Focal Spot Size and Intensity Distribution and Their Importance in Magnification Radiography**
- **Problems with Automatic Brightness Control Systems for Image Intensification Fluoroscopy**
- **Psychophysics and Observer Performance Evaluation**
- **Ultrasound Imaging and Diagnosis**
- **Ultrasound Intensity Measurements**
- **Three panel discussions addressing areas of critical interest in Medical Imaging by leaders in the field.**

*August 1—Jointly sponsored SPIE/AAPM Technical Session on COMPUTERIZED AXIAL TOMOGRAPHY.

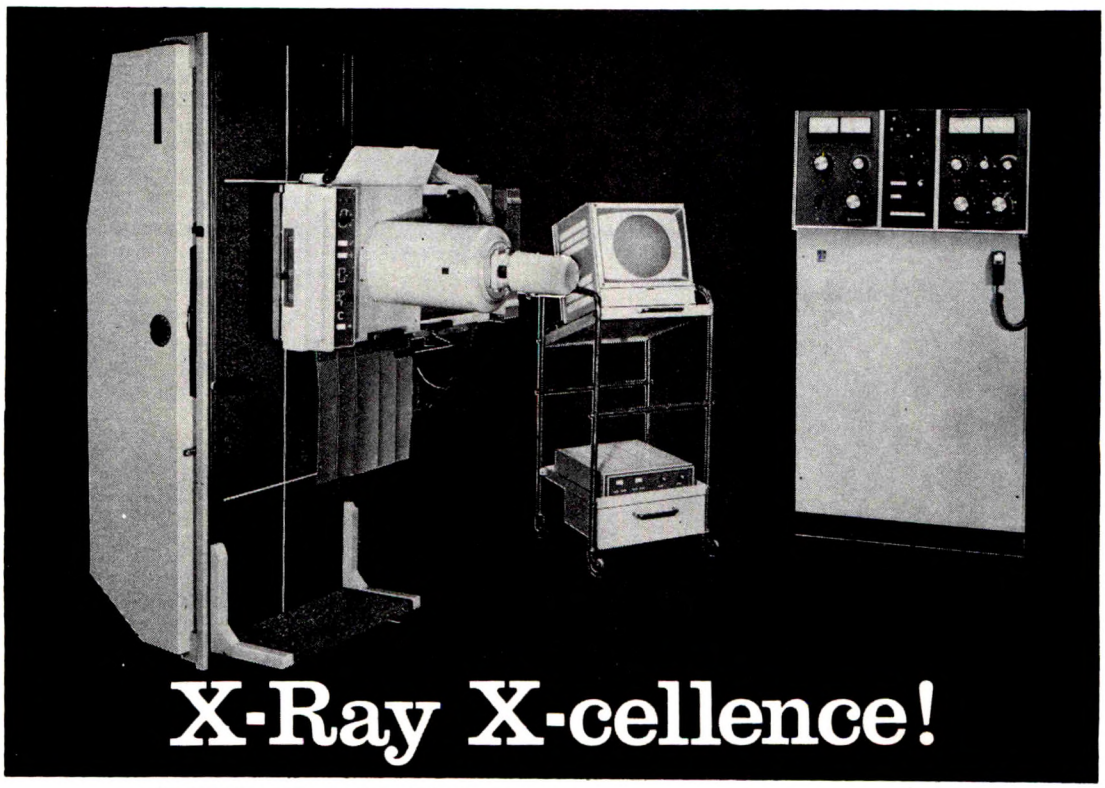
For full program information and registration contact SPIE Headquarters, P. O. Box 1146, Palos Verdes Estates, California 90274 (213) 378-1216.



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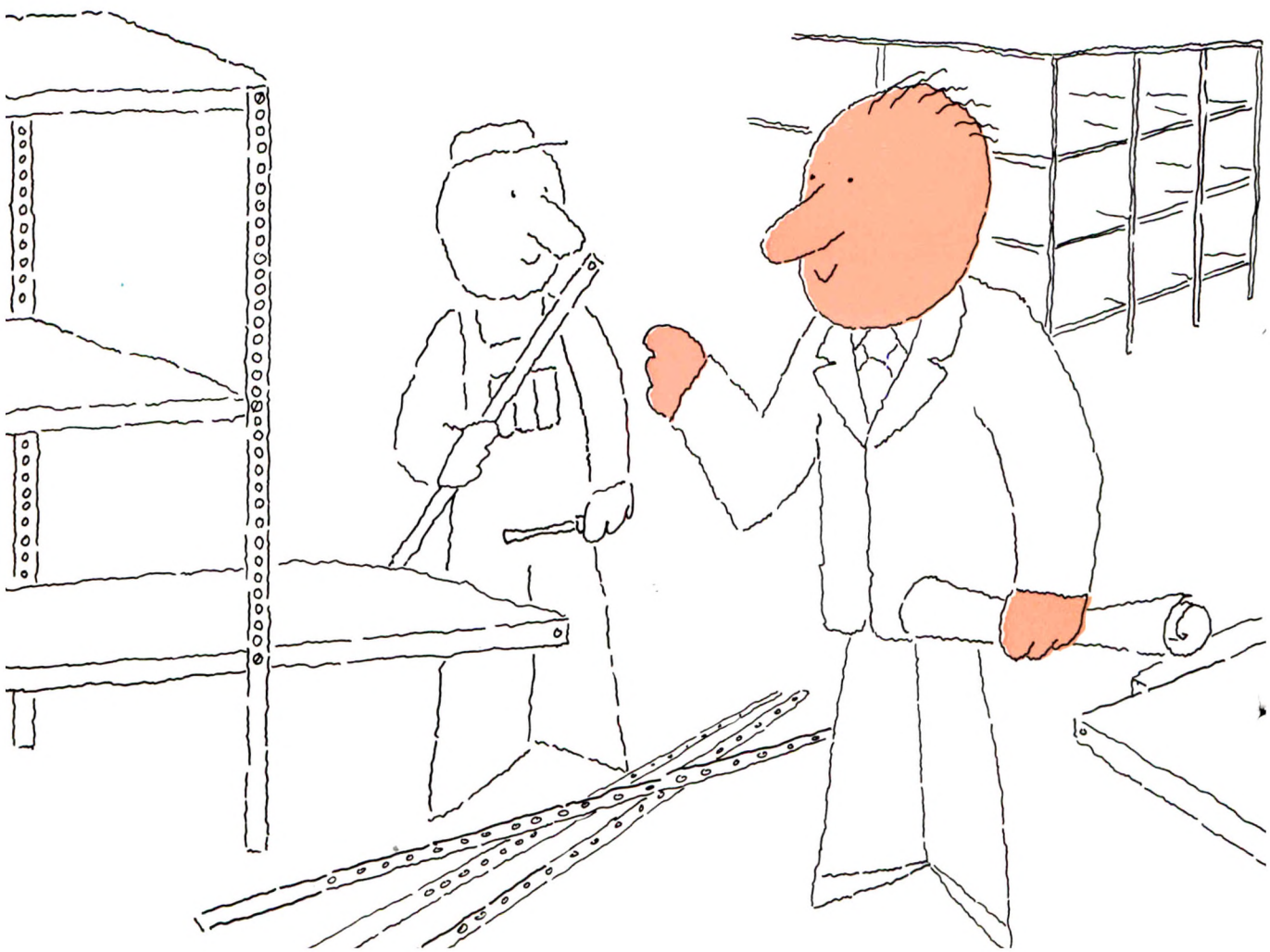
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and exciting things for you.

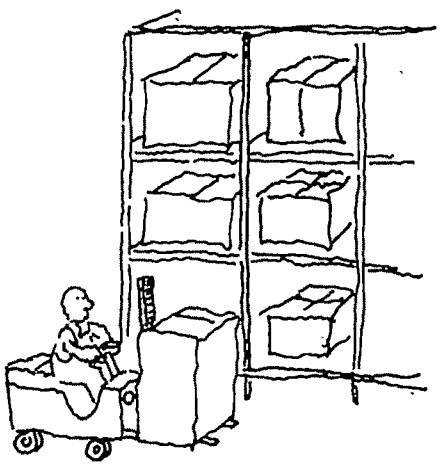
most important to you, we're adding more technically skilled Ilford men and women all around the country to help you get the most from your equipment and our materials.

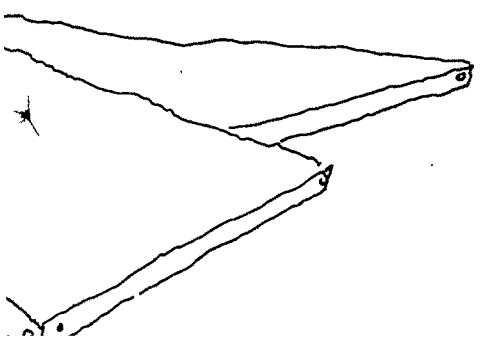
If you don't use Ilford yet, keep an eye on us. On our full line of films for general radiography. On our new family of cine films. On our intensifying screens and chemicals. On our quality control monitor system. And most importantly, on the thing that puts them all together for you—The Ilford Man, an X-ray professional who knows your department's needs inside out.

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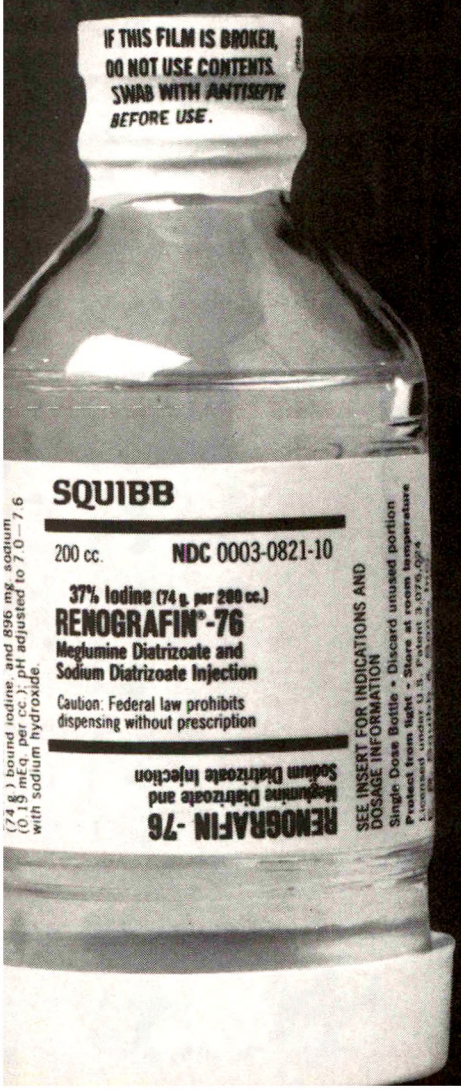
The Ilford Man is on the move.

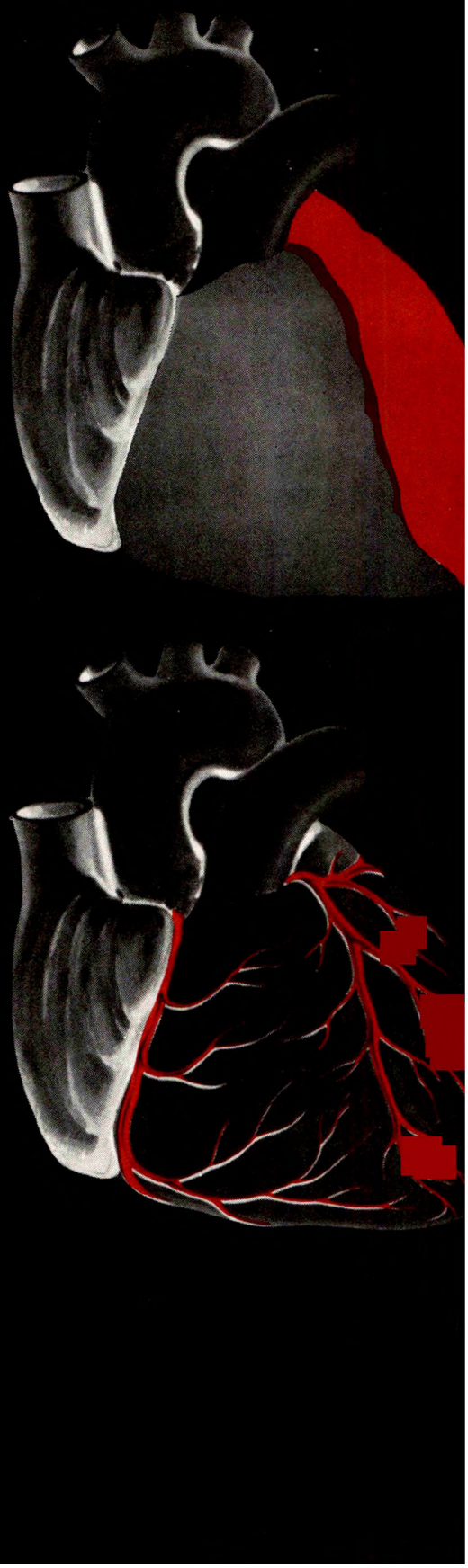




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nografin-76 is supplied as a sterile, aqueous lution providing 66% meglumine diatrizoate d 10% sodium diatrizoate with 0.32% sodium rate as a buffer and 0.04% disodium edetate a sequestering agent. The solution contains proximately 37% (370 mg./cc.) bound iodine d approximately 4.48 mg. (0.19 mEq.) sodium r cc.

ntraindications: In patients with a hypersenivity to salts of diatrizoic acid. Urography ntraindicated in patients with anuria.

arnings: A definite risk exists with the use of ntrast agents in excretion urography in paents with multiple myeloma. There has been uria with progressive uremia, renal failure d death. This risk of the procedure in these tients is not a contraindication; however, rtial dehydration in preparation for study is t recommended since it may predispose for ecipitation of myeloma protein in renal bules. No therapy, including dialysis, has en successful in reversing this effect. yeloma should be considered in persons over before undertaking urographic procedures. In cases of known or suspected pheochrocytoma, if the physician feels that the posle benefits outweigh the considered risks, diopaque materials should be administered th extreme caution; however, an absolute nimum of material should be injected, the ood pressure should be assessed throughout e procedure, and measures for treating a pertensive crisis should be available.

Contrast media may promote sickling in mozygous individuals when injected I.V. or ra-arterially. Although a history of sensitivity iodine *per se* or to other contrast media is t an absolute contraindication, administran of diatrizoate requires extreme caution in ch cases. Renografin-76 should be used in egnant patients only when the physician ems its use essential to the welfare of the ient since safe use during pregnancy has been established. Perform thyroid function ts prior to administration of Renografin-76 ce iodine-containing contrast agents may er the test results.

Veigh the inherent risks against necessity performing *angiocardiography* in cyanotic ants and patients with chronic pulmonary physema. In *pediatric angiocardiography,* a e of 10 to 20 cc. may be particularly hazous in infants weighing less than 7 kg.; this is probably significantly increased if these ants have preexisting right heart "strain," t heart failure, and effectively decreased obliterated pulmonary vascular beds. Per- *urography* with extreme caution in pers with severe concomitant hepatic and al disease. Perform *selective coronary arography* only in selected patients and those whom expected benefits outweigh the produral risk.

cautions: Diagnostic procedures involving of contrast agents should be performed er the direction of personnel with prereqte training and a thorough knowledge of the ticular procedure. Appropriate facilities uld be available for coping with situations ch may arise as a result of the procedure for emergency treatment of severe reons to the contrast agent itself; competent sonnel and emergency facilities should be lable for at least 30 to 60 minutes after I.V. inistration since delayed reactions have n known to occur. These severe lifeatening reactions suggest hypersensitivity e contrast agent. A personal or family hisof asthma or allergic rhinitis or

special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 cc. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents; therefore, if known or suspected hepatic or biliary disorder exists, administration of Renografin-76 should be postponed following the ingestion of cholecystographic agents. Consider the functional ability of the kidneys before injecting Renografin-76. Use cautiously in severely debilitated patients and in those with marked hypertension. Bear in mind the possibility of thrombosis when using percutaneous techniques.

In *excretion urography,* adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be hazardous; this also applies to *pediatric angiocardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography,* hypotension or moderate decreases in blood pressure seem to occur frequently with intra-arterial (brachial) injections; this is transient and usually requires no treatment. Monitor blood pressure during the immediate 10 minutes after injection. It is recommended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and adequate facilities for immediate resuscitation and cardioversion.

Adverse Reactions: Nausea, vomiting, flushing, or a generalized feeling of warmth are the reactions seen most frequently with intravascular injection. Symptoms which may occur are chills, fever, sweating, headache, dizziness, pallor, weakness, severe retching and choking, wheezing, a rise or fall in blood pressure, facial or conjunctival petechiae, urticaria, pruritus, rash, and other eruptions, edema, cramps, tremors, itching, sneezing, lacrimation, etc. Antihistaminic agents may be of benefit; rarely, such reactions may be severe enough to require discontinuation of dosage. There have been a few reports of a burning or stinging sensation or numbness, of venospasm or venous pain, and of partial collapse of the injected vein. Neutropenia or thrombophlebitis may occur. Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovascular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, agitation, confusion, and cyanosis progressing to unconsciousness. An allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoid shock may occur. Temporary renal shutdown or other nephropathy may occur.

Adverse reactions as a consequence of *excretion urography* include cardiac arrest, ventricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to generalized vasodilation. Risks of *aortography* procedures include injury to aorta and neighboring organs, pleural puncture, renal damage (including infarction and acute tubular necrosis

procedure in presence of preexistent renal disease, retroperitoneal hemorrhage from translumbar approach, spinal cord injury and pathology associated with syndrome of transverse myelitis, generalized petechiae, and death following hypotension, arrhythmia, and anaphylactoid reactions. In *pediatric angiocardiography,* arrhythmia and death have occurred. During *peripheral arteriography,* hemorrhage from puncture site, thrombosis of the vessel, and brachial plexus palsy (following axillary artery injection) have occurred. During *selective coronary arteriography* and *selective coronary arteriography with left ventriculography,* transient ECG changes (most patients); transient arrhythmias (infrequent); ventricular fibrillation (from manipulation of catheter or administration of medium); hypotension; chest pain; myocardial infarction; transient elevation of creatinine phosphokinase (occurred in about 30% of patients tested); fatalities have been reported; hemorrhage, thrombosis, pseudoaneurysms at puncture site, dislogment of arteriosclerotic plaques, dissection of coronary vessels, and transient sinus arrest have occurred due to the procedure.

For full prescribing information, consult package insert.

How Supplied: Available in 20 cc. and 50 cc. single-dose vials and in 200 cc. single-dose bottles.

SQUIBB HOSPITAL DIVISION



THE SOFT·STEERABLE CATHETER SYSTEM FOR NON-OPERATIVE REMOVAL OF RETAINED BILIARY STONES VIA THE BURHENNE TECHNIQUE*

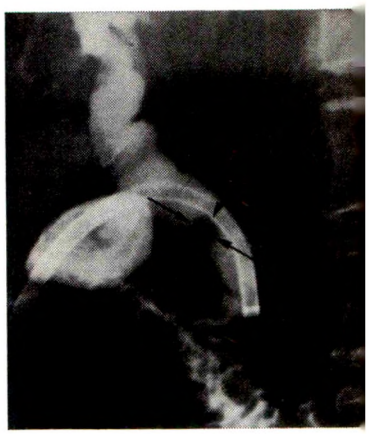


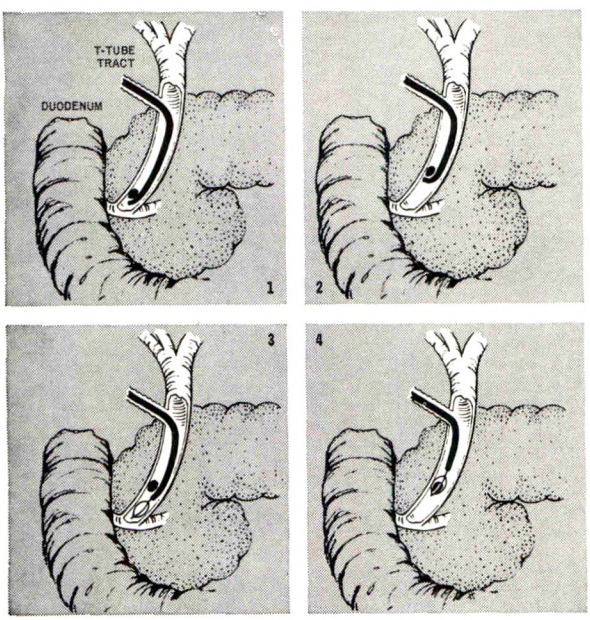
CONCEPT

Using the Burhenne technique and the MEDI-TECH steerable catheter, a significant number of non-operative removals of retained biliary stones have been achieved with consistent success.*

The safe, simple procedure, developed by Dr. H. Joachim Burhenne, is performed as a follow-up to cholecystectomy, after it has been determined, by a postoperative cholangiogram, that there are retained stones. The procedure is performed, at least four weeks after surgery, by removing the T-tube and advancing the MEDI-TECH Soft Steerable Catheter through the remaining T-tube tract.

Unique maneuverability of the soft-steerable catheter tip permits the physician to dislodge, manipulate and reposition the stone(s). Contrast media is injected through the Luer-Lock and the Open Lumen of the MEDI-TECH catheter to enhance visualization. Then, with the stone in desired position, a wire basket or snare is passed through the Tuohy-Borst fitting and the open lumen to the site. When the stone is secured, the entire assembly is withdrawn. Although the soft-tip provides sufficient torque for this procedure, patient discomfort is minimal and no anesthesia or pre-medication is used.





*REFERENCE

Burhenne, H. J., Non-operative Retained Biliary Tract Stone Extraction — A New Roentgenologic Technique, AMER. J. ROENTGENOLOGY, Vol. CXVII, No. 2, pp. 388-399, February 1973.

AN X-RAY CINE FILM of the Burhenne procedure is available. Please contact MEDI-TECH to arrange a viewing of the film and to obtain copies of reference articles plus complete information on the MEDI-TECH/BURHENNE Biliary Catheter System.

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Anterior: 834,518 counts/1070 sec (17.8 min)
Comments: Metastatic meningioma

B. 15 mCi ^{99m}Tc-EHDP
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High Sensitivity Collimator
Speed: 32 cm/min, Length: 170 cm, Width: 60 cm
Posterior: 961,752 counts/1054.3 sec (17.6 min)
Comments: Cancer of breast. Polaroid image; posterior view taken with detector under table

C. 15 mCi ^{99m}Tc-EHDP
Scanned 4 hr post injection
Low-Energy, All-Purpose Collimator
Speed: 48 cm/min, Length: 175 cm, Width: 60 cm
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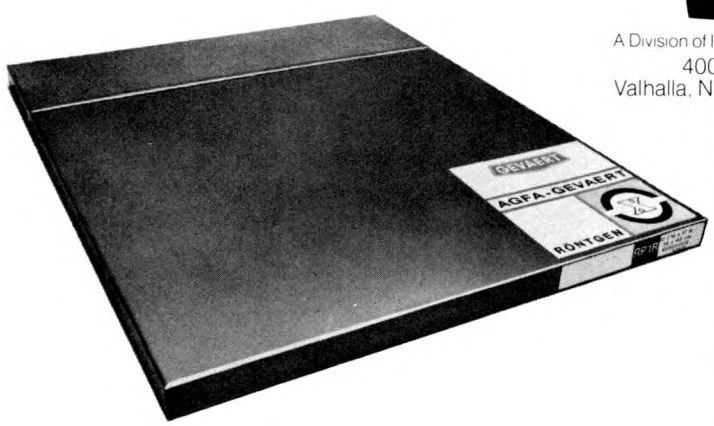
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CERVICAL ESOPHAGEAL WEBS—A ROENTGEN-ANATOMIC CORRELATION*

OBSERVATIONS ON THE PHARYNGOESOPHAGUS

By JAMES L. CLEMENTS, JR., M.D., GEORGE W. COX, M.D., WILLIAM E. TORRES, M.D., *and* H. STEPHEN WEENS, M.D.
ATLANTA, GEORGIA

WITH the advent of cineroentgenography and video tape recording, increasing attention has been given to the appearance of the pharynx and upper esophagus under normal and pathologic conditions. Present roentgenologic methods have made it possible to shed new light upon the roentgen anatomy and physiology of the pharyngoesophagus and recognize certain features which are as yet not completely understood with regard to their clinical significance.

In the roentgenologic analysis of the pharyngoesophagus, we are concerned in this study with 3 structures which may be the cause of misunderstanding or even roentgenologic misinterpretation.

1. *Webs and folds* (Fig. 1). These structures are found along the anterior aspect of the proximal pharyngoesophageal junction and esophagus as thin shelf-like defects measuring 1–2 mm. in thickness. The webs or folds protrude for various lengths into the anterior aspect of the esophageal lumen and have, on occasion, a tendency to become circumferential. Multiplicity of webs is not uncommon.

2. *The postcricoid impression* (Fig. 1). This defect is extremely variable, consisting of narrow or broad nodular or beak-like impressions in the anterior portion of the barium column immediately posterior or below the cricoid cartilage. The pronounced structural change of this defect during the act of deglutition is characteristic for this structure.

3. *The cricopharyngeal indentation* (Fig. 1). This is a rounded or tongue-like impression upon the posterior aspect of the barium column in the cervical esophagus. It has been attributed to contraction or hypertrophy of the cricopharyngeal muscle and may vary considerably in depth.

In order to contribute to further clarification of these roentgen structures, a two-fold study was initiated.

In the first part of our study, we have recorded our observations of the proximal esophagus—based on video tape recordings

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P.Q., Canada, September 25–28, 1973.
From the Department of Radiology, Emory University School of Medicine and Grady Memorial Hospital, Atlanta, Georgia.

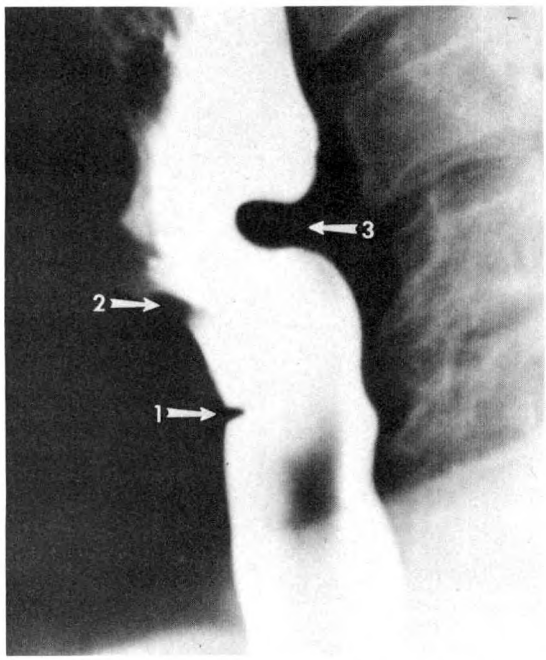


FIG. 1. Roentgenogram demonstrating the 3 esophageal structures of interest. 1. Web. 2. Postcricoid impression. 3. Cricopharyngeal indentation.

—in 100 unselected patients referred to our department with a wide variety of gastrointestinal symptoms. In many instances, these symptoms were minimal, leading to no specific roentgenologic diagnosis. This series of patients was evaluated to assess the incidence of the 3 structures mentioned above in a general hospital population.

For the purpose of gaining further pathologic-anatomic information, a second separate study was carried out. In this study, unselected anatomic material obtained from 50 autopsies was analyzed in an effort to find and examine the anatomic equivalent of cervical esophageal webs. In this second study limited attention was given to the appearance of the cricopharyngeal muscle.

MATERIAL AND METHOD

One hundred unselected patients referred to the Radiology Department for a large variety of clinical reasons were studied with video tape recording. Each patient had a contrast medium examination of the hypopharynx and cervical esophagus under fluoroscopic control during the act of deglutition. Studies were obtained in the erect lateral, frontal, and oblique projections.

In 50 unselected cadavers who came to autopsy the cervical esophagus, trachea and larynx were removed *en bloc*. The specimens were roentgenographed and the esophagus was partially and fully distended with contrast medium (Fig. 2, *A–C*). The proximal ends of the specimen were clamped close to the junction of the hypopharynx and esophagus with barium being introduced from below through a catheter placed in the occluded inferior lumen of the esophagus. Following this procedure, roentgenograms of the esophagus were obtained, and those specimens which demonstrated abnormalities were fixed in formalin for subsequent sectioning and microscopic study.

RESULTS

CLINICAL SERIES

The abnormalities detected in this study are summarized in Table 1. Webs were demonstrated in 8 patients (8 per cent). Two of the patients had a history of dysphagia. The other 6 patients gave no history of difficulty in swallowing. Postcricoid defects were seen in 71 (71 per cent) of the patients studied. Thirteen of these patients (62 per cent) had dysphagia, whereas in the group of patients with no history of dysphagia, postcricoid defects were observed in 58 (74 per cent). Cricopharyngeal indentations were seen in 18 of 100 patients (18 per cent). Only 1 of these patients had dysphagia.

ANATOMIC SERIES

Table II summarizes the pathologic changes found in the study of the anatomic specimen. Eight cases (16 per cent) demonstrated webs (Fig. 3; 4; 5; and 6). Of these cases, 5 were females and 3 were males. The ages ranged from 30 to 73 years. The principal causes of death were as follows: hemorrhage from disseminated superficial gastrointestinal ulcerations; pulmonary embo-

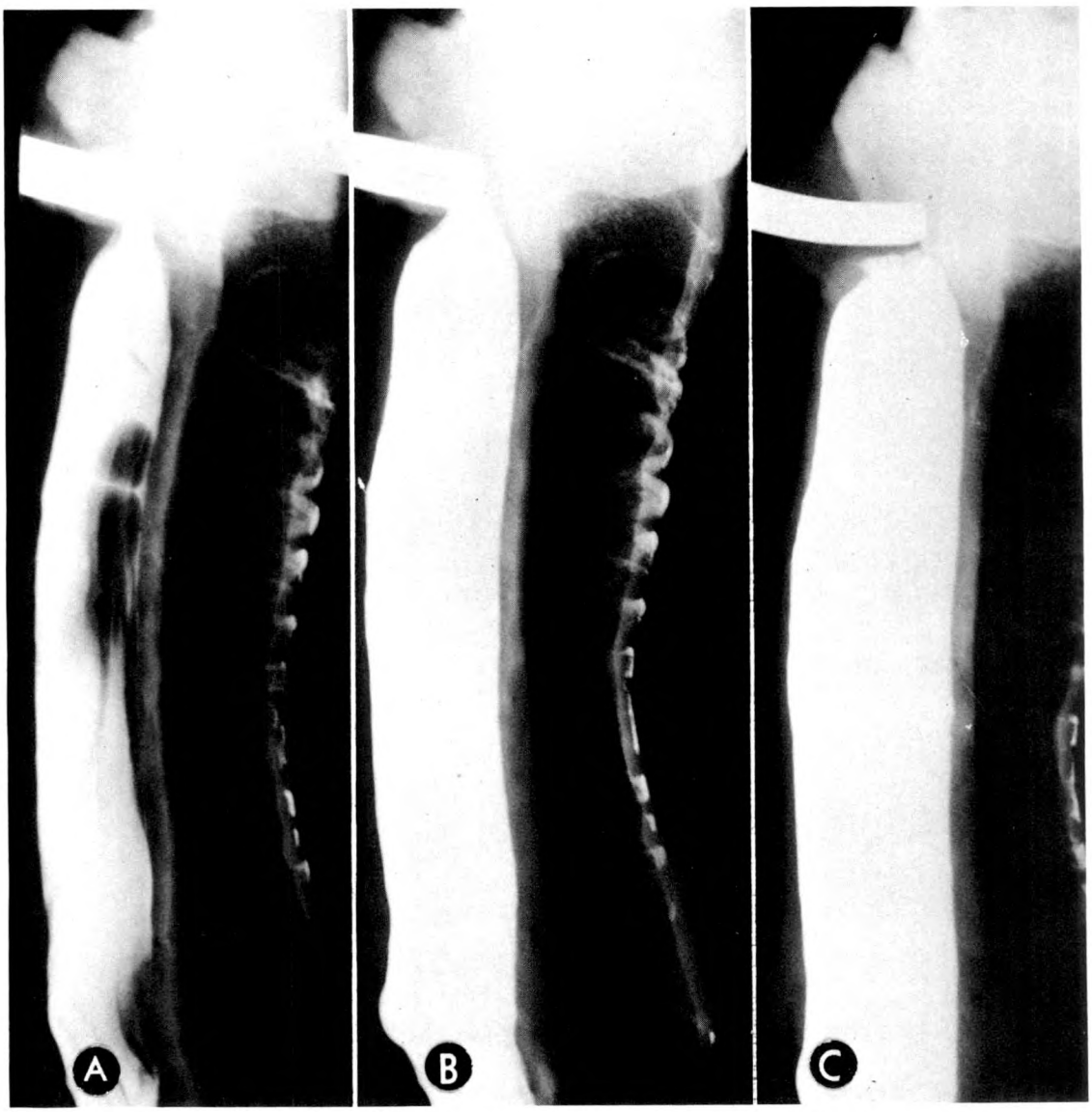


Fig. 2. (*A–C*) Roentgenograms of a normal anatomic specimen in varying stages of distention by liquid barium.

Table I

ABNORMALITIES IN CLINICAL SERIES

	Total Patients (100)	Dysphagia (21)		No Dysphagia (79)	
		No.	Per Cent	No.	Per Cent
Transverse folds	8	2	9.5	6	7.6
Cricopharyngeal dysfunction	18	1	5	17	21.5
Postcricoid defects	71	13	62	58	74

TABLE II

PATHOLOGIC CHANGES IN ANATOMIC SPECIMENS

	Dysphagia	Anemia	Other Esophageal Pathology	Plummer-Vinson	Gastrointestinal Disease
Esophageal folds (8)	0	2	2	0	1
No esophageal folds (42)	1	20	0	0	14

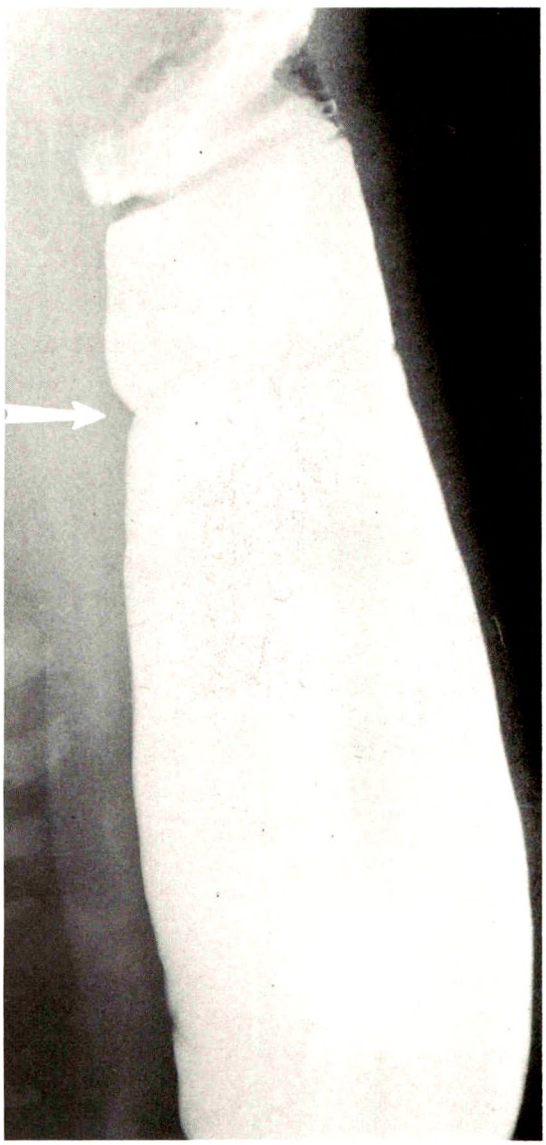


FIG. 3. Roentgenogram of anatomic specimen demonstrating a web (arrow). The larger transverse defect superiorly is an artifact.

lism; septicemia; subarachnoid hemorrhage; Laennec's cirrhosis with esophageal varices; and, in 3 cases, cardiac arrest. There was no history of dysphagia in these patients, but there was anemia in 2 by hematocrit determination. One of the patients showed other esophageal pathology (varices), but the remaining 7 patients gave no clinical history of pharyngeal, esophageal or thyroid abnormalities.

In the specimens in which no webs were observed, a history of dysphagia was noted in 1 patient and a history of anemia in 20. In 2 of the anatomic specimens the webs were no longer visible with increased distention of the esophagus. This suggests that webs may be freely movable structures



FIG. 4. Photograph of gross anatomic specimen demonstrating a large web.

which would not impede the passage of food (Fig. 7, *A–C*).

The histologic examination showed that the webs consisted of transverse folds of essentially normal mucosa and submucosa (Fig. 8; 9; and 10). None of these structures incorporated actual muscularis mucosae, although in several instances a slight tenting of the muscularis mucosae at the base of the web was noted. None of the webs showed epithelial dysplasia, or was there an inflammatory infiltration of the submucosa.

Postcricoid defects were rarely demonstrated in the anatomic study because the specimens were clamped in the area at which these defects occur.

Thirteen specimens demonstrated changes suggestive of cricopharyngeal hypertrophy, one of which contained also an esophageal web (Fig. 11, *A* and *B*). Only one of these particular specimens was studied by histologic methods (Fig. 12). This single specimen showed histologic findings consistent with cricopharyngeal hypertrophy (Fig. 13). The appearance of the cricopharyngeal muscle will be the subject of further investigation.



Fig. 5. Photograph of gross anatomic specimen demonstrating a large web. Note presence of smaller web below.

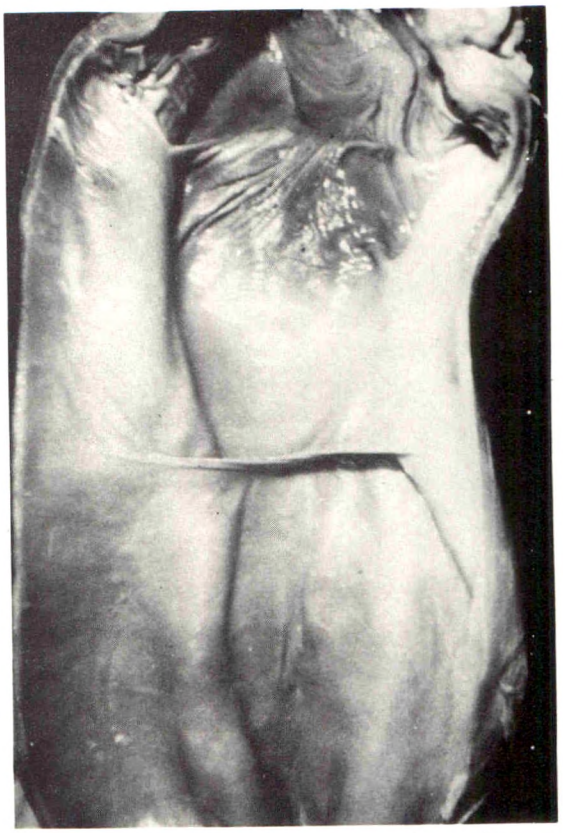


Fig. 6, Photograph of gross anatomic specimen demonstrating a large web.

DISCUSSION

WEBS AND FOLDS

In a discussion of esophageal webs, their relationship to the so-called Plummer-Vinson syndrome should be considered. Vinson[16] in 1921 presented a series of cases in which he associated the presence of anemia with postcricoid dysphagia. No roentgenographic or esophagoscopic abnormalities were noted in these patients. Vinson stated that Plummer had first recognized this relationship, and it is for this reason that the entity is usually referred to as the Plummer-Vinson syndrome in the United States. Kelly[8] and Paterson[10] in England made similar observations noting also, that

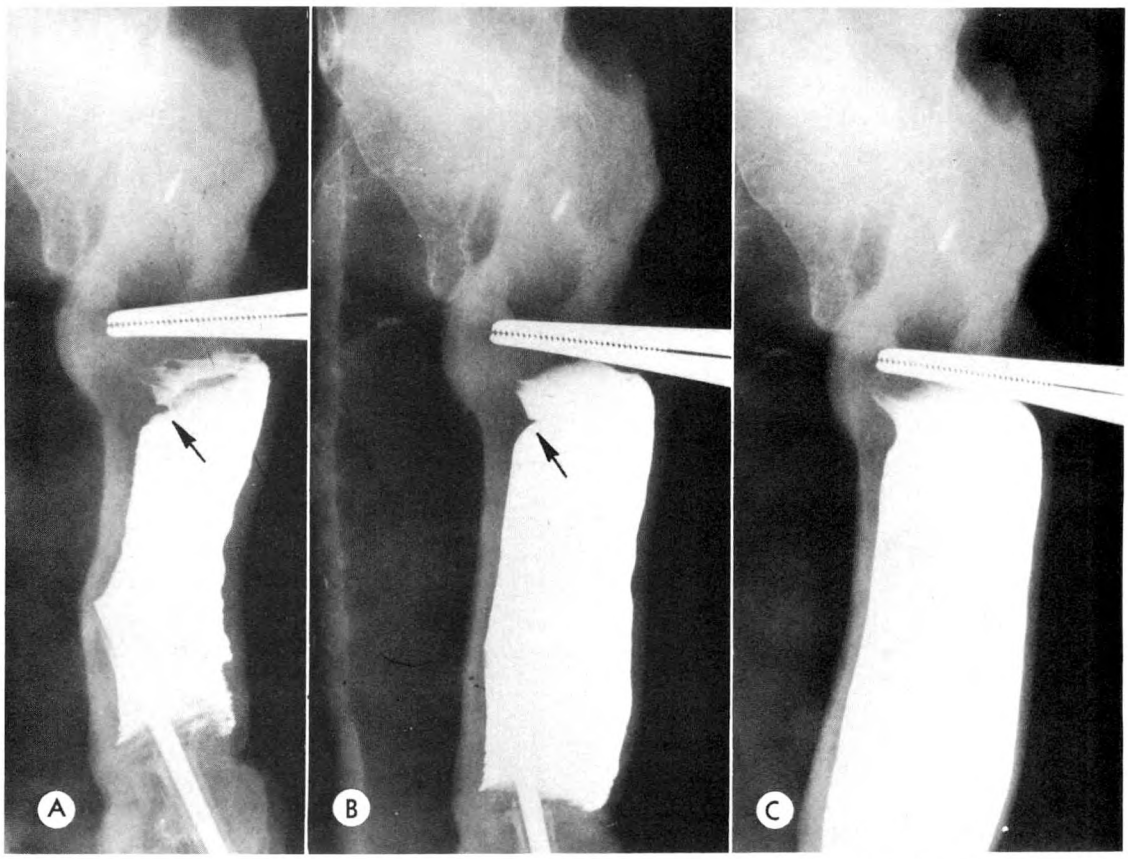


FIG. 7. (*A–C*) Roentgenograms of anatomic specimen in which increasing distention caused effacement of the web (arrows).

in their patients atrophic changes of the tongue, pharynx and upper esophagus occurred. Subsequently, Waldenström and his associates,[17,18] demonstrated iron deficiency in their patients and introduced the term sideropenic dysphagia. They were the first to demonstrate roentgenologically esophageal webs.

Gradually the spectrum of this syndrome has been widened to include glossitis, achlorhydria, spoon-shaped finger nails, splenomegaly, and spasm in the pharynx or esophagus.[13] Jones[7] stated that in order to diagnose this condition, only dysphagia with some of the accompanying signs and symptoms is necessary.

With further roentgen observations it has become evident that esophageal webs may be found in other conditions such as thyroid disease, pernicious anemia, ulcerative colitis, and certain mucosal disorders such as epidermolysis bullosa and benign mucous membrane pemphigus.[3,13] Furthermore, webs have also been observed in individuals without evident underlying disorders.

Elwood *et al.*[5] have made an important contribution to the prevalence of the Plummer-Vinson syndrome by utilizing an epidemiologic approach. Based on a study in a community of nearly 5,000 individuals in Wales, they concluded that there is no evidence of the existence in the general population of a syndrome in which dysphagia is associated with anemia. These authors pointed out that their findings would pertain only to the particular population group under study and that geographic factors and preselection of patients in hospital populations may account for the wide statistical variations found in the literature.

Nevertheless, Chisholm and associates,[2,3]

in an extensive clinical analysis, contend that there is a strong association between iron deficiency anemia and postcricoid webs. These authors realized that only a minority of patients with iron deficiency anemia will develop webs and therefore investigated other factors which could be responsible for this development. They noted that among iron deficient patients those with postcricoid webs showed a much higher incidence of stomatitis, glossitis, edentia and thyroid disease than those without webs. On the basis of laboratory investigation they suggest that autoimmune disturbances may play an important role in the development of mucosal abnormalities in iron deficient patients.

The general incidence of esophageal webs is not well known. In patients with iron deficiency anemia Chisholm and associates[3] observed an incidence of approximately 10 per cent. MacMillan[9] found esophageal webs in 7 per cent of 1,600 patients with dysphagia. Elwood *et al.*[5] recognized postcricoid webs in 16 of 104 women with dysphagia, but in none of 21 men with difficulty in swallowing.

With regard to the incidence in normal individuals, only scanty information is available. Chisholm *et al.*[3] did not find any webs in 98 healthy individuals. Elwood *et al.*[5] found postcricoid webs in less than 1 per cent of the general female population.

In the group of 100 unselected patients

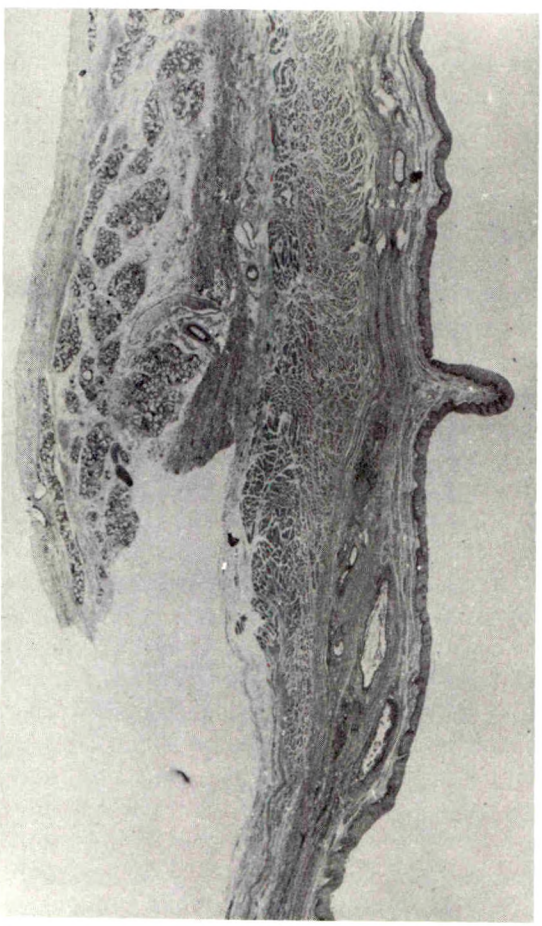


Fig. 8. Photomicrograph of web demonstrating the normal appearing mucosa and submucosa of the structure.



Fig. 9. Photomicrograph of a deep web demonstrating the normal appearing mucosa and submucosa of the structure. Note slight tenting of muscularis mucosae at the base of web.

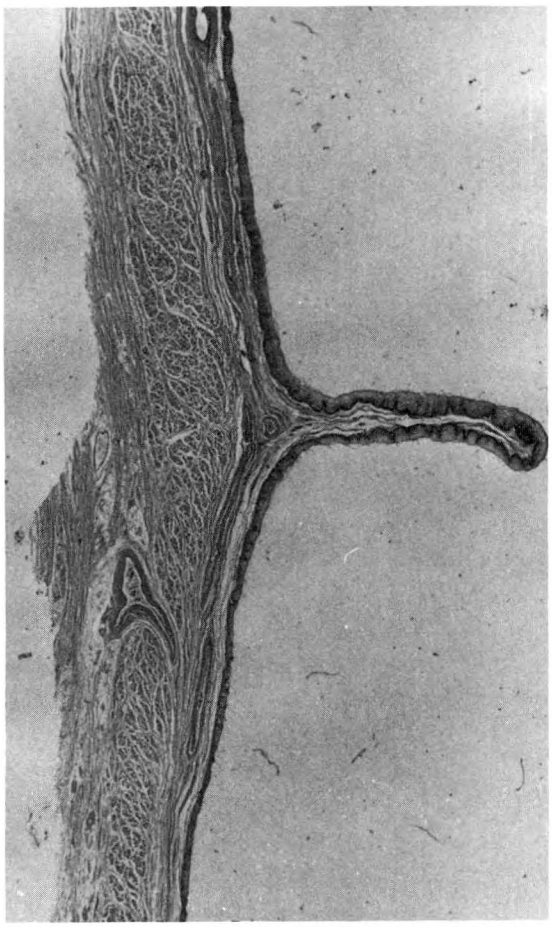


FIG. 10. Photomicrograph of a deeper web demonstrating the normal appearing mucosa and submucosa of the structure. Note slight tenting of muscularis mucosae at the base of web.

referred to our department with a large variety of gastrointestinal symptoms, webs were recognized in 8 instances (8 per cent). In our autopsy series, webs were found in 8 of 50 specimens (16 per cent). This incidence of esophageal webs was higher than we had anticipated.

It should be pointed out again that these figures pertain only to a distinct hospital population and would not be applicable to the general population or other groups of individuals. Nevertheless, these figures reflect the incidence of webs a radiologist may encounter in actual hospital practice.

Dysphagia was noted in only 2 of 8 patients with esophageal webs found in our clinical series. One of these patients had a hiatus hernia and dysphagia disappeared following surgical repair. The other patient had a carcinoma of the lower esophagus. In the anatomic series none of the 8 patients with webs had a history of dysphagia. We were, therefore, unable to find any correlation between dysphagia and the presence or absence of webs in these two series. Seaman[13] has previously expressed doubt that webs alone would be asymptomatic unless web formation was very extensive. Our findings indicate that in a general hospital population webs should be rarely the cause of dysphagia.

Most publications concerning esophageal webs in the Plummer-Vinson syndrome provide only limited information about the histologic changes in this entity. Autopsy observations stress atrophy of the mucous membrane and areas of keratinization with chronic inflammatory reaction of the submucosa.[7] Degenerative and atrophic changes of the muscular layers have also been observed.

Others[14] found on biopsy of upper esophageal webs a normal squamous cell epithelium with mild chronic inflammatory changes in the submucosa.

In 10 cases of postcricoid webs observed by Chisholm *et al.*[2] biopsies were reported as normal in 5. In the other 5 cases the common abnormality was hyperkeratosis and infiltration with inflammatory cells.

Reviewing our clinical and anatomic series we were not only impressed by the high incidence of webs, but also by the lack of mucosal changes and absence of inflammatory reaction. Although we cannot rule out in our patients a past history of iron deficiency or autoimmune disturbances, it appears likely to us that in most of our patients these webs are simple transverse folds of the mucous membrane and that they are probably not related to the Plummer-Vinson syndrome. This would imply that 2 types of web-like structures may occur in the pharyngeal esophagus. The first type would be related to epithelial changes as seen in the Plummer-Vinson syndrome or primary mucosal diseases. The second type would represent simple plication of normal mucosa.

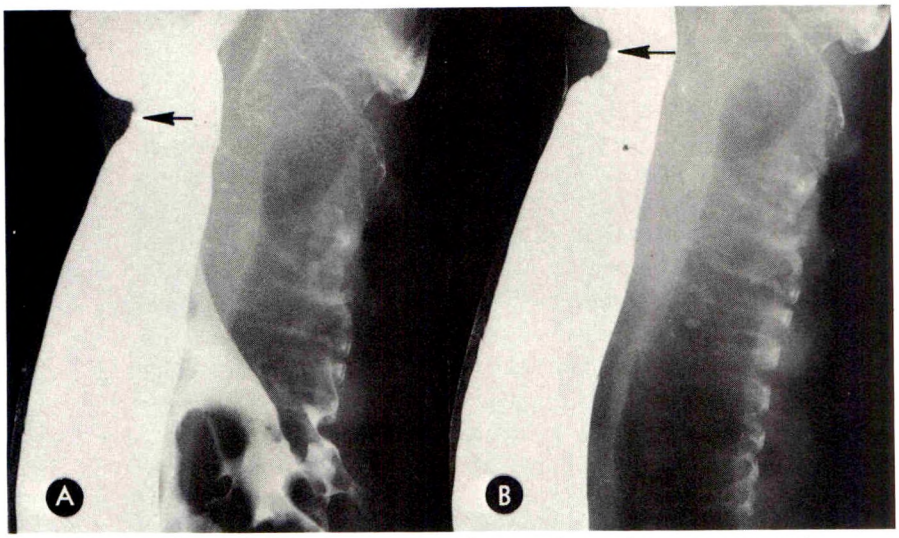


Fig. 11. (*A* and *B*) Roentgenograms of anatomic specimen demonstrating cricopharyngeal indentation (arrows).

THE POSTCRICOID IMPRESSION

This defect has been described and studied in detail by Pitman and Fraser.[11] These authors referred to previous work by Butler on esophageal veins and performed injection studies of their own. They postulated that this impression could be ascribed to the laxity of the mucous membrane over the normally varicose submucosal veins in the postcricoid region. They recognized the postcricoid indentation in 86 per cent of patients with dysphagia and in 90 per cent of control subjects without dysphagia. Seaman[13] noted that this defect could be observed with considerable frequency and considered it also as a normal variant.

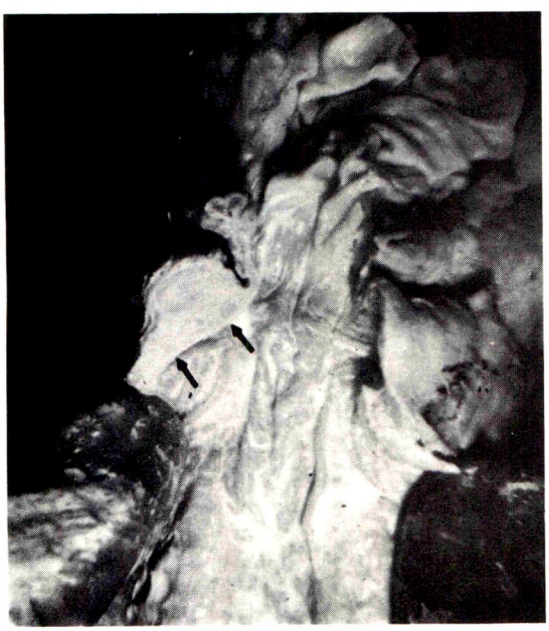


Fig. 12. Photograph of gross specimen demonstrating cricopharyngeal hypertrophy (arrows).

In our clinical observation, the postcricoid defect was found in 62 per cent of patients with and 74 per cent of patients without dysphagia (Table 1). In our anatomic studies, repeatedly considerable laxity and flexibility of the postcricoid mucosa was noted which was often cast into fairly prominent transverse folds. Inasmuch as we did not conduct injection studies of the venous plexus, we cannot state that the varicosities are essential for creation of the defects or that the laxity of the mucous membrane alone could produce this roentgen phenomenon.

We are in full agreement with Pitman and Fraser[11] as well as Seaman[13] that the postcricoid indentation is a normal variant and not related to any particular disorder.

THE CRICOPHARYNGEAL INDENTATION

A posterior indentation of the esophageal lumen is often observed at the level of the lower cervical spine. This impression has been commonly referred to as esophageal

lip[15] or spasm or hypertrophy of the cricopharyngeal muscle. Most observers agree that this esophageal narrowing is caused by contraction of the transverse fibers of this muscle. Nevertheless, divergence of opinion remains as to the pathologic and clinical significance of this roentgenologic defect.

In the resting condition, the cricopharyngeal muscle is in a stage of tonic contraction, assuring closure of the proximal esophagus and separation of the respiratory system from the digestive tract. On deglutition, after initiation of pharyngeal peristalsis, the cricopharyngeal muscle relaxes, permitting the advancing food bolus to enter the esophagus without resistance. Seaman[12] has postulated that incoordination of pharyngoesophageal motility may suffice to explain this posterior esophageal defect and that it is not necessary to attribute the cricopharyngeal indentation to spasm or muscle hypertrophy. If the cricopharynx fails to open at the proper time or if it contracts prematurely, a defect in the barium column would be readily apparent.

It is noteworthy that most often patients with this defect show no evidence of dysphagia. However, if the defect is more pronounced and accompanied by difficulty in swallowing, this entity is usually referred to as cricopharyngeal achalasia.

In 50 patients without dysphagia, Siebert *et al.*[15] found mild cricopharyngeal indentation in 29 instances and a more pronounced defect in 7 cases. Therefore, this group of observers as well as Johnstone[6] believe that the cricopharyngeal indentation is probably of no pathologic significance. On the other hand, prominence of the cricopharyngeal muscle has been repeatedly observed in patients with primary muscle disorders, vagal paralysis and postsurgical states such as laryngectomy.[12] Wilkins[19] and Blakeley *et al.*[1] have shown that myotomy may relieve dysphagia in this group of patients.

Crichlow[4] as well as Seaman[12] consider the cricopharyngeal indentation as a pathologic phenomenon reflecting neuromuscular dysfunction. The latter author believes that patients may adapt themselves to abnormal swallowing and therefore may not display manifestations of dysphagia. This author recognized this defect in approximately 5 per cent of his patients.

In our video tape studies of 100 unselected patients, cricopharyngeal indentations were observed in 18 instances. Only 1 of the cases with this roentgenologic defect had dysphagia; the remaining 17 cases had no difficulty in swallowing. In contrast, there were 20 patients with dysphagia in whom such a defect could not be demonstrated (Table 1).

Our roentgenologic-pathologic studies of cadaver specimens showed frequently a shallow indentation of the barium column

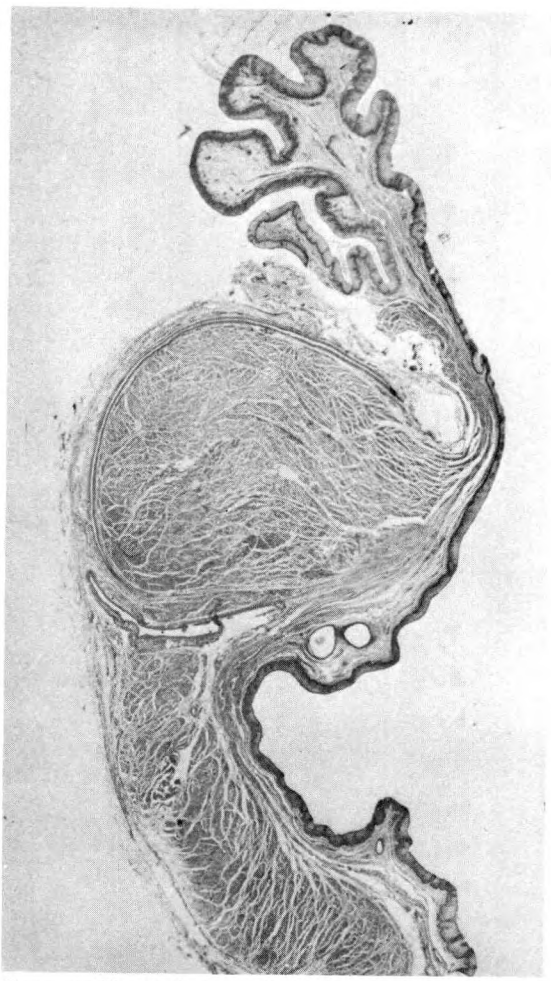


FIG. 13. Photomicrograph of section taken through hypertrophied cricopharyngeus seen in Figure 12.

at the level of the cricopharyngeal muscle. Inasmuch as our studies were largely directed towards the recognition of esophageal webs, a dissection of the cricopharyngeal muscle was not carried out. However, in 1 specimen (Fig. 13) the histologic examination showed a pronounced hypertrophy of the cricopharyngeal muscle.

Reviewing these observations it is conceivable that shallow cricopharyngeal indentations such as recorded by Siebert *et al.*[15] in more than half of their asymptomatic patients carry probably no pathologic significance. The more pronounced lip-, tongue- or shelf-like indentations may reflect cricopharyngeal abnormality even in the absence of clinical symptoms.

SUMMARY

The incidence of pharyngoesophageal webs in 100 unselected patients with a large variety of gastrointestinal symptoms and in autopsy observations of 50 unselected cases was 8 and 16 per cent, respectively.

Histologic studies revealed that the webs observed on autopsy consisted of transverse folds of normal mucosa and submucosa. There were no muscular atrophy, keratinization, or inflammatory changes observed as are seen in the classic Plummer-Vinson syndrome.

Esophageal webs appear to be common in a hospital population. This type of web is probably of no clinical significance as it produces no clinical symptoms.

The incidence and clinical significance of 2 other structures of the pharyngoesophagus, the postcricoid impression and the cricopharyngeal indentation, are briefly discussed.

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REFERENCES

1. Blakeley, W. R., Garety, E. J., and Smith, D. E. Section of cricopharyngeus muscle for dysphagia. *A.M.A. Arch. Surg.*, 1968, *96*, 745–760.
2. Chisholm, M., Ardran, G. M., Callender, S. T., and Wright, R. A. Follow-up study of patients with post-cricoid webs. *Quart. J. Med.*, 1971, *40*, 409–420.
3. Chisholm, M., Ardran, G. M., Callender, S. T., and Wright, R. Iron deficiency and autoimmunity in post-cricoid webs. *Quart. J. Med.*, 1971, *40*, 421–433.
4. Crichlow, T. V. L. Cricopharyngeus in radiography and cineradiography. *Brit. J. Radiol.*, 1956, *29*, 546–556.
5. Elwood, P. C., Jacobs, A., Pitman, R. G., and Entwistle, C. C. Epidemiology of Paterson-Kelly syndrome. *Lancet*, 1962, *2*, 716–720.
6. Johnstone, A. S. Non-malignant conditions of oesophagus. *Brit. J. Radiol.*, 1946, *19*, 101–106.
7. Jones, R. F. McN. Paterson-Brown-Kelly syndrome. *J. Laryng. & Otol.*, 1961, *75*, 529–561.
8. Kelly, A. B. Spasm of entrance to oesophagus. *J. Laryng.*, 1919, *34*, 285–289.
9. MacMillan, A. S. Statistical study of diseases of esophagus. *Surg., Gynec. & Obst.*, 1935, *60*, 394–402.
10. Paterson, D. R. Clinical type of dysphagia. *J. Laryng.*, 1919, *32*, 289–291.
11. Pitman, R. G., and Fraser, G. M. Post-cricoid impression on oesophagus. *Clin. Radiol.*, 1965, *16*, 34–39.
12. Seaman, W. B. Cineroentgenographic observations of cricopharyngeus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *96*, 922–931.
13. Seaman, W. B. Significance of webs in hypopharynx and upper esophagus. *Radiology*, 1967, *89*, 32–38.
14. Shamma'a, M. H., and Benedict, E. B. Esophageal webs: report of 58 cases and attempt at classification. *New England J. Med.*, 1958, *259*, 378–384.
15. Siebert, T. L., Stein, J., and Poppel, M. H. Variation in roentgen appearance of "esophageal lip." Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *81*, 570–575.
16. Vinson, P. O. Hysterical dysphagia. *Minnesota Med.*, 1921, *5*, 107–108.
17. Waldenström, J., and Hallen, L. Iron and epithelium: some clinical observations. *Acta med. scandinav.*, 1938, Suppl. 90, 380–405.
18. Waldenström, J., and Kjellberg, S. R. Roentgenological diagnosis of sideropenic dysphagia (Plummer-Vinson's syndrome). *Acta radiol.*, 1939, *20*, 618–638.
19. Wilkins, S. A. Indications for section of cricopharyngeus muscle. *Am. J. Surg.*, 1964, *108*, 533–538.

ENHANCED VISUALIZATION OF ESOPHAGEAL VARICES BY BUSCOPAN*

By CHUNG-I LIU, M.D.
TAIPEI, TAIWAN

VISUALIZATION of small esophageal varices by conventional roentgenographic methods may be very difficult. Although various efforts such as the use of Trendelenburg position with Valsalva or Mueller maneuvers,[6] dextran infusion,[8] esophageal balloon,[5] and cineradiography[1,3] have been tried, successful visualization of esophageal varices still varies from 20 to 70 per cent.[4] Dalinka *et al.*[2] reported an unequivocal demonstration of esophageal varices by intravenous or intramuscular administration of 30 mg. Pro-Banthine in 9 of 17 patients, in whom varices were not delineated by the conventional roentgenographic methods. Since Pro-Banthine is contraindicated in patients with glaucoma and must be used with caution in patients having cardiac disease or prostatic hypertrophy,[2] the author has introduced Buscopan as an alternative enhancement agent.

The purpose of this report is to present the experience with the use of Buscopan in an attempt to enhance the visualization of esophageal varices.

MATERIAL AND METHOD

Twenty-five patients with a clinical history suggesting cirrhosis of the liver with normal or equivocal findings of esophageal varices in the conventional esophagogram were studied.

After the conventional esophagography, a mouthful of thick barium swallow was given and 20 mg. Buscopan was administered intravenously. When the esophageal peristalsis ceased and the esophagus dilated (usually within 2 to 3 minutes), multiple spot-roentgenograms were made both in the supine and prone oblique projections. No Valsalva or Mueller maneuver was needed during fluoroscopy or roentgenography.

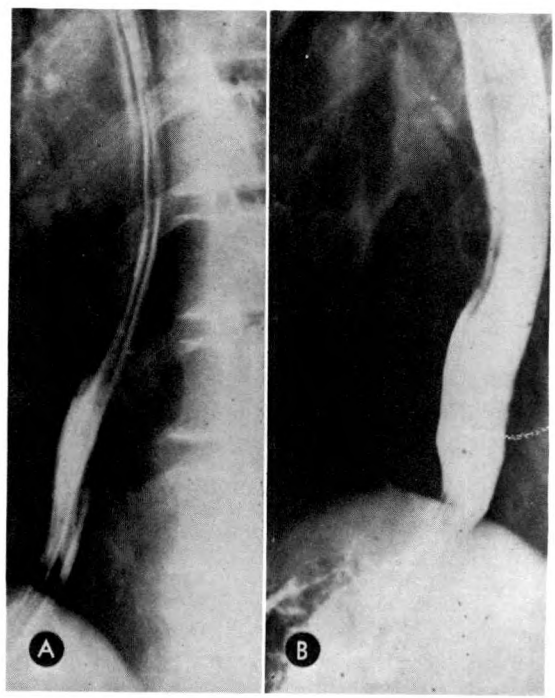


FIG. 1. Normal esophagograms (*A*) by conventional esophagography and (*B*) by the enhanced method.

RESULTS

In 25 patients with normal or equivocal findings of esophageal varices, suggested by conventional esophagography, esophageal varices were clearly demonstrated by the enhanced method in 14 cases (Fig. 2, *A–D*; 3, *A–C*; and 4, *A–D*). The remaining 11 patients with negative findings (Fig. 1, *A* and *B*) were later proven to have hepatoma (1), thalassemia (1), duodenal ulcer (1), and subacute hepatitis (4); 4 patients had either normal liver function, normal liver scan or a negative needle biopsy.

DISCUSSION

The most frequent causes of failure to delineate small esophageal varices by con-

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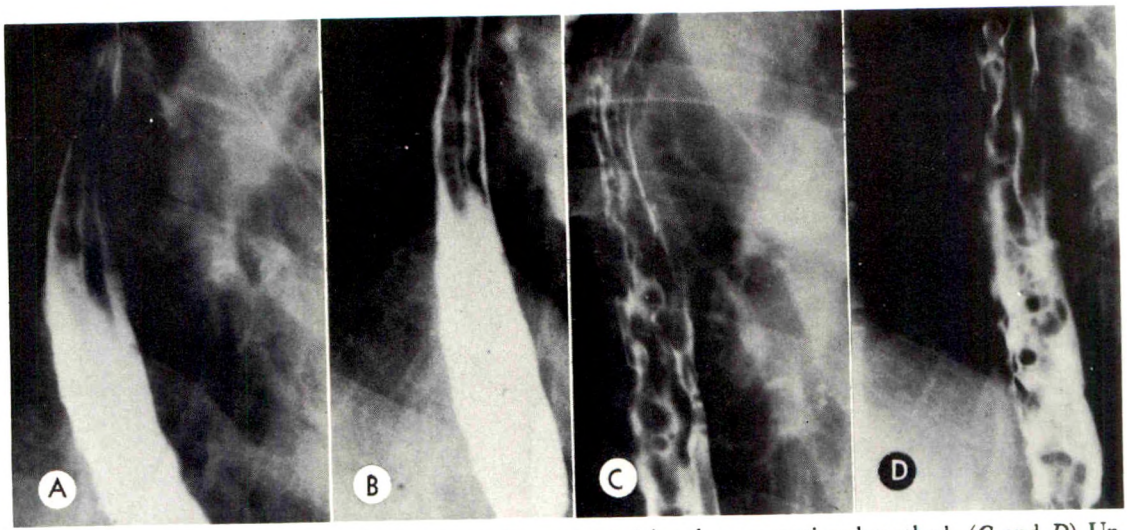


Fig. 2. (*A* and *B*) No esophageal varices are demonstrated by the conventional method. (*C* and *D*) Unequivocal esophageal varices are delineated by the enhanced method.

ventional esophagography were the result of active esophageal peristalsis and high muscular tonicity of the esophagus. These factors tend to force the blood out of the loosely supported submucosal esophageal varicosities and render them roentgenologically invisible.[6] Buscopan acts on the parasympathetic ganglia in the walls of the viscera, and it exerts a specific antispasmodic action on the smooth muscle of the

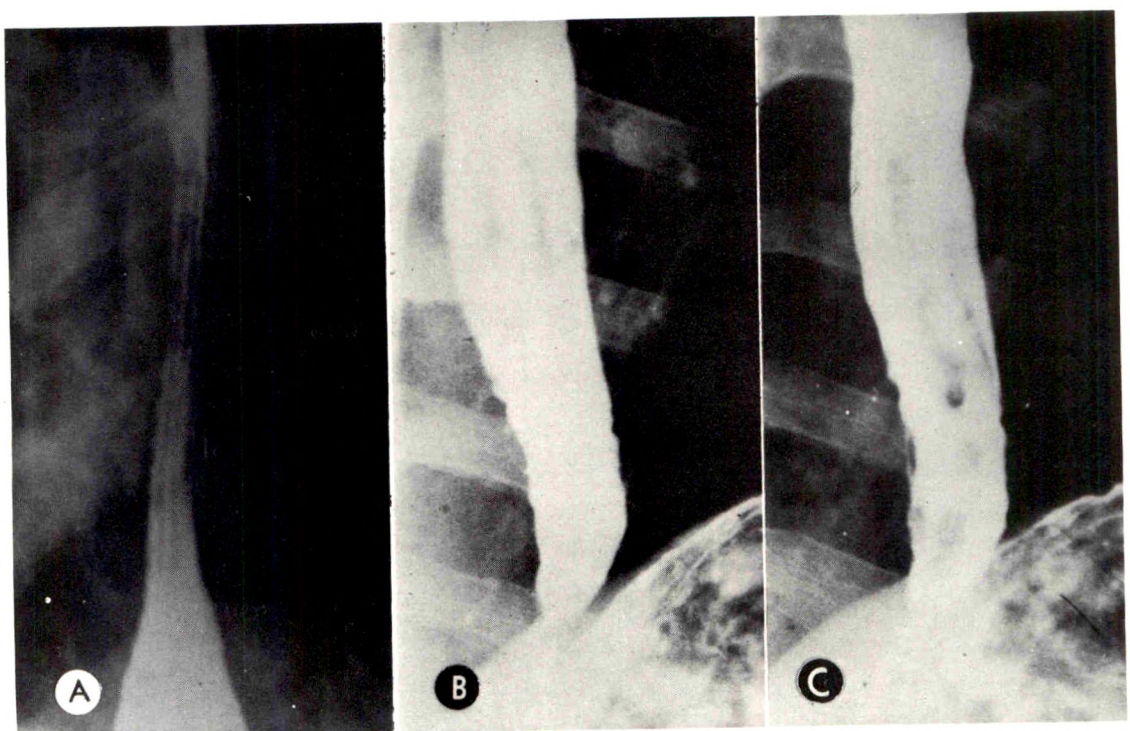


Fig. 3. (*A*) Conventional esophagogram shows normal or equivocal esophageal varices. (*B* and *C*) Enhanced esophagograms reveal unequivocal esophageal varices.

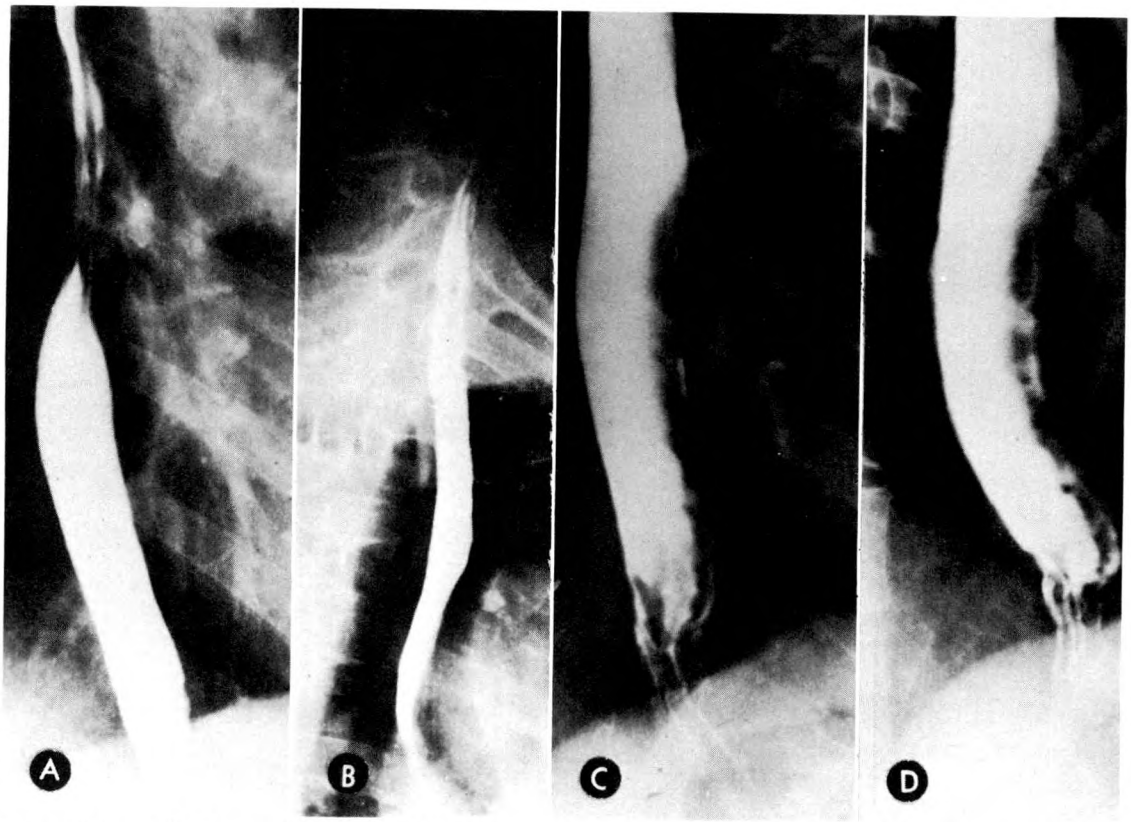


FIG. 4. (*A* and *B*) Conventional method shows normal esophagograms. (*C* and *D*) Enhanced method demonstrates esophageal varices.

gastrointestinal tract. Thus, after an intravenous administration, it abolishes the esophageal peristalsis and causes relaxation and dilatation of the esophagus. The varices fill with blood and become visible, if an optimum amount of barium remains in the lumen.

Buscopan has no side-effects on the central nervous system, the eyes, the salivary glands or the heart, when it is given in therapeutic doses.[7] Thus, it is much safer than Pro-Banthine.

The enhanced method is inexpensive and easy to perform. It causes very little discomfort and provides minimum risk to the patient. It requires only minimal radiation exposure of the patient as well as of the examiner, and takes very little time. No special effort such as the Valsalva maneuver is needed. In our series, no single false negative or false positive case was recorded, so that the accuracy of this enhanced method is almost 100 per cent.

CONCLUSION

Twenty-five patients, with a clinical history suggesting cirrhosis of the liver with normal or equivocal findings of esophageal varices, were studied by the intravenous administration of 20 mg. of Buscopan.

Unequivocal esophageal varices were demonstrated in 14 patients. The 11 patients with negative findings were later proven to have no evidence of cirrhosis of the liver.

This enhanced method is safe, inexpensive, easy to perform, and causes no discomfort or risk to the patient. Its accuracy is almost 100 per cent.

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REFERENCES

1. Adler, D. C., Haverback, B. J., and Meyers, H. I. Cineradiography of esophageal varices. *J.A.M.A.*, 1964, *189*, 77–80.
2. Dalinka, M. K., Smith, E. H., Wolfe, R. D., Goldenberg, D., and Langdon, D. E. Pharmacologically enhanced visualization of esophageal varices by Pro-Banthine. *Radiology*, 1972, *102*, 281–282.
3. Feist, J. H., and Riley, R. R. Diagnosis of esophageal varices: improvement of diagnostic accuracy by cinefluorography. *Radiology*, 1969, *93*, 861–866.
4. Kirsh, I. E., Blackwell, C. C., and Bennett, H.D. Roentgen diagnosis of esophageal varices: comparison of roentgen and esophagoscopic findings in 502 cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *74*, 477–485.
5. Nathan, M. H. Diagnosis of esophageal varices by new radiologic method: preliminary report. *Radiology*, 1959, *73*, 725–731.
6. Nelson, S. W. Roentgenographic diagnosis of esophageal varices. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *77*, 599–611.
7. Philippot, E., and Dallemagne, M. J. A propos de l'action curarisante du butylscopolammonium (Buscopan). *Arch. internat. pharmacodyn.*, 1953, *93*, 337–340.
8. Preger, L. Enhanced visualization of esophageal varices by dextran infusion: preliminary report. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 468–471.

NONOBSTRUCTIVE MUCOSAL DIAPHRAGMS OR RINGS OF THE GASTRIC ANTRUM IN ADULTS*

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THE occurrence of an antral diaphragm in an adult patient was first described in 1949 by Sames.[23] Felson *et al.*[7] reviewed the literature in 1969 and found 35 published cases. Their own 4 patients and 2 others bring the total number of reported adults with antral mucosal diaphragm to 41.[4,7,10] The majority of these patients had prepyloric obstruction and/or peptic ulcers requiring surgical intervention.[2,6,7,9,10,16,18,20,23] The diagnosis of an antral diaphragm, however, may be difficult both at laparotomy and inspection of resected stomach unless the lesion is suspected on preoperative roentgenologic or gastroscopic examinations.[1,6,10,16,22]

The clinical manifestation of the antral diaphragm usually correlates with the size of the central aperture of the mucosal septum.[2,10,16,18] Therefore, nonstenotic forms of the lesion may be asymptomatic or produce mild and nonspecific upper abdominal discomfort late in life.[7,12,15,22]

Our recent experience with 6 cases of nonobstructive diaphragms or rings of the antrum suggests that this abnormality is more common than the scarcity of reports would indicate. These lesions are probably often overlooked or misinterpreted even in those patients who do undergo barium examination of the gastrointestinal tract.[2,7,10,12,18,22]

Therefore, it seems appropriate to: (a) describe the characteristic roentgenologic and gastroscopic presentations of nonobstructive antral diaphragm; (b) review the pertinent clinical and pathologic features of the previously reported cases; (c) provide technical guidelines for demonstration and differentiation of the lesion; and (d) emphasize the coexistence and similarities of the Schatzki's ring of the esophagus and the antral rings in our patients.

MATERIAL AND METHOD

The series consists of 6 patients seen over a 3 year period at the Hospitals of the University of Chicago and the Medical College of Virginia. The age, sex and pertinent roentgenologic findings of these patients are summarized in Table 1.

Five of the patients had several years' history of intermittent upper abdominal discomfort usually manifested as belching, bloating, and postprandial epigastric pain. These patients each had 2 or more previous upper gastrointestinal series. However, the diagnosis of an antral diaphragm had not been made on the earlier examinations, although the abnormality was evident retrospectively in 2 instances. Only in Case v were the previous roentgenograms and clinical records not available for review. In Cases I and II the antral diaphragm was first recognized during performance of an anticholinergic-aided hypotonic gastroduodenal examination.[8]

The patients have been followed for a period of 6 to 32 months, each having at least 2 repeat examinations confirming persistence of the abnormality. In addition, cinefluorographic documentation of the antral diaphragm was obtained in 2 cases.

Four of the 6 cases had an associated sliding hiatal hernia (Table 1). This was manifested roentgenographically by a Schatzki's ring in 3, and by a patulous gastroesophageal junction and reflux in

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TABLE I

SUMMARY OF CASES

Case	Age and Sex	Measurement (mm.) of the Antral Diaphragms*				Associated Roentgenologic Findings in Upper Gastrointestinal Examination
		Thickness	Distance from Pylorus	Diameters of the Ring		
				Aperture	Base	
I.	66 F	2.5	45	20	37	Hiatal hernia, Schatzki's ring
II.	41 M	2.0	35	24	50	Hiatal hernia, reflux, deformed bulb
III.	43 M	2.0	35	25	35	Duodenal ulcer
IV.	50 F	3.5	70	20	32	Hiatal hernia, Schatzki's ring
V.	65 F	2.0	40	20	35	Hiatal hernia, Schatzki's ring
VI.	28 F	2.5	30	22	40	Duodenal ulcer

* Average measurement on 3 spot film roentgenograms without correction for magnification.

1. Two patients had duodenal ulcer, and a third patient presented with markedly deformed duodenal bulb (Table 1). However, except in Case 1 in whom a superficial ulceration was seen at gastroscopy, no gastric ulcer has ever been demonstrated roentgenographically in our cases.

Gastroscopic examination was performed in 3 patients including Case 1, in whom the lesion was photographed on 2 occasions (Fig. 2, *A* and *B*). To date, no gastric surgery has been performed on these patients because the nonobstructive nature of antral diaphragm and nonspecificity of symptoms did not justify the operative risk. However, 2 patients had previous abdominal operations: cholecystectomy and repair of a ventral hernia in Case 1, and cholecystectomy in Case II. No reference to any gastric abnormality was made in the operative reports of these patients.

It should be emphasized that 5 of these patients had prolonged clinical history of postprandial epigastric pain variously attributed to sliding hiatal hernia, gastritis or peptic ulcers. Furthermore, Case II had alcoholic cardiomyopathy, recurrent pancreatitis and duodenal ulcer, the latter resulting in marked deformity of the duodenal bulb over a period of 12 years (Fig. 3, *A* and *B*). The complicated clinical history of our cases is similar to that of previously reported adult patients with antral diaphragm. Therefore, only the following detailed case history has been selected and is presented as an example.

REPORT OF A CASE

CASE 1. This 66 year old Black female was admitted to the University of Chicago Hospitals for the third time in February 1971. Her complaints were epigastric burning sensation, bloating, nausea and backpain. The symptoms had been present intermittently for about 10 years, but increasing in the past 3 months, while she had been taking up to 3.0 grams of Aspirin per day for osteoarthritis of the spine. She was known to have mild diabetes, being treated with 20 Units of Insulin daily.

Her past clinical history revealed that in 1950 she had undergone repair of a small ventral hernia in another hospital. An upper gastroduodenal series done at that time was reported as normal. In 1956 the patient was admitted to the University of Chicago Hospitals with 1 year history of recurrent episodes of severe but noncolicky epigastric pain. The pain radiated to the subscapular regions, did not relate to meals, and had not responded to antacids. An oral cholecystogram demonstrated multiple calcified gallstones. The barium examination of the gastrointestinal tract was considered normal. Subsequently, cholecystectomy was performed for chronic cholecystitis and gallstones. Following surgery, the patient's symptoms diminished, requiring only occasional treatment with phenobarbital and belladonna.

Prior to her third hospital admission in 1971 a gastroduodenal series was obtained because of the above described symptoms. This examina-

tion demonstrated a Schatzki's ring of the distal esophagus with a sliding hiatal hernia without reflux. The second part of the duodenal loop was redundant and appeared indented laterally by an extrinsic mass. To better evaluate the latter finding a "tubeless" hypotonic duodenography was performed following intramuscular injection of 30 mg. of propantheline bromide. The study did not reveal any abnormality in the duodenum; however, a persistent ring-like deformity was noted in the prepyloric region (Fig. 1, *A–C*). This antral ring or diaphragm was located 4.5 cm. proximal to the pylorus, had 2–3 mm. thickness and a central aperture of nearly 2 cm. diameter. The lesion was retrospectively apparent on one spot roentgenogram of the examination done in January 1956.

An upper gastrointestinal endoscopy performed 2 weeks later demonstrated Schatzki's ring, atrophic gastritis and a 3 mm. mucosal erosion in the prepyloric region. The presence of the antral ring was not appreciated during the procedure, although the obtained Gastrocamera pictures clearly showed the lesion corresponding to its roentgenographic location and appearance (Fig. 2*A*). At this time the patient's symptoms were considered to be mainly a result of excessive Aspirin ingestion. Therefore, she was placed on milk, bland diet, antacids and small doses of Darvon. A gastroduodenal series in April 1971 demonstrated no change in the previously described findings. The repeat gastroscopy confirmed presence of a nonobstructive antral diaphragm with slight erythematous margins of its central aperture (Fig. 2*B*). No ulcer was recognized on this occasion, although the endoscopic diagnosis of atrophic gastritis was maintained. A subsequent gastric juice analysis revealed no free acid but the response to Histalog-Test was positive.

During the past 2½ years the patient has been asymptomatic following instructions on dietary measures and proper mastication of solid food. However, a recent upper gastrointestinal series has re-demonstrated the antral diaphragm and the Schatzki's ring.

ANATOMY

Antral mucosal diaphragm is a thin septum projecting perpendicular to the longitudinal axis of the antrum. It is 2–4 mm. in thickness, and located 1.5 to 7 cm. proximal to the pylorus.[2,7,10,22] Histologically, the lesion consists of 2 layers of normal gastric mucosa with a common submucosa and muscularis mucosae. In some cases there has been evidence for nonspecific inflammatory reaction without fibrosis.[7,26,28] The serosal and muscular coats of gastric wall do not participate in the formation of antral diaphragm. However, some degree of muscular hypertrophy may develop proximal to the partially obstructive lesions.[6,23] Antral diaphragm does not produce any change on the external surface of the stomach, although in one instance an ill-defined superficial groove was noted.[23]

In the majority of reported cases the central aperture had a diameter of only a few millimeters.[2,7,16,22] This opening cannot be dilated without tearing.[18] In our cases and a few others the orifice was 1 cm. or more (Table 1).[12,28] However, complete diaphragm without an opening has been reported in infants who present with gastric outlet obstruction soon after birth.[5,29]

ETIOLOGIC CONSIDERATIONS

The etiology of antral diaphragm has remained controversial. A congenital origin has been postulated based on occurrence of the lesion in infants or children,[5,16,29] and the usual absence of inflammatory changes.[10,12,15,18] The reason for clinical manifestation of such a lesion first in adulthood has been attributed to progressive narrowing of the aperture due to recurrent marginal erosions, or mucosal edema due to gastritis.[2,12,15,22] Additional factors for delayed appearance of symptoms are said to be poor mastication and diminished gastric peristaltic force with advancing age.[2,7,12,18,23,28]

Other authors suggest that antral diaphragm, at least in adults, represents an acquired lesion secondary to healing of linear or circumferential ulcers.[6,19,20] This hypothesis is supported by:

a. Concurrent demonstration of antral diaphragm and gastric ulcers or mucosal inflammation, both in children and adults.[6,7,18,20,22,26,28]
b. Absence of the lesion on previous upper gastrointestinal series of some of

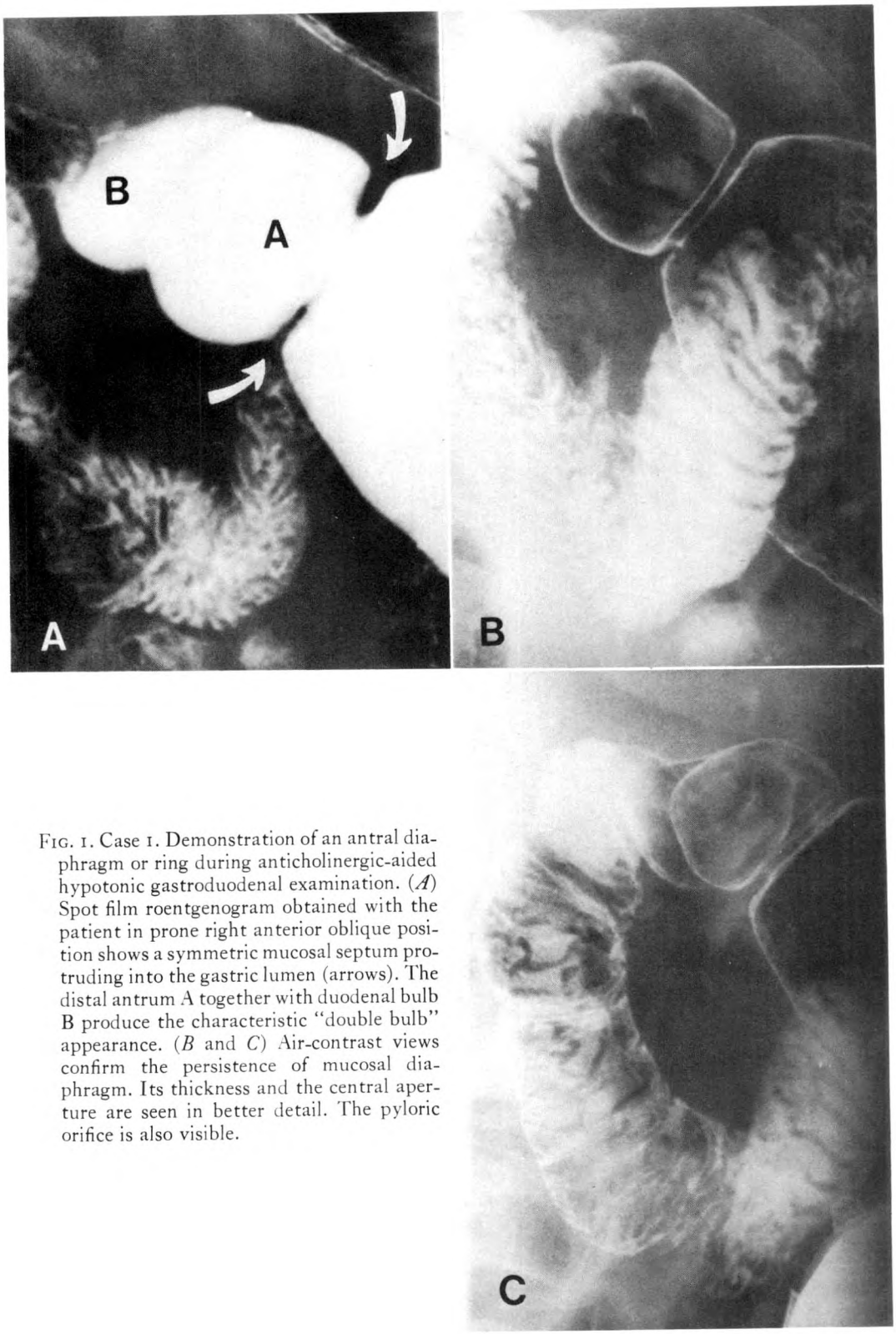


Fig. 1. Case 1. Demonstration of an antral diaphragm or ring during anticholinergic-aided hypotonic gastroduodenal examination. (*A*) Spot film roentgenogram obtained with the patient in prone right anterior oblique position shows a symmetric mucosal septum protruding into the gastric lumen (arrows). The distal antrum A together with duodenal bulb B produce the characteristic "double bulb" appearance. (*B* and *C*) Air-contrast views confirm the persistence of mucosal diaphragm. Its thickness and the central aperture are seen in better detail. The pyloric orifice is also visible.

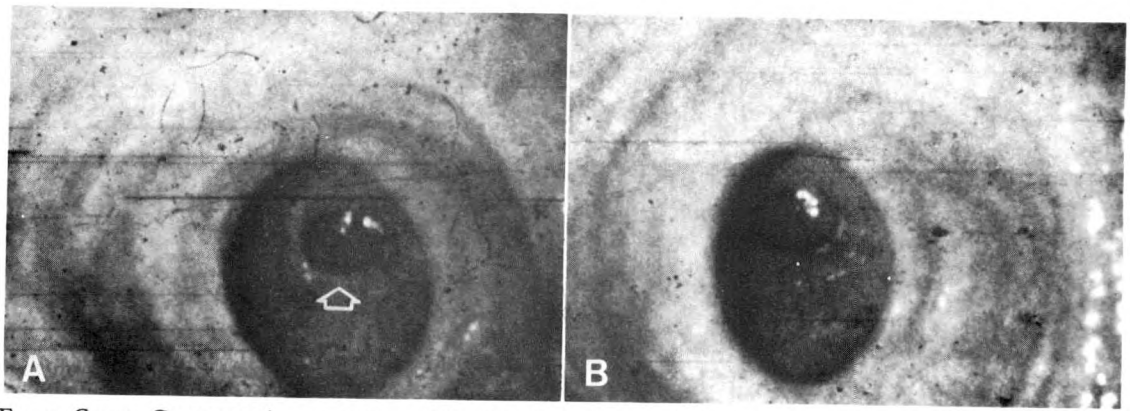


FIG. 2. Case 1. Gastroscopic appearance of the antral diaphragm in Case 1. (*A*) Initial examination in February 1971 shows intraluminal projection of the mucosal diaphragm with a large central opening through which the pyloric orifice (arrow) is seen. (*B*) A comparable Gastrocamera picture from the study in April 1971 shows an unchanged configuration of the lesion.

our patients and others in whom a diaphragm was subsequently recognized.[7,22]

c. Reported development of an obstructive mucosal diaphragm in the pyloric region following gastroduodenostomy.[20]
d. Morphologic resemblance of antral diaphragms to the pyloric and postbulbar diaphragms complicating peptic ulcers.[3,19,20]

CLINICAL FEATURES

The symptoms of these patients depend on the degree of gastric obstruction due to small diaphragmatic aperture. Most of the reported cases have had several years history of recurrent epigastric pain, bloated sensation, nausea and vomiting occurring shortly after the ingestion of food.[2,7,10,18] These manifestations of gastric outlet obstruction, together with anorexia and weight loss may bring the patient to medical attention under suspicion of a pyloric carcinoma.[18,22] More commonly, the clinical and roentgenologic findings lead to erroneous diagnosis of peptic ulceration with spasm or deformity of the antrum.[2,6,7,12,18,22] The subjective response of some patients to medication of antacids, antiemetics, and dietary measures may even delay the correct diagnosis for many years.[18,22,23]

Our patients and a few previous cases did not have gastric retention.[2,9,12,15] One can reasonably assume that a diaphragmatic opening equal or larger in size than the pyloric orifice (*e.g.*, 1.0 cm.) would not be obstructive to the passage of normally digested gastric content. The fact that these patients are nevertheless symptomatic can be explained by 2 observations:

a. Most patients are older edentulous individuals who do not efficiently masticate the solid food.[2,7,16] In at least 2 instances quoted by Banks *et al.*[2] the symptoms started following removal of teeth.
b. The associated abnormalities such as hiatal hernia, gastritis or peptic ulcers either contribute or are responsible for the clinical manifestations. Even as an incidental finding, however, the true nature of antral diaphragm should be correctly recognized and differentiated from other abnormalities.[11,21,24,30]

Gastric or duodenal ulcers have been reported in nearly one-fourth of the cases, either prior to or at the time of diagnosis of an antral diaphragm.[6,7,10,18,20] Three of our cases also had duodenal ulcer, and a third patient had a superficial gastric ulcer noted on gastroscopy.

Although concurrent demonstration of Schatzki's ring or hiatal hernia and antral diaphragm is not described in the litera-

ture, they were associated in 4 of the 6 cases (Table 1).

The distribution of our patients regarding their age and sex (Table 1) is comparable with previous reports.[2,10,22] The 25 cases recently reviewed by Hait *et al.*[10] consisted of 14 females (56 per cent) and 11 males (44 per cent). The age at the time of the diagnosis ranged from 28 to 74 years, but the majority were beyond the fifth decade of life.

It should be noted that complete antral diaphragm representing a form of gastric atresia is usually diagnosed within the first few days of life.[5,10,16,29] In contrast, the nonstenotic lesion may remain clinically silent until adulthood. Thus, the age distribution of all patients at the time of diagnosis of antral diaphragm results in a biphasic pattern similar to that observed with congenital annular pancreas.[16]

ROENTGENOLOGIC DIAGNOSIS

A correct diagnosis of antral ring or diaphragm is not difficult if the radiologist is familiar with the entity. In a review of 12 cases by Banks *et al.*[2] the lesion had been correctly diagnosed in 9. In the remaining 3 patients the diaphragm was retrospectively visible on preoperative roentgenograms. However, Hait *et al.*[10] note recently that a correct roentgenologic diagnosis was made in only 12 of 25 reported cases.

Roentgenographically, the lesion presents as a persistent, sharp, symmetric annular structure dividing the antrum (Fig. 1, *A–C*; and 3–7). Thus, the chamber produced distal to the diaphragm may re-

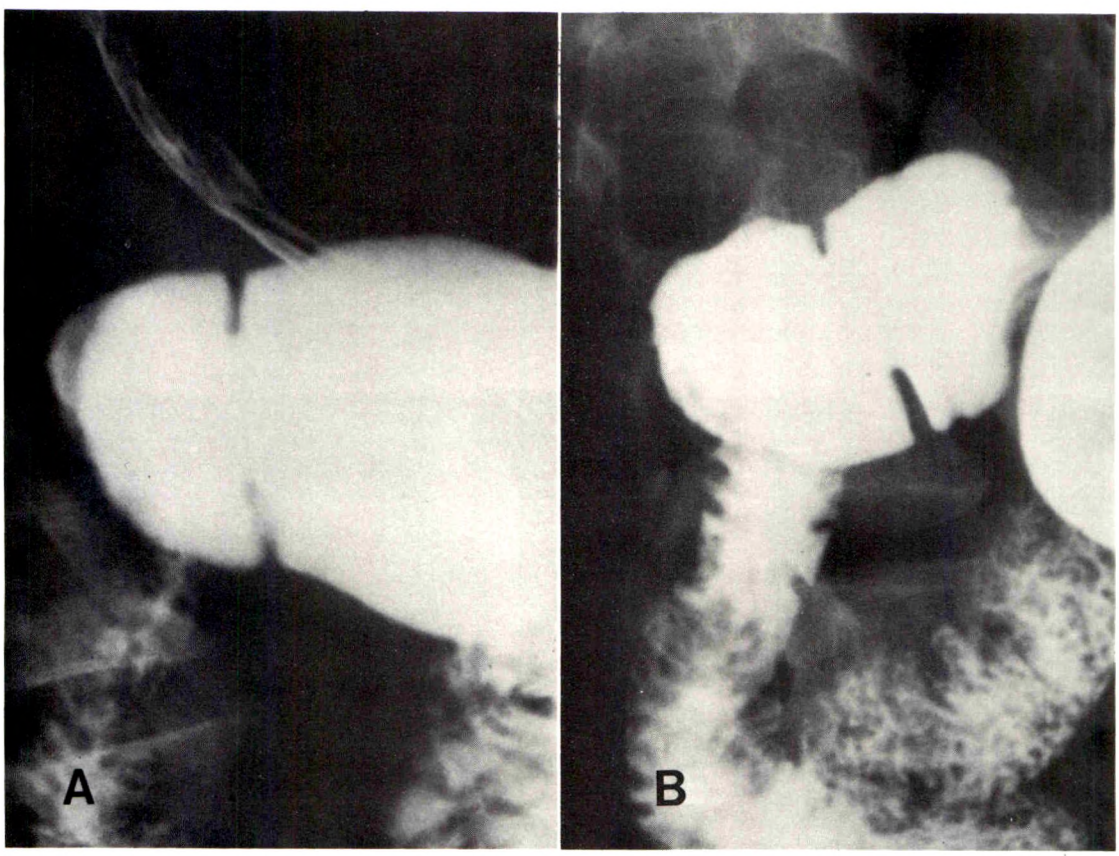


FIG. 3. Case 11. (*A* and *B*) Roentgenograms from 2 examinations done 6 months apart demonstrate the persistent ring-like appearance of a nonobstructive diaphragm. The deformed bulb is projected posterior to the antrum.

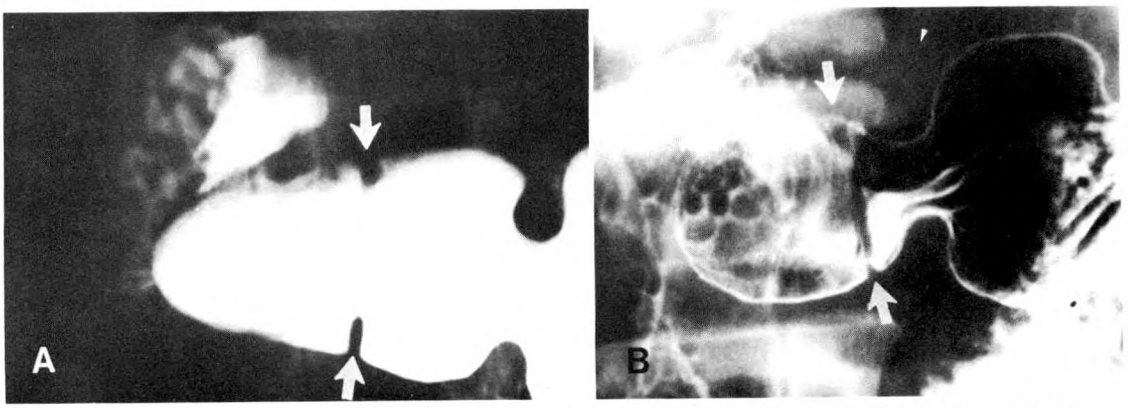


FIG. 4. Case III. Demonstration of a nonstenotic mucosal diaphragm (arrows) in barium filled (*A*) and air-contrast views (*B*) of the antrum.

semble and be mistaken for a duodenal bulb. Indeed several authors have referred to a characteristic "double bulb" in describing the abnormality.[2,7,10]

Antral diaphragms with small aperture often result in marked delay in gastric evacuation of barium.[5,6,16] A "jet-effect" may be seen due to forced passage of contrast material through the orifice of the diaphragm. Association of the lesion with transpyloric antral mucosal prolapse is reported by Melamed *et al.*[15] The authors suggest that the normal cephalad movement of the mucosa during antral systole (Golden's phenomenon) is prevented by the diaphragm. However, this impression has not been confirmed by others, and we have observed retrograde movement of the antral mucosa in the presence of antral diaphragm (Fig. 5, *A* and *B*).

The diaphragm or ring is best demonstrated when the antrum is relaxed and the lesion is seen in profile. This is achieved by performing fluoroscopy and spot-filming with the patient in a recumbent right anterior oblique position (with respect to the table top). The opposite projection, namely the left posterior oblique, is essential for air-contrast delineation of the lesion (Fig. 1, *B* and *C*). Maximal distention of segments immediately proximal and distal to the antral ring is absolutely necessary for

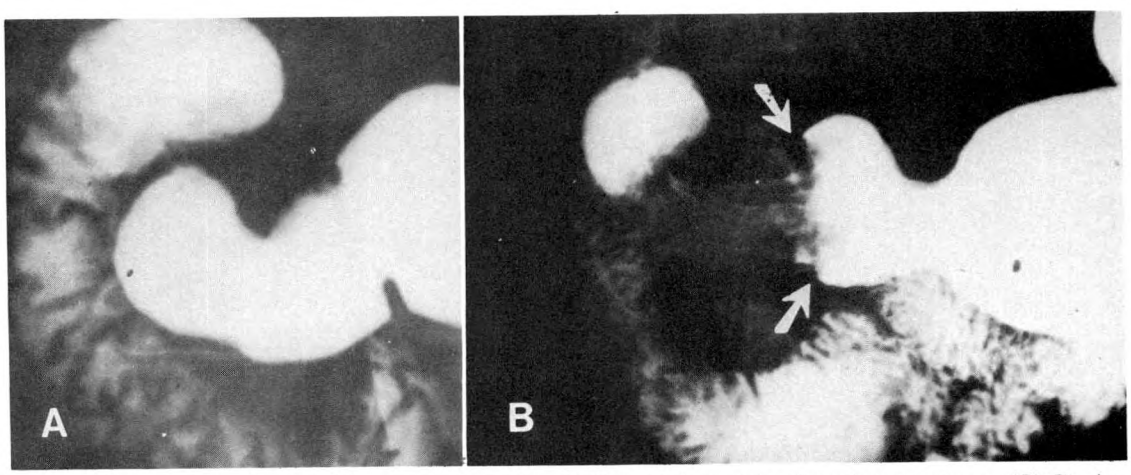


FIG. 5. Case IV. (*A*) The ring-like appearance of a diaphragm is seen in the relaxed antrum. (*B*) During antral systole there is a sharp demarcation at the level of diaphragm (arrows), with retrograde prolapse of antral mucosa.

adequate demonstration of this lesion as it is also true with Schatzki's ring.[14,25] Therefore, compression views are not helpful, and roentgenograms taken in upright position seldom show the abnormality. Cinefluorography or serial spot-filming, with patient in recumbent positions noted above, are often adequate for documentation of the lesion. However, a better delineation of the antral rings will often require air-contrast examination of the stomach following administration of anticholinergic agents.[8] In fact, this technique was solely responsible for correct recognition of the lesion in Cases I and II.

GASTROSCOPIC FINDINGS

Gastroscopic diagnosis of the antral diaphragm had been made in only 2 of 39 cases reviewed by Felson *et al.*[7] Since then, 2 additional reports of successful gastroscopic visualization have come to our attention.[4,12] In this series the roentgenologic demonstration of antral diaphragm was most significant for subsequent visualization of the lesion in 3 patients who underwent gastroscopy. Furthermore, the correct recognition of antral diaphragm will not only depend on experience of the endoscopist, but also on a careful examination of the fully relaxed and air-distended stomach. Therefore, premedication of patients with anticholinergic-spasmolytic drugs is absolutely necessary.

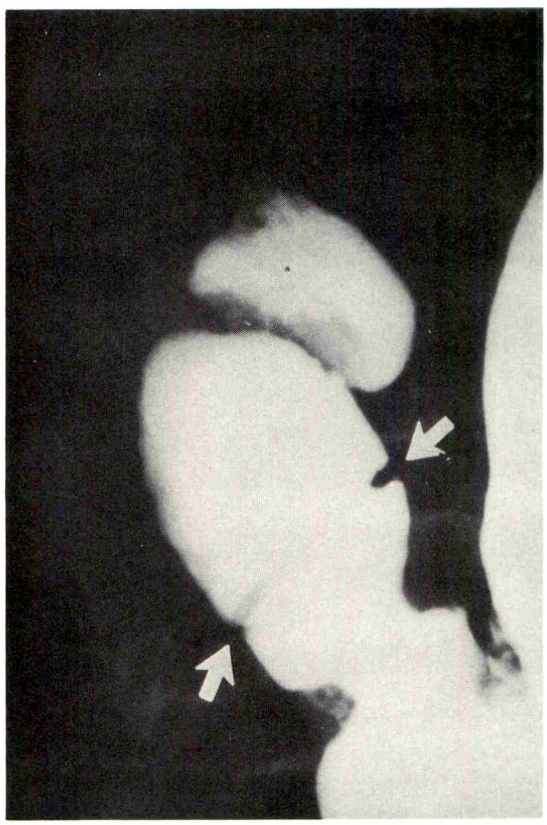


Fig. 6. Case v. Antral diaphragm (arrows) presenting as a sharp annular structure similar to the Schatzki's ring of distal esophagus.

At gastroscopy the antral diaphragm is clearly seen to project into the lumen (Fig. 2, *A* and *B*). It is covered by normal appearing mucosa, but occasional marginal erosions or adjacent ulcerations may oc-

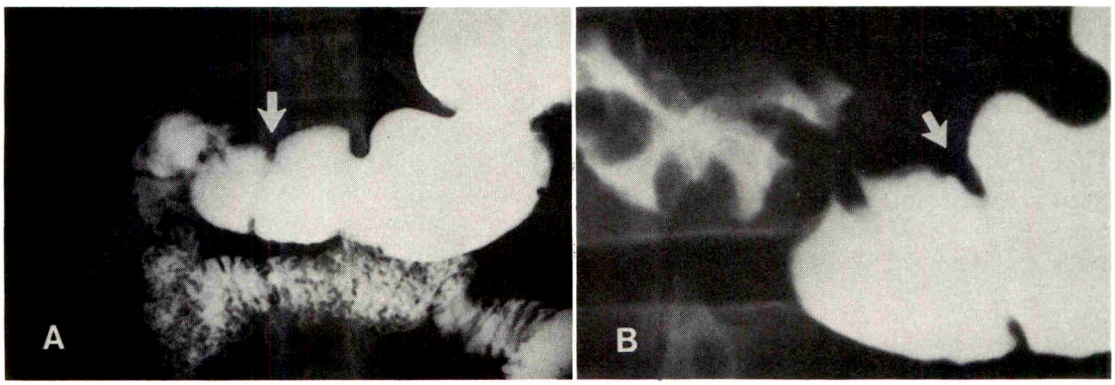


Fig. 7. Case vi. (*A*) The antral diaphragm (arrow) as recognized retrospectively on this roentgenogram of April 1971. (*B*) Spot film roentgenogram of a study in June 1973 confirms the persistence of the lesion (arrow). The irregularity of the antral mucosa is due to gastritis in this patient who also has a duodenal bulb ulcer.

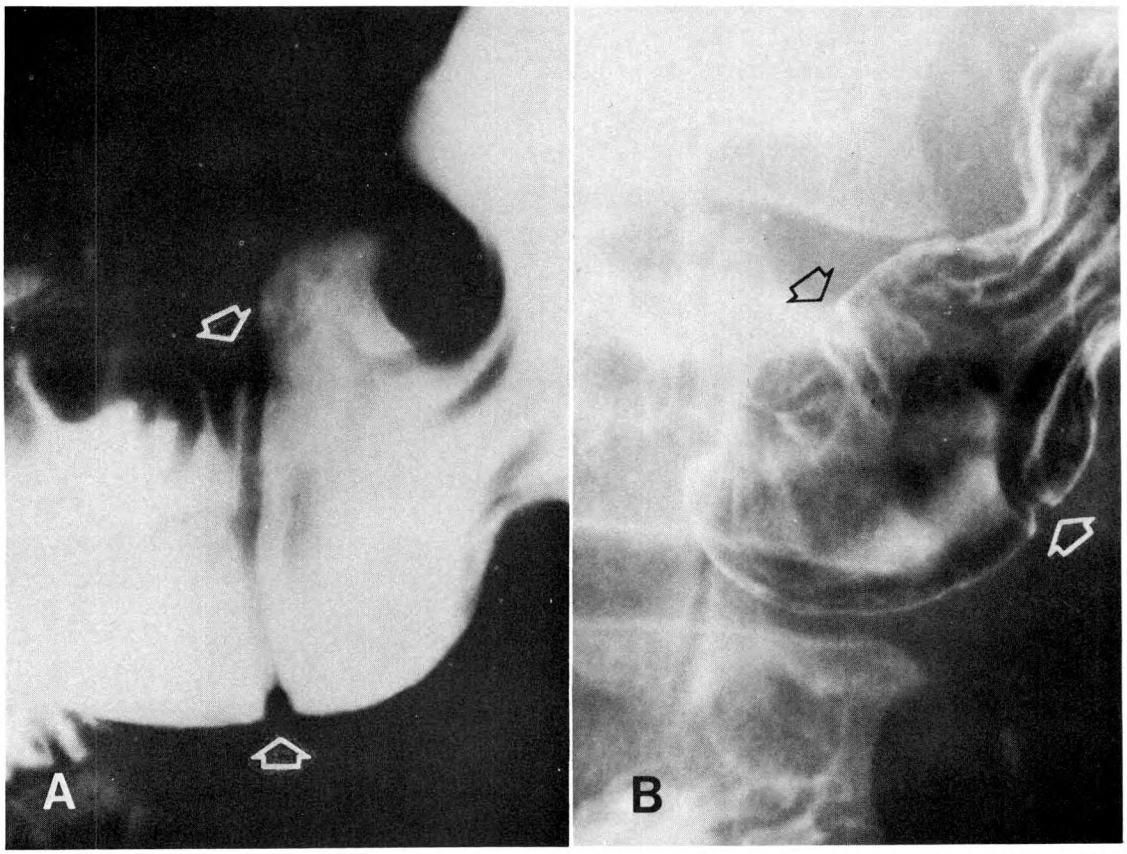


FIG. 8. Pseudo-diaphragm in a 59 year old man with gastritis. (*A*) This spot film roentgenogram shows multiple vertically oriented folds, one of which simulates a mucosal diaphragm (arrows). (*B*) Air-contrast view delineates the gastric folds (arrows) which do not encircle the antrum. This is an important distinction from antral diaphragms shown in Figures 1 and 4.

cur.[1,2,27] The aperture of the diaphragm is usually central and round. It may assume ovoid shape particularly during passage of peristaltic waves.[12] The diaphragm does not participate or disappear with peristalsis because it does not contain muscular layers of the gastric wall. The diameter of its opening does not exceed a rather fixed maximum even with fully air-distended antrum. Furthermore, the orifice remains continuously open in contrast to the intermittent closure of pylorus as seen during gastroscopy.[1,7,27]

TREATMENT AND SURGICAL PATHOLOGIC ASPECTS

Nearly all of the previous cases of antral diaphragm underwent gastric surgery either to relieve obstruction or because the true nature of the antral diaphragm was not correctly assessed on preoperative roentgenograms. In the absence of any associated abnormality, incision or excision of the diaphragm, or antrectomy are the methods of choice.[7,10,18,22] As mentioned above, antral diaphragm does not result in any visible change on the serosal surface of the stomach. Therefore, recognition of the lesion requires gastrotomy and a careful digital palpation of the antrum's inner wall.[10,16,18,22] It has even been recommended that surgeons routinely perform a digital exploration of segments adjacent to any gastric or duodenal incision, and search for a possible mucosal diaphragm.[3,10]

Several authors have pointed out the difficulties in recognizing the lesion in the surgical pathologic specimen.[2,6] Particularly

when the antrum is dissected longitudinally, the diaphragm tends to retract and virtually disappear among the surrounding gastric rugae. This is also the probable reason why only one antral diaphragm has ever been reported as incidental autopsy finding.[7] Application of the air-insufflation method used by MacMahon *et al.*[14] for postmortem demonstration of Schatzki's ring would probably facilitate recognition of antral diaphragms in the future.

It is our opinion that antral diaphragms with an aperture of over 1.0 cm. do not require surgical treatment. These lesions are nonobstructive, since there is no physiologic requirement for a larger orifice than that of pylorus. Our cases like a few others did not have gastric retentions, and it was noted that the diaphragmatic aperture would easily accommodate the tip of gastroscope. Therefore, it seems logical that the presenting symptoms of these patients are related to hiatal hernia and peptic ulcers rather than the antral diaphragm. Consequently, the treatment should be directed toward the associated abnormalities. In addition, the significant relation of inadequate mastication and production of symptoms in these often edentulous patients should be kept in mind, and necessary instructions about the diet and dental care be provided. Finally, it is hoped that further knowledge about the antral diaphragm would stimulate technical development of instruments for dilatation or incision of symptomatic diaphragms during gastroscopy.[12]

CORRELATIONS BETWEEN THE LOWER ESOPHAGEAL RING AND ANTRAL DIAPHRAGM

Four of our 6 cases had a sliding hiatal hernia, and in 3 instances a typical Schatzki's ring of the esophagus was evident. Previous authors have not referred to such association, although it may have been present in some patients who were in the older age group. Our limited experience does not permit any conclusions about the significance of this association. For didactic purposes, however, one may summarize the similarities which exist between the Schatzki's ring[14,25] and the antral diaphragm or ring. Both lesions are seen in older subjects who are usually asymptomatic if the diameter of the orifice exceeds 1 cm. Roentgenographically, the lesions present as a sharp, annular structure of 2–4 mm. thickness. Their demonstration is best achieved by maximal distention of immediately proximal and distal segments, particularly when the patient is in a recumbent oblique position.[7,25]

It is interesting to note that anatomic architecture of the resected antral diaphragm[2,7,10,16,18] closely resembles that of the lower esophageal ring.[14] However, we can only postulate that the antral diaphragm also demarcates the transitional zone of 2 different mucosal linings. According to Oi *et al.*[17] the fundic and antral types of gastric mucosa have a border zone which is, on average, 2–3 mm. wide and 5–10 cm. proximal to the pylorus. On this basis, it would appear that the width and location of the border zone and antral rings correspond in some instances. Oi *et al.*[17] also point out the occurrence of linear ulcers in the border zone. Rhind[19,20] believes that the healing of the linear and circumferential ulcers is the causal factor for the formation of mucosal diaphragms. Unfortunately, detailed description about the nature of the mucosal lining of resected antral diaphragms is not available. Thus, the hypothesis will need support of additional histopathologic data. Similarly, a possible relation of antral diaphragm and ectopic smooth muscle bundles of the antrum originally described by Lattes[13] deserves investigation.

DIFFERENTIAL DIAGNOSIS

Roentgenologic differential diagnosis of antral diaphragm or ring includes a few distinguishable possibilities. Redundancy of the gastric mucosa and prominent rugal folds could produce a confusing appearance.[7,15,21] These and the vertical antral folds often seen with gastritis are usually

multiple, wider and irregular in outline (Fig. 7, *A* and *B*).[21,30] They do not fully encircle the antrum, and fail to produce the characteristic and persistent annular structure of a diaphragm in an air-contrast distended antrum (Fig. 1, *A–C*; and 8, *A* and *B*).

Perigastric adhesions and cystogastrocolic bands should be considered in the differential diagnosis of antral rings.[7,11,24] The extrinsic compression produced by these adhesive bands is usually limited to the anterior antral wall. Melamed *et al.*[15] note that the peristalsis may be hampered by compressive bands, while no interference occurs in the presence of an antral diaphragm.

Constricting peristaltic waves and localized spasm are recognized by virtue of their transient nature, and usually asymmetric indentation on the gastric curvatures. These functional changes are easily eliminated by anticholinergic-spasmolytic agents.[8] Therefore, we prefer the air-contrast examination of the stomach by the "hypotonic" method. It will permit better visualization of antral diaphragm, and more accurate differentiation from other lesions which may simulate a diaphragm on the conventional gastroduodenal series. This method, together with careful gastroscopic evaluation, should provide the correct diagnosis of an antral diaphragm, and thereby minimize the number of unnecessary gastrectomies performed for this simple lesion.

SUMMARY

Antral diaphragm or ring is a persistent symmetric mucosal septum of 2–4 mm. thickness which protrudes vertically into the lumen a few centimeters proximal to the pylorus. The majority of 41 previously reported adult patients underwent surgery to relieve gastric outlet obstruction resulting from the small orifice of the antral diaphragm.

Six cases of nonstenotic form are herein described and the pertinent literature is reviewed. In this series there were 4 female and 2 male patients. The average age at the time of diagnosis was 50 years.

It is noted that obstructive symptoms are absent if the diameter of diaphragmatic aperture exceeds 1 cm. Nevertheless, patients often present with prolonged history of intermittent postprandial epigastric pain and sensation of fullness. These are mainly due to associated peptic ulcers and/or hiatal hernia as documented in all cases of this series. The coexistence of the lower esophageal ring and antral diaphragm in 3 patients is also described with emphasis on clinical and morphologic similarities between these lesions.

Roentgenographic demonstration of antral diaphragm requires examination of the patient in recumbent oblique position, and maximal air-contrast distention of the stomach. Premedication with anticholinergic drugs is useful for both gastroscopic and roentgenologic visualization of the lesion.

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REFERENCES

1. Banks, P. A., and Waye, J. D. Gastroscopic appearance of antral web. *Gastrointest. Endosc.*, 1969, *15*, 228–229.
2. Banks, P. A., Waye, J. D., Waitman, A. M., and Cornell, A. Mucosal diaphragm of gastric antrum. *Gastroenterology*, 1967, *52*, 1003–1008.
3. Bilbao, M. K., Frische, L. H., Rösch, J., Benson, J. A., Jr., and Dotter, C. T. Postbulbar duodenal ulcer and ring-stricture: cause and effect. *Radiology*, 1971, *100*, 27–35.
4. Conway, N. Pyloric antral mucosal diaphragm. *Brit. M. J.*, 1965, *1*, 970–971.

5. Cremin, B. J. Congenital pyloric antral membranes in infancy. *Radiology*, 1969, *92*, 509–512.
6. Farman, J., Cywes, S., and Werbeloff, L. Pyloric mucosal diaphragms. *Clin. Radiol.*, 1968, *19*, 95–99.
7. Felson, B., Berkmen, Y. M., and Hoyumpa, A. M. Gastric mucosal diaphragm. *Radiology*, 1969, *92*, 513–517.
8. Ferrucci, J. T., Jr., and Benedict, K. T., Jr. Anticholinergic-aided study of gastrointestinal tract. *Radiol. Clin. North America*, 1971, *9*, 23–39.
9. Gross, K. E., and Durham, M. W. Pyloric antral mucosal diaphragm: report of case. *Radiology*, 1953, *61*, 368–372.
10. Hait, G., Esselstyn, C. B., Jr., and Rankin, G. B. Prepyloric mucosal diaphragm (antral web): report of case and review of literature. *A.M.A. Arch. Surg.*, 1972, *105*, 486–490.
11. Jenkinson, E. L., and Hamernik, F. J. Roentgenologic deformities of pyloric portion of stomach with absence of surgical and pathological findings. *Radiology*, 1948, *51*, 798–805.
12. Katz, L. A. Asymptomatic mucosal diaphragm of gastric antrum: report of case. *Gastrointest. Endosc.*, 1968, *15*, 106–107.
13. Lattes, R. Ectopic smooth muscle in human gastric mucosa. *Am. J. Path.*, 1947, *23*, 501–511.
14. MacMahon, H. E., Schatzki, R., and Gary, J. E. Pathology of lower esophageal ring: report of case, with autopsy, observed for nine years. *New England J. Med.*, 1958, *259*, 1–8.
15. Melamed, A., Haukohl, R. S., and Callan, R. E. Pyloric antral mucosal diaphragm with transpyloric mucosal prolapse. *Radiology*, 1960, *74*, 452–457.
16. Munro, A. I. Prepyloric mucosal diaphragm. *Brit. J. Surg.*, 1963, *50*, 981–982.
17. Oi, M., Oshida, K., and Sugimura, S. Location of gastric ulcer. *Gastroenterology*, 1959, *36*, 45–56.
18. Parish, R. A., Kanavage, C. B., Wells, J. A., and Moretz, W. H. Congenital antral membrane. *Surg., Gynec. & Obst.*, 1968, *127*, 999–1004.
19. Rhind, J. A. Mucosal stenosis of pylorus. *Brit. J. Surg.*, 1959, *46*, 534–540.
20. Rhind, J. A. Pyloric antral mucosal diaphragm. *Brit. M. J.*, 1965, *1*, 1309.
21. Ricketts, W. E., Kirsner, J. B., and Palmer, W. L. Large, otherwise normal gastric rugae simulating tumor of stomach: report of 3 cases. *Gastroenterology*, 1947, *8*, 123–130.
22. Rowling, J. T. Prepyloric septum: rare anomaly. *Brit. J. Surg.*, 1959, *47*, 162–166.
23. Sames, C. P. Case of partial atresia of pyloric antrum due to mucosal diaphragm of doubtful origin. *Brit. J. Surg.*, 1949, *37*, 244–246.
24. Samuel, E. Cystogastrocolic band: radiological considerations. *Brit. J. Radiol.*, 1952, *25*, 19–21.
25. Schatzki, R., and Gary, J. E. Lower esophageal ring. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *75*, 246–261.
26. Schwartz, I. R., Hirsch, E., Mule, J. E., and Bluestone, L. Antral mucosal diaphragm: clinical and roentgen characteristics, with first reported case of vibrio fetus in human bile. *Am. J. Gastroenterol.*, 1966, *45*, 366–373.
27. Sokol, E. M., Shorofsky, M. A., and Werther, J. L. Mucosal diaphragm of gastric antrum. *Bull. Gastrointest. Endosc.*, 1965, *12*, 20–21.
28. Swartz, W. T., and Shepard, R. D. Congenital mucosal diaphragm of pyloric antrum. *J. Kentucky M. A.*, 1956, *54*, 149–151.
29. Touroff, A. S. W., and Sussman, R. M. Congenital prepyloric membranous obstruction in premature infant. *Surgery*, 1940, *8*, 739–755.
30. Vaughan, W. W. Antral gastritis: roentgenologic and gastroscopic findings. *Radiology*, 1945, *44*, 531–542.

ACQUIRED PARAESOPHAGEAL AND DIS-PARAESOPHAGEAL HERNIAS*

COMPLICATIONS OF HIATAL HERNIA REPAIR

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PSEUDOTUMOR effect after hiatal hernia repair simulating neoplasm of the gastric fundus has been previously described.[3,4]

The purpose of this communication is to present 3 cases of acquired herniations of the stomach through the diaphragm which occurred after hiatal hernia repair, and which may simulate diaphragmatic tumors or traumatic rupture of the diaphragm. The mechanism of hernia formation and method of diagnosis are also discussed.

REPORT OF CASES

CASE I. P.C., a 52 year old Caucasian male, was admitted to the University of Utah Medical Center Hospital with a 20 year history of heartburn and 6 weeks of postprandial precordial pain unrelieved by medical management. He had had an outside upper gastrointestinal series (UGI) which showed a sliding hiatus hernia, and this was confirmed on a repeat UGI (Fig. 1). Two days later he was taken to surgery and a Hill hiatus hernia repair was done. The hiatus admitted 4 fingers before closure, and was closed with 4 No. 1 Ethiflex sutures to the point where 1½ finger tips could be passed. The patient recovered uneventfully except for a urinary tract infection, and has since been asymptomatic. Approximately 6 weeks later a repeat UGI showed a small persistent hiatal hernia without reflux, and the defect from the Hill repair was noted. Six months after this, a follow-up UGI showed a larger paraesophageal hiatal hernia, and an acquired diverticulum near the cardia (Fig. 2). The patient remained asymptomatic.

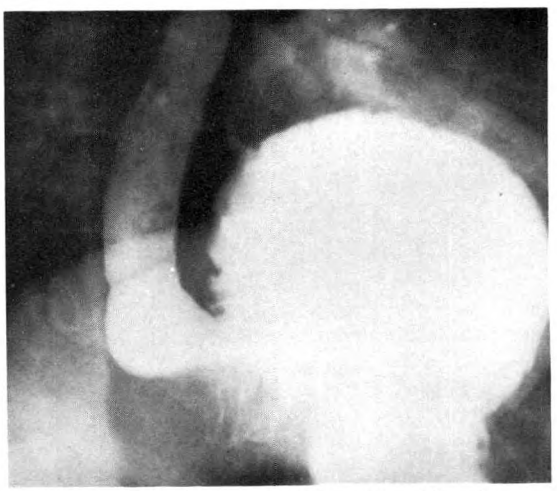


FIG. 1. Case I. Large sliding hiatal hernia prior to repair.

CASE II. F.O., a 55 year old Caucasian female, presented at the emergency room of the University of Utah Medical Center Hospital in atrial fibrillation. Her admission chest roentgenogram showed a mass in association with the left hemidiaphragm (Fig. 3). Sputum cytologies were positive. The history of a previous hiatal hernia repair was obtained and barium examination proved the mass to be a herniation of the stomach. Bronchoscopy was done with negative results. The patient converted to normal sinus rhythm on Quinidine. She then refused further diagnostic work-up and left the hospital on procaine amide maintenance therapy. Examination of the outside hospital record describing her hiatal hernia repair revealed that an abdominal approach had been used. The fundus of the stomach was sutured to the undersurface of the diaphragm for immobilization.

CASE III. C.C., a 33 year old Caucasian male, was admitted to the Latter Day Saints Hospital for evaluation of recurrence of vague lower chest pain following a hiatal hernia repair 7 months previously. The previous repair had been done with a transthoracic approach, and a fundoplication procedure was carried out. Roentgenologic examination revealed a hernia

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through the left hemidiaphragm (Fig. 4). The patient was subsequently re-operated and has done well since.

DISCUSSION

Since the early part of the century when William J. Mayo first began surgical correction for hiatal hernia,[2] there have been many procedures advocated. The early procedures in which the diaphragmatic opening was simply reduced in size (herniorrhaphy) proved unsatisfactory because of high recurrence rates. More recently, there has been concentration on maintaining the stomach in a subdiaphragmatic position by gastropexy. Some surgeons have also advocated extensive modifications in the fundus of the stomach to maintain a long intra-abdominal section of esophagus and create a new valve-like function at the esophagogastric junction (fundoplication).

Regardless of what further surgery is done, however, the diaphragmatic opening is usually still narrowed in one fashion or another.[9] In the Allison,[1] Hill,[5] and Krupp and Rosetti[6] fundoplication operations, this is accomplished in a similar manner. A No. 18 Levin tube is passed into the stomach to ensure maintenance of the lumen. After surgical exposure of the area, 3 to 4 sutures are utilized to oppose the two sides of the esophageal hiatus through the right hemidiaphragm. Sufficient opening is

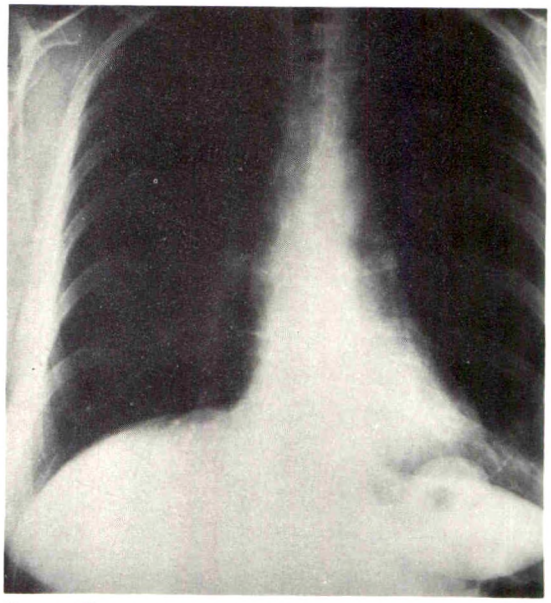


FIG. 3. Case II. Posteroanterior chest roentgenogram showing mass effect on left hemidiaphragm.

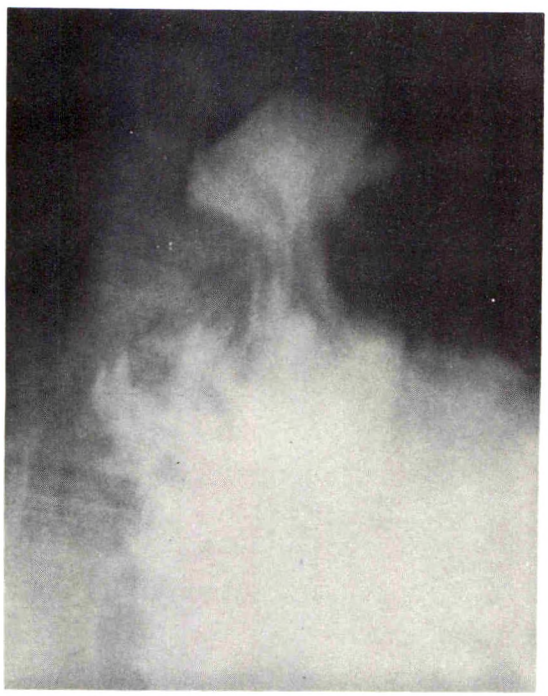


FIG. 2. Case I. Paraesophageal hiatal hernia after Hill repair.

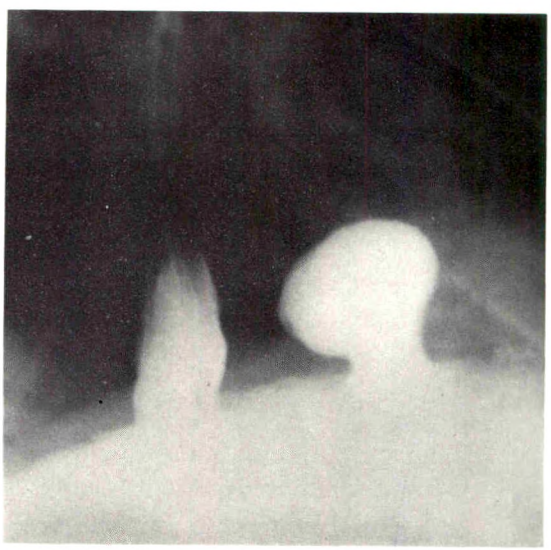


FIG. 4. Case III. Disparaesophageal hernia through opening in left hemidiaphragm created for admission of surgeon's fingers.

left in the hiatus to pass the tip of an index finger alongside the esophagus in order to ensure against stricture.

The Skinner and Belsey[8] Mark IV operation provides a similar closure of the hiatus, except that a somewhat tighter constriction is suggested. They advocate narrowing to the point that digital exploration of the remaining hiatal opening suggests the resistance encountered in the anal sphincter.

Allison[1] suggests fewer sutures (1, or at the most 2) in an attempt to maintain what he describes as the normal sphincter-like function of the right crus.

Nissen,[7] in describing his simple gastropexy to the anterior abdominal wall, does not advocate closure of the hiatus, apparently in order to avoid the more extensive surgery associated with mobilization of the left lobe of the liver.

All of the above mentioned procedures then further provide for some form of gastropexy in which the area of the esophagogastric junction is fastened below the diaphragm.

The transthoracic approaches which are currently used are similar in their methods of closure of the hiatus and gastropexy. If fundoplication is used, it is also similar, but may extend above the diaphragm or in some methods involve it. An incision is also made through the left hemidiaphragm to admit the surgeon's fingers to the abdomen and allow him more maneuverability in placing the gastropexy and fundoplication.

The end result of all the procedures is a situation in which the distal end of the esophagus and the cardia are securely anchored below the diaphragm, and the free part of the fundus is against the old hiatus which has usually been suture-closed.

If there is re-expansion of the hiatus around the esophagus, a postoperative paraesophageal hiatus hernia will have been created. This is the situation which arose in our first case.

Involvement of the hemidiaphragm with sutures as either a part of the gastropexy or fundoplication results in local traction on the hemidiaphragm. Combined with the generalized pressure on the underside of the hemidiaphragm from the fundus of the stomach, irregularities can be produced which may closely resemble a mass arising from the diaphragm. This was the effect produced in our second case.

When the transthoracic approach is used, the opening created for the surgeon's fingers is suture-closed in much the same way as the natural hiatus is narrowed. If the sutures in this area fail or are too widely spaced, the pressure of the fundus of the stomach will cause herniation well away from the esophageal hiatus. We have called this a disparaesophageal hernia, based on the Latin root for "separate." Our third case presented in just this manner, and at his second surgery was found to have broken the Tevdek sutures which were used to close the incision in his diaphragm.

Any of the 3 defects described could closely mimic a primary diaphragmatic tumor on plain film roentgenograms, if the amount of herniation were small. In trauma cases the defects produced could resemble closely the plain film roentgenographic findings seen in rupture of the diaphragm. The disparaesophageal hernia can also produce the same symptoms as simple hiatal hernia, as was seen in our third case.

When the clinical history or previous in-

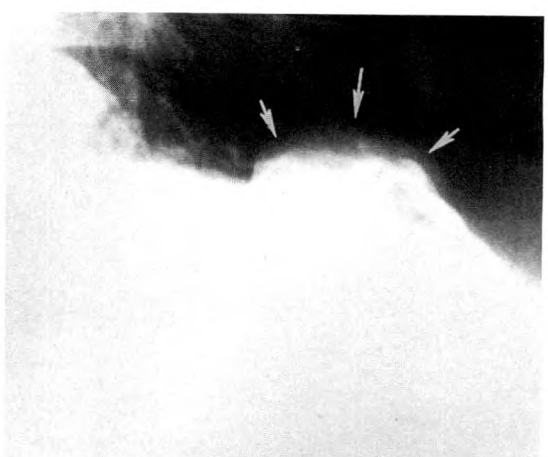


FIG. 5. Barium swallow in supine Trendelenburg position showing fundus of stomach filling hernia (arrows).

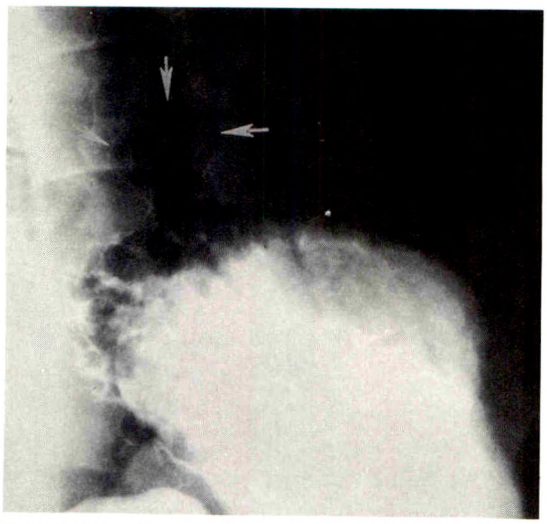


Fig. 6. Upright roentgenogram after barium swallow showing air contrast of disparaesophageal hernia (arrows).

cisions indicate a prior hiatal hernia repair and diaphragmatic masses are seen on plain film roentgenograms, the possibility of acquired postsurgical hernia can be simply investigated. The patient is given barium by mouth and placed in the supine Trendelenburg position. The pressure of the barium in the fundus of the stomach then fills the hernia sac (Fig. 5). As a further demonstration, the patient is returned to the horizontal prone or upright position, and the air contrast shows barium coating the lining of the stomach within the hernia sac (Fig. 6).

SUMMARY

Modern methods of hiatal hernia repair require fixation of the gastroesophageal junction below the diaphragm, as well as repair of the hernia defect.

Postsurgical herniations through the diaphragm may occur. With the gastroesophageal junction fixed in place, the new herniations will be either beside the esophagus (paraesophageal) or separated from it by intervening diaphragm (disparaesophageal). Irregularities of the diaphragm may be created by sutural attachments.

Any of these may simulate diaphragmatic tumor or traumatic rupture of the diaphragm. Either type of hernia may be symptomatic.

The mechanism of hernia formation in 3 such cases is described, and methods of diagnosis are discussed.

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REFERENCES

1. Allison, P. R. Reflux esophagitis, sliding hiatal hernia, and anatomy of repair. *Surg., Gynec. & Obst.*, 1951, *92*, 419–431.
2. Beckman, E. H. Diaphragmatic hernia. *Surg., Gynec. & Obst.*, 1909, *9*, 154–156.
3. Cohen, W. N. Fundoplication repair of sliding esophageal hiatus hernia: its roentgenographic appearance. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 625–631.
4. Ettinger, A., Paul, R. E., Jr., and Moran, J. M. Gastric pseudotumor after fundoplication. *Gastroenterology*, 1971, *61*, 299–304.
5. Hill, L. D. Effective operation for hiatal hernia: eight year apprasial. *Ann. Surg.*, 1967, *166*, 681–692.
6. Krupp, S., and Rosetti, M. Surgical treatment of hiatal hernias by fundoplication and gastropexy (Nissen repair). *Ann Surg.*, 1966, *164*, 927–934.
7. Nissen, R. Gastropexy as lone procedure in surgical repair of hiatus hernia. *Am. J. Surg.* 1956, *92*, 389–392.
8. Skinner, D. B., and Belsey, R. H. R. Surgical management of esophageal reflux and hiatus hernia. *J. Thoracic & Cardiovasc. Surg.*, 1967, *53*, 33–54.
9. Welch, C. E. Surgery of Stomach and Duodenum. Fifth edition. Year Book Publishers, Chicago, 1973, pp. 110–135.

SPONTANEOUS REDUCTION OF HERNIA "EN MASSE"*

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THE reduction of a femoral or inguinal hernia "en masse" can be basically defined as the reduction of a hernia sac together with its intestinal contents so that the retained bowel still remains incarcerated. This is a rare form of acute intestinal obstruction that occasionally confronts the surgeon, but with which most radiologists are unfamiliar. We have recently encountered a patient with this entity who demonstrated many unusual features.

It is the purpose of this report to acquaint the radiologist with this disease and to add this additional unusual case to the literature.

REPORT OF A CASE

S.T., a 47 year old Caucasian man, entered the hospital complaining of abdominal pain and distention. Three years ago, he had a left inguinal hernia that disappeared spontaneously without surgery or manipulation of the hernia. A physical examination revealed a scaphoid abdomen with high pitched bowel sounds. No abdominal masses could be palpated, his rectal examination was nonrevealing, and there was no evidence of an inguinal or femoral hernia.

Conservative therapy was attempted for 3 days and his initial symptoms subsided somewhat. However, abdominal roentgenograms revealed distended air and fluid filled loops of small intestine indicating a mechanical obstruction (Fig. 1, *A* and *B*). A barium enema examination was normal. A subsequent upper gastrointestinal and small intestinal series demonstrated dilated small intestine that was obstructed within the pelvis. Barium, however could be seen entering a small knuckle of ileum (Fig. 2, *A* and *B*).

At surgery a collar of peritoneum was found to encircle an ischemic knuckle of ileum. The diagnosis of an intra-abdominal hernia with reduction "en masse" was established. Postoperatively, the patient did well and had a complete recovery.

DISCUSSION

The absolute definition of hernia reduction "en masse" is somewhat confusing. Most authors agree that it basically implies reduction of the hernia sac with its intestinal contents into a displaced position. Some include strangulation and intra-abdominal displacement in the definition,[5] while others imply only incarceration and removal from the inguinal canal.[4] In any event the lesion is quite rare and has been quoted by Pearse[3] to occur in approximately 1 of 13,000 hernias.

Four types of "en masse" reductions have been described (Fig. 3):

1. Retropubic—A reduction by hernia sac rupture posteriorly with displacement of the viscus behind the pubis.

2. Intra-abdominal—Inversion of the hernia sac with a peritoneal collar torn away with the strangulated bowel and displaced into the abdominal cavity.

3. Preperitoneal—Reduction of the hernia sac with displacement into the preperitoneal area.

4. Preperitoneal locule—Reduction of the hernia sac into a preperitoneal locule from the inguinal pre-preperitoneal pouch.

The latter 2 categories are actually interparietal hernias which occur by transfer of the hernia from its original position in its scrotal or crural sac to a preformed preperitoneal locule.[1] It is questionable whether these represent a reduction "en masse" in the absolute definition of the entity. In any case, interparietal hernias account for over 90 per cent of all "en masse" reductions reported in the litera-

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ture, so that exclusion of the latter 2 categories would further decrease the incidence of this already rare occurrence.[1–3]

Clinically and roentgenologically, reduction "en masse" is suggested by the persistence of persistent signs of an intestinal strangulated obstruction after apparent reduction by taxis or manipulation of a hernia. This is frequently accompanied by pain or a tearing sensation at the time of reduction. Most unusual are the spontaneous reductions that have been recorded.[5]

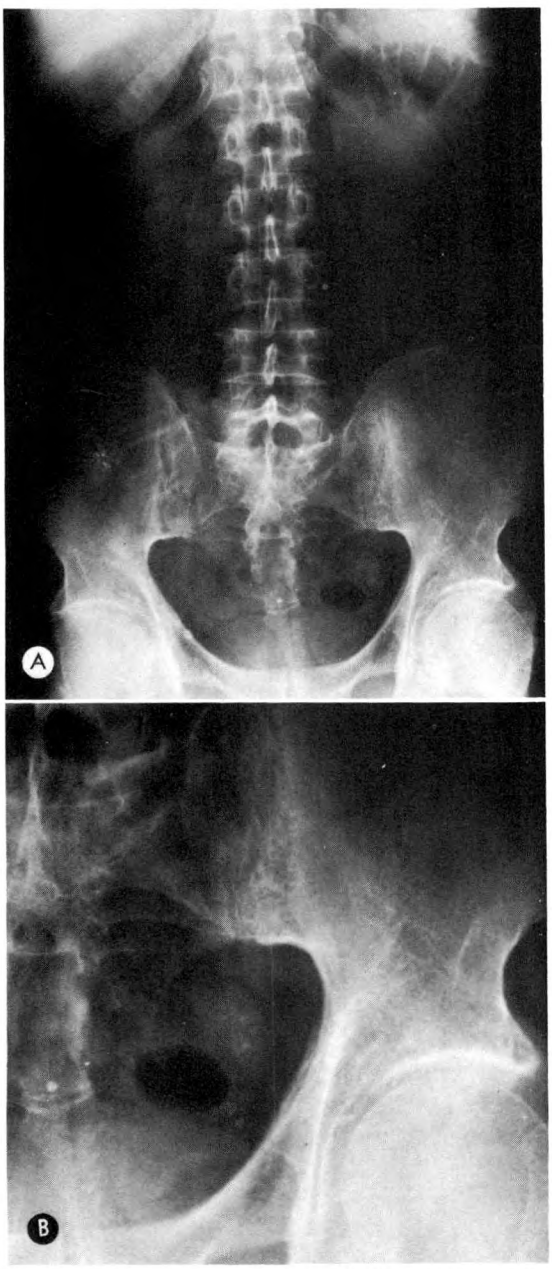


FIG. 1. (*A*) Note the small bowel dilatation and the single oval collection of air in the left pelvis projected above the perivesical fat line. (*B*) Blow-up of the pelvis demonstrating the knuckle of air filled bowel.

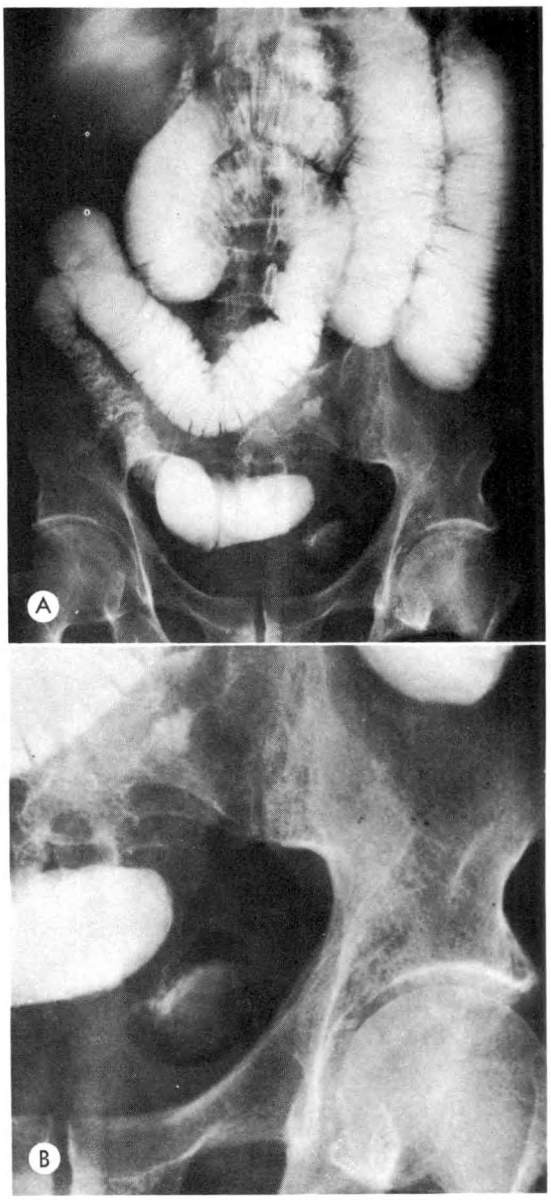


FIG. 2. (*A*) A 3 hour roentgenogram from the small bowel series reveals the site of mechanical obstruction in the left pelvis with barium entering the small air filled knuckle seen on the plain roentgenogram. (*B*) Blow-up of air and barium containing knuckle of bowel in the pelvis.

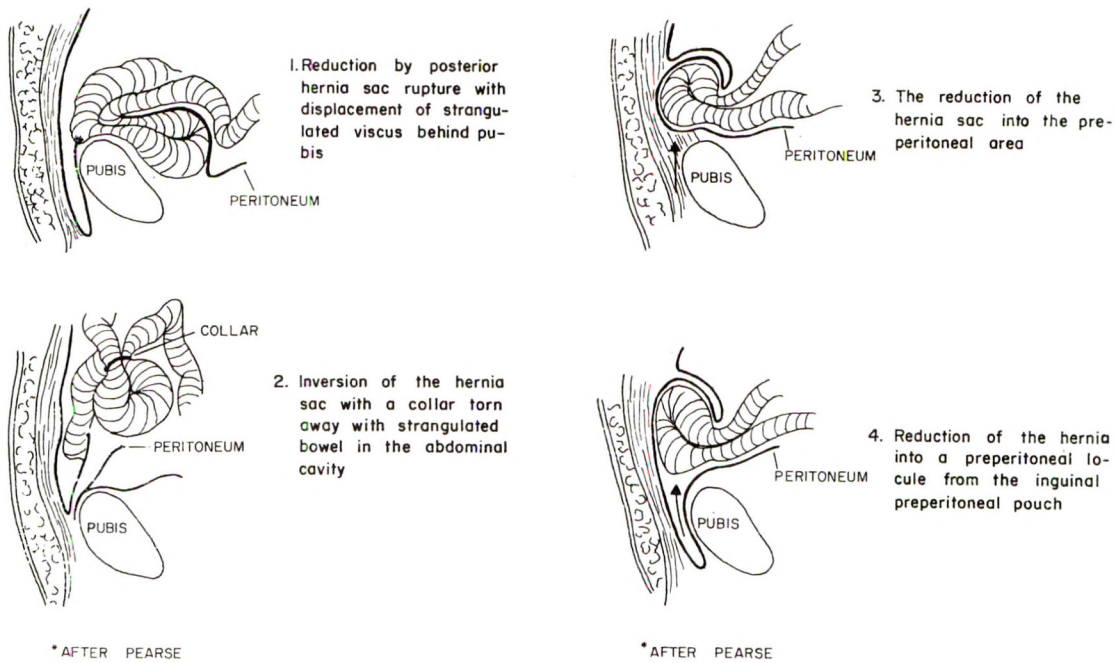


FIG. 3. Adapted from Pearse.[3] Demonstrated are the forms that "en masse" reduction might take.

Depending upon the displacement of the hernia, a tender mass may be palpated either high in the inguinal canal, above the inguinal ring, or low in the abdomen on the side of the reduction.

Roentgenographically, the signs of a small bowel intestinal obstruction are present, with multiple distended loops of small bowel noted proximal to the level of obstruction. Depending upon the condition of the incarcerated loop, varying features of strangulated obstruction may be superimposed. Small bowel studies may be quite revealing, if barium reaches and enters the closed loop and demonstrates the obstructed knuckle of bowel confirming the presence of an internal hernia. With a previous history of attempted reduction or spontaneous disappearance of a hernia and the roentgenographic signs described, the correct diagnosis of reduction of "en masse" could be suggested.

Early surgical intervention is necessary. An abdominal approach is usually preferred, since the exact position of the obstructing lesion cannot always be determined clinically or roentgenographically.[3] The prognosis after reduction "en masse" is not always good due to the delay in time from the onset of symptoms to the surgical intervention. This is even more evident in cases of spontaneous reduction.

Our patient was somewhat atypical in that his reduction was spontaneous, his symptoms were delayed for 3 years, and that despite a long delay from the onset of his symptoms to surgical intervention, he did quite well following the operative procedure.

SUMMARY

A rare and unusual case of reduction "en masse" is reported and the clinical and roentgenologic features discussed.

Although there are not specific roentgenographic signs of hernia reduction "en masse," it should be considered as a cause of intestinal obstruction in patients with persistent bowel obstruction following reduction of inguinal or femoral hernia.

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REFERENCES

1. Lower, W. E., and Hicken, H. F. Interparietal hernias. *Ann. Surg.*, 1931, *94*, 1070–1087.
2. Nyhus, L., and Harkin, H. Hernia. J. B. Lippincott Company, Philadelphia, 1964, pp. 619–620.
3. Pearse, H. E. Strangulated hernia reduced 'en masse.' *Surg., Gynec. & Obst.*, 1931, *53*, 822–828.
4. Querna, M. H. Reduction of hernia 'en masse.' *Am. J. Surg.*, 1969, *118*, 539–540.
5. Zimmerman, L. M., and Anson, B. J. Anatomy and Surgery of Hernia. Williams & Wilkins Company, Baltimore, 1953 and 1967, pp. 40–43.

DIAGNOSTIC VALUE OF HYPOTONIC DUODENOGRAPHY*

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HYPOTONIC duodenography is the most rewarding procedure now generally available for the detection of abnormal masses adjacent to the duodenal loop. It is simple to perform and offers no risk to the patient. Nevertheless, this procedure is underutilized and, because it is underutilized, lesions of the pancreas often remain as diagnostic enigmas. Regrettably, the physician in private practice uses hypotonic duodenography least of anyone.

Despite the fact that this method has been little used in our hospital, in the past 5 years it has aided us in discovering carcinoma of the head of the pancreas in 12 patients. Before hypotonic duodenography was employed in our institution, carcinoma of the pancreas was rarely detected on roentgenograms.

METHOD

We employ a tube positioned in the duodenal cap or distal portion of the stomach.[4] Tube position is not critical; in fact, the procedure can be done with the tube in the fundus. Prolonged attempts to enter the cap are unwarranted. We have found the tubeless method to be less satisfactory primarily because of lack of control.[3,6]

Sixty milligrams of propantheline bromide* are administered intramuscularly. We have found lesser amounts to be unreliable in producing the desired relaxation of the duodenal loop.[6] Films are exposed with barium in the loop and with air contrast. It is essential that the films be of excellent quality.

Obstructive jaundice is a prime indication, but any combination of symptoms and signs pointing to the upper abdomen that cannot be explained by other, routine procedures may be considered an indication for the hypotonic duodenography. It is also helpful in clarifying indefinite findings from routine examination of the gastrointestinal tract.

Although Pro-Banthine produces transient dry mouth and vague eye symptoms, no serious complications result from the procedure. Gastric dilatation may be avoided by withholding food and liquid for 3 hours after the procedure.[2] Occasionally urinary retention in men with prostatism may require catheterization. Pro-Banthine is, of course, contraindicated for persons with glaucoma or specific allergy to the drug.

REPORT OF CASES

The following cases exemplify lesions seen by us in the past 5 years.

CASE I. The patient was a 51 year old woman with a 1 week history of jaundice, which was increasing in severity. She also had vague epigastric pain and low grade fever. She had had a cholecystectomy for stones and gave evidence of mature onset diabetes. Results of an examination of the gastrointestinal tract were interpreted as normal. The hypotonic duodenogram, however, showed nodular pressure on the inner side of the loop, typical of carcinoma of the head of the pancreas (Fig. 1).

CASE II. The patient was a 57 year old man with jaundice of 10 days' duration, poor appetite, and fatigue. There was no complaint of pain. He had had a previous partial gastrectomy for ulcer. The hypotonic duodenogram showed an intraluminal diverticulum arising at the site of previous anastomosis. In addition, there was an irregular filling defect thought to represent the ampulla. At operation, the defect

* Pro-Banthine® vials, manufactured by Searle and Co., San Juan, Puerto Rico 00936.

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was found to be a 1.8 cm. carcinoma of the head of the pancreas adjacent to the ampulla, which obstructed the common duct. In retrospect, the carcinoma was found to be clearly evident and probably should have been suspected originally because of the irregularity of the defect (Fig. 2).

Case III. A 33 year old Japanese man had a history of vague epigastric pain and indigestion. A roentgenographic examination of the gastrointestinal tract showed normal findings. The hypotonic duodenogram disclosed nodular pressure on the inner side of the loop and inferior aspect of the cap, as well as a deep incisura on the opposite wall of the duodenum (Fig. 3, *A* and *B*). The incisura was consistent with the type of deformity seen in postbulbar ulcer,[1,9] but the nodular pressure defect was thought to represent a carcinoma of the head of the pancreas. Both findings were confirmed at operation, when the patient was found to have a carcinoma in the head of the pancreas eroding into the duodenum and ulceration.

Case IV. A 70 year old woman had jaundice of 1 week's duration, vague indigestion, and fatigue. The findings from gastrointestinal examination were interpreted as normal. Hypotonic duodenography revealed nodular pressure on the inner side of the duodenal loop (Fig. 4). At operation the head of the pancreas was biopsied directly; no tumor could be found in the specimen. In light of the findings from the duodenogram, the duodenum was opened and the pancreas biopsied through the wall in the region shown to be abnormal. At this time, the specimen demonstrated carcinoma of the head of the pancreas. The duodenogram had been of added assistance in directing the surgeon to the lesion.

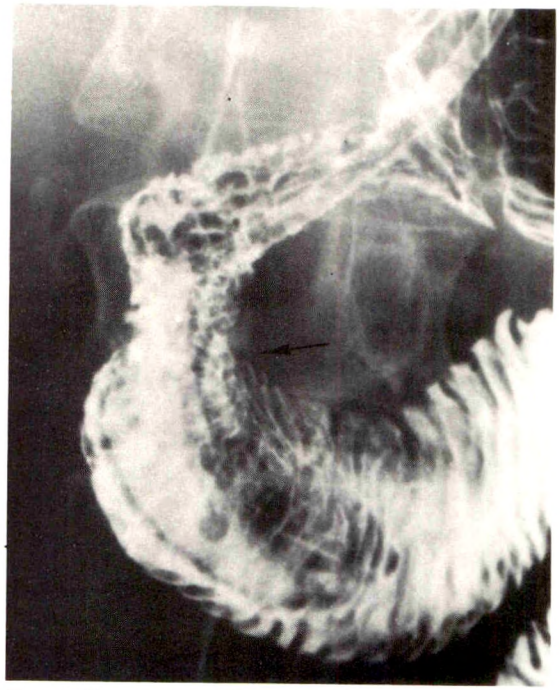


Fig. 2. Case II. Irregular filling defect (arrow) represents a carcinoma of the pancreas, 1.8 cm. in size. Note also the large intraluminal diverticulum.

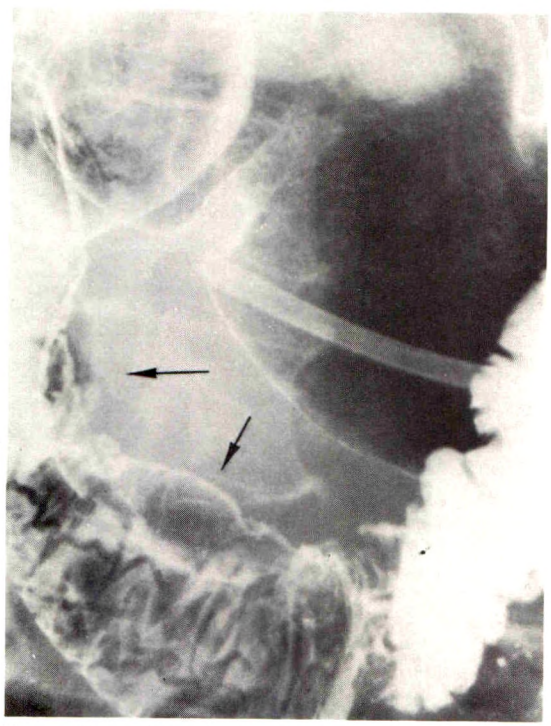


Fig. 1. Case I. Nodular pressure (arrows) on the inner side of the duodenal loop is typical of carcinoma of the head of the pancreas.

Case V. The patient was a 71 year old man with jaundice of 3 weeks' duration, increasing in severity, and who had had 3 or 4 episodes of vague epigastric distress. He had lost 20 pounds over the course of 3 months. A gastrointestinal series showed normal findings. The duodenogram showed nodular pressure on the inner side of the duodenal loop—consistent with a tumor mass of the head of the pancreas (Fig. 5). At operation, a carcinoma of the common bile duct was found, which had invaded the pancreas and adjacent lymph nodes.

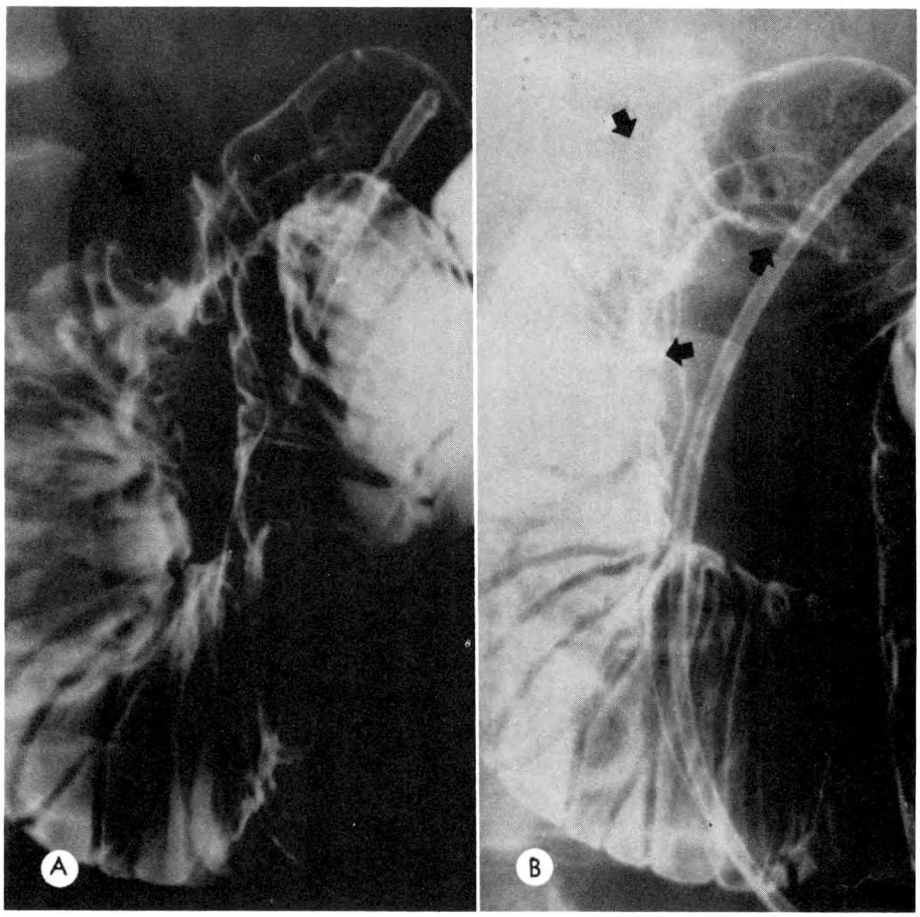


Fig. 3. Case III. (*A* and *B*) Nodular pressure (lower arrows) on the inner side of the duodenal loop is due to carcinoma of the head of the pancreas. The incisura (upper arrow) on the opposite wall is consistent with the type of deformity seen in postbulbar ulcer.

Case VI. A 41 year old man had a history of vague indigestion of several months' duration. He had had 2 gastrointestinal examinations. The first gave normal findings. The second revealed a lesion in the distal third of the duodenum, the nature of which was uncertain. The hypotonic duodenogram showed the lesion to encircle the bowel; it appeared typical of a carcinoma. In the central portion of the lesion was an ulcer. In addition, nodules were seen pressing along the inner side of the loop just proximal to the encircling lesion (Fig. 6). At operation, these nodules were found to be lymph nodes involved by metastatic carcinoma from the primary lesion at the distal third of the duodenum.

Case VII. A 55 year old woman had had jaundice for 3 weeks. A gastrointestinal examination showed questionable flattening along the inner side of the duodenal loop. The hypotonic duodenogram revealed a large mass impressing upon the outer aspect of the duodenal loop (Fig. 7). At operation this mass was found to be carcinoma of the gallbladder with extension into the liver.

Case VIII. A 64 year old Japanese woman had weight loss, chills, and fever, in addition to anemia of 9 months' duration. There was no jaundice or pain. An intravenous cholangiogram showed normal findings, but the gastrointestinal examination gave evidence of pressure on the inner side of the duodenal loop. A hypotonic duodenogram was obtained for further evaluation; it showed marked pressure on the inner side of the loop (Fig. 8). A small calcified scar was present in the upper lobe of the right lung.

At operation, a rubbery mass was found in the head of the pancreas, pressing upon the loop. Biopsy of this mass disclosed a granulomatous process with many acid-fast organisms (found positive for tuberculosis). Similarly involved lymph nodes were found throughout the upper abdomen. The patient responded well to treatment with ethanbutol hydrochloride and isoniazide.

Case ix. A 69 year old man had epigastric pain of 5 days' duration, anorexia, and obstructive jaundice. The gastrointestinal examinations gave findings considered normal. The hypotonic duodenogram showed irregular pressure along the inner side of the duodenal loop (Fig. 9). At operation, the patient was found to have a stone in the distal end of the common duct, with obstruction. Chronic pancreatitis was also evident, which was responsible for the pressure on the loop.

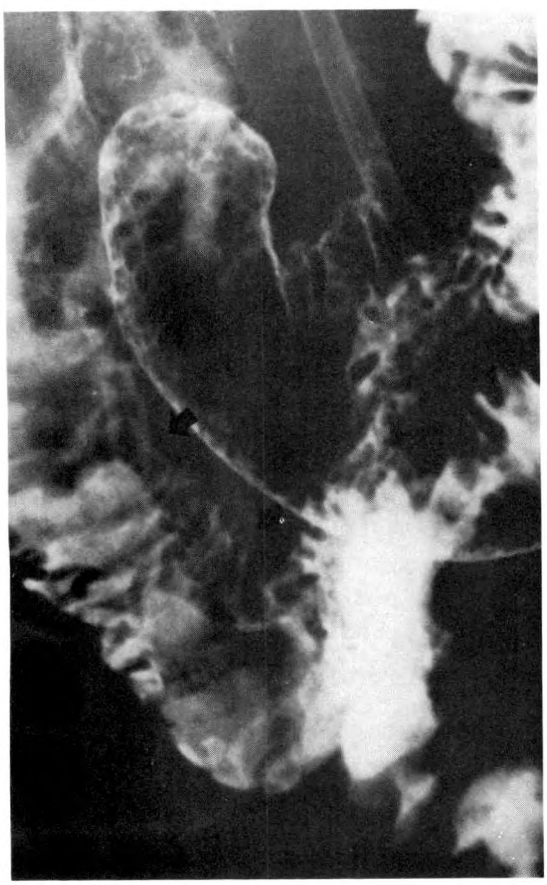


Fig. 4. Case iv. Nodular pressure (arrows) on the inner side of the duodenal loop is due to carcinoma of the head of the pancreas.

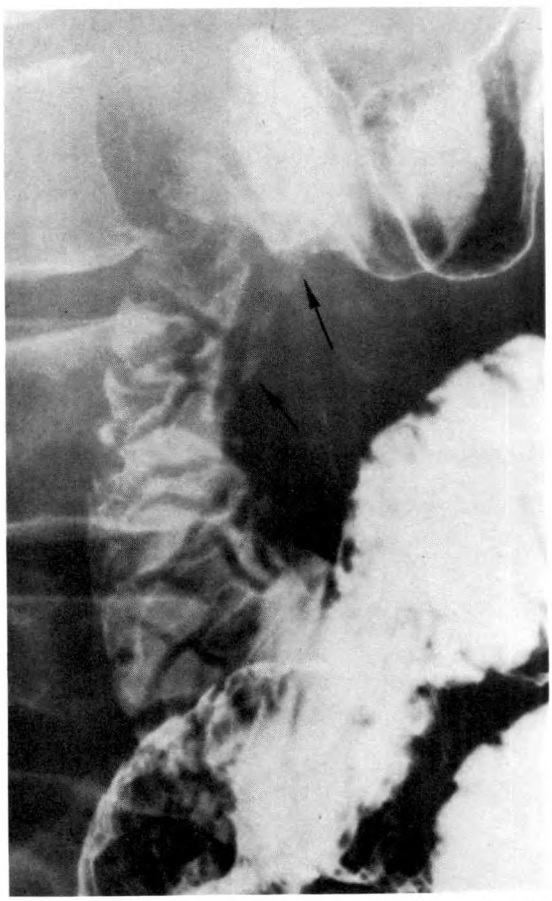


Fig. 5. Case v. Nodular pressure (arrows) on the inner side of the loop resulted from carcinoma of the common bile duct with invasion of the duodenum.

Case x. A 67 year old man had vague upper abdominal pain for many years as well as acute episodes of nausea and vomiting (especially immediately after eating). Nothing abnormal could be ascertained from the gastrointestinal examination. The hypotonic duodenogram showed a deep incisura in the lateral aspect of the mid portion of the descending duodenum. It also showed slight flattening of the mucosal folds along the inner aspect of the loop between the incisura and the duodenal cap (Fig. 10). Fluoroscopically, this area appeared irritated and showed relaxation only fleetingly, despite an injection of 60 mg. of Pro-Banthine. The possibility of an annular pancreas or fibrous band with pancreatitis was considered. At operation a broad fibrous band was found to be responsible for the incisura. Pancreatitis was not evident at the time of the

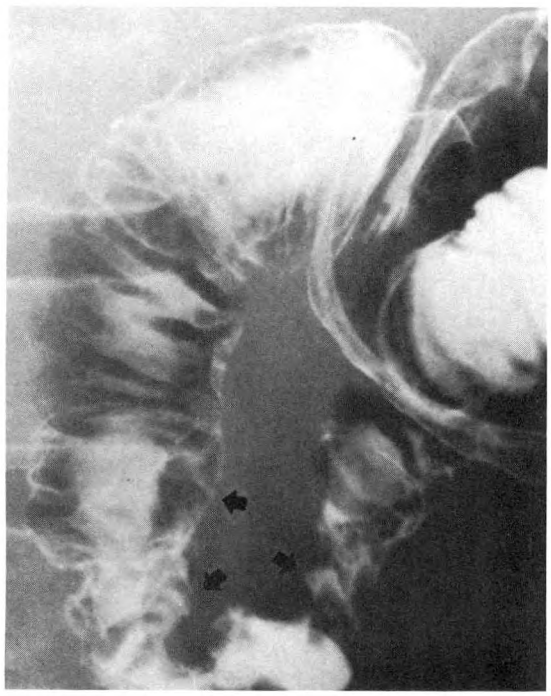


Fig. 6. Case vi. Encircling carcinoma of the distal third of the duodenum with ulceration (lower right arrow) can be seen. In addition there is nodular pressure on the inner side of the duodenum just proximally, a result of involvement of lymph nodes (left arrows) by the metastatic tumor.

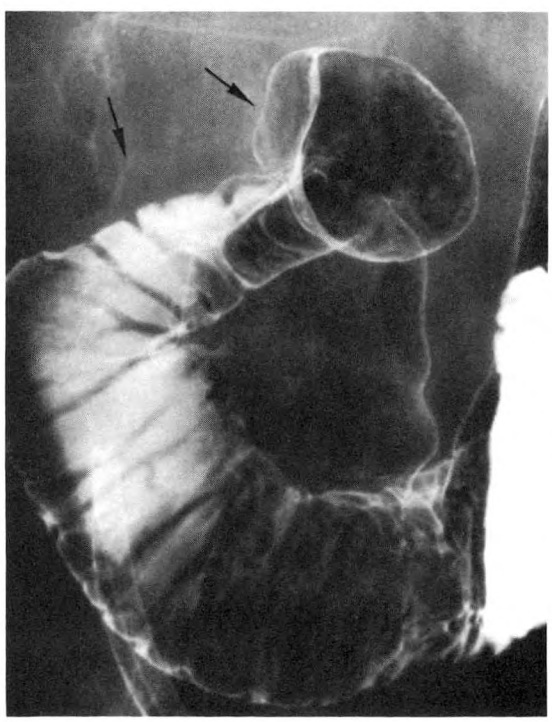




Fig. 8. Case viii. Irregular pressure (arrows) on the inner side of the duodenum is due to tuberculosis of the lymphatic system of the pancreas.

operation; the surgical procedure, however, was performed 3 weeks after the duodenography.

Case xi. A 52 year old Samoan woman had upper abdominal pain of several months' duration, weight loss over the course of a year, and poor appetite. Total bilirubin was slightly elevated and blood glucose was consistently elevated up to 160 mg. per 100 ml. Gastrointestinal examination revealed nothing abnormal. The hypotonic duodenogram showed an oval pressure defect on the superior aspect of the small bowel at the ligament of Treitz, consistent with a metastatic lesion to the bowel wall or direct pressure from a tumor in the body

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Fig. 7. Case vii. Large pressure defect (arrows) on the outer side of the duodenum is due to carcinoma of the gallbladder.

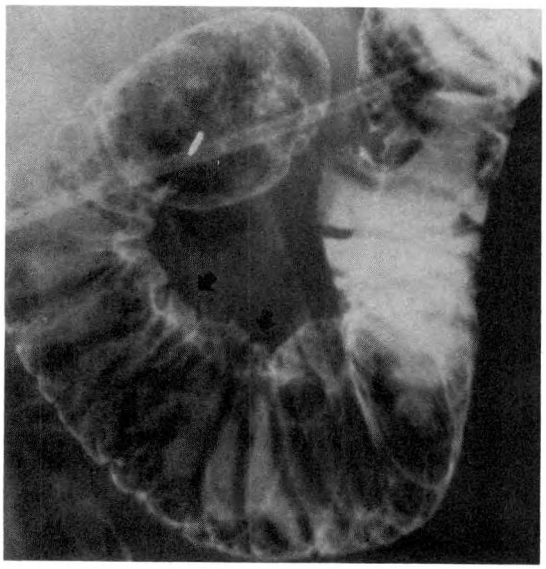


Fig. 9. Case IX. Irregular pressure defect (arrows) on the inner side of the duodenum results from pancreatitis. This defect is shallower and somewhat less nodular than the pressure usually seen in carcinoma of the head of the pancreas.

of the pancreas (Fig. 11). Surgery was recommended but refused by the patient, who did not return for follow-up examination.

Case XII. A 76 year old man had vague upper abdominal pain and distention, constipation, and a palpable mass in the left epigastrium. Findings of the gastrointestinal examination were considered normal. The hypotonic duodenogram showed irregular pressure along the inferior aspect of the stomach and a local pressure defect on the superior aspect of the small bowel, just beyond the ligament of Treitz (Fig. 12). This patient was not recommended for surgical treatment, but he subsequently developed abdominal carcinomatosis and died. The exact diagnosis was not made, but carcinoma of the body of the pancreas is the most likely possibility.

DISCUSSION

The methods employed in hypotonic duodenography may vary; excellent detail films of the relaxed duodenal loop, however, must result. Any deviation from the normal pattern of duodenal mucosa and contour is significant. It may not be possible to ascertain the precise reason for a deformity, but it is usually possible to detect a lesion in proximity to the loop.

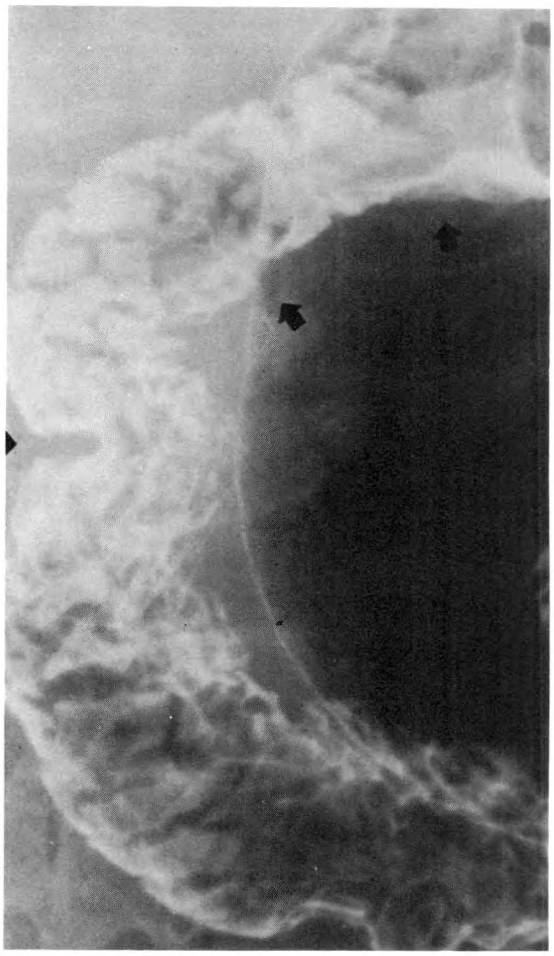


Fig. 10. Case X. Deep incisura (left arrowhead) of the outer side of the descending portion of the duodenum due to a fibrous band. In addition there is slight flattening of folds (right arrows) along the inner side of the proximal duodenum thought to be due to pancreatitis.

With pancreatitis (Case XI), pressure on the loop tends to be shallower and less nodular than in cancer of the pancreas. Other findings which may be encountered in this condition are irritability, enlarged sweep of the loop, spiculation along the inner loop, thickened mucosal folds, and enlarged ampulla.[8]

The pancreas is uniquely resistant to tuberculosis infection. Upper abdominal tuberculosis, in general, is basically a disease of the lymphatic system with organ involvement secondary. Only a few cases of

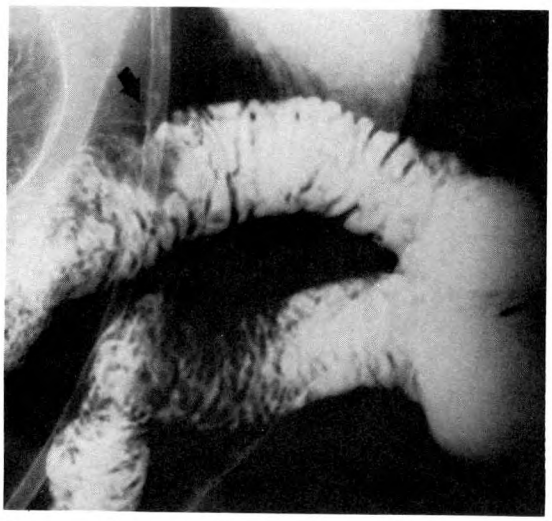


Fig. 11. Case xi. Pressure (arrow) on the small bowel at the ligament of Treitz is consistent with metastatic tumor or direct pressure from a mass in the pancreas.

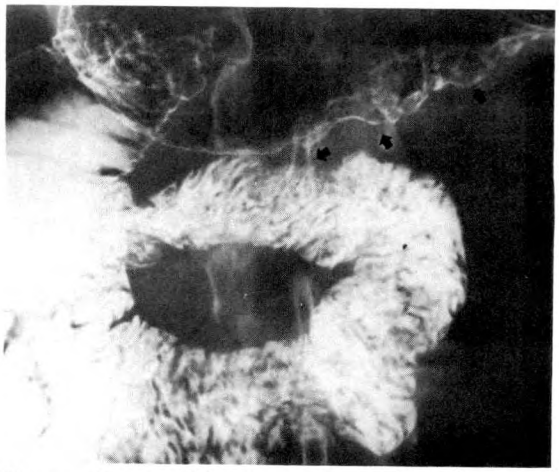


Fig. 12. Case xii. Nodular pressure (upward arrows) along the greater curvature of the stomach in addition to local pressure (lateral arrow) on the superior aspect of the small bowel just beyond the ligament of Treitz are thought to be caused by carcinoma of the body of the pancreas.

tuberculosis of the pancreas have been recorded.[5,7] Tubercles may occur in the pancreas when there is miliary tuberculosis, but large lesions are uncommon.[7] There are 3 pathologic forms of pancreatic tuberculosis: involvement of lymph nodes (tubercles), within the pancreas; tuberculous abscess; and pericanalicular sclerosis. In Case viii, involvement appears to have been of the first type. The diagnosis may be highly difficult, but when upper abdominal symptoms occur in a patient with pulmonary tuberculosis, the possibility of spread below the diaphragm should be considered. The presence of calcified lymph nodes in the abdomen is a roentgenographic clue to this possibility. Hypotonic duodenography in this case was especially helpful in demonstrating pressure on the duodenum from adjacent lymph node masses.

Carcinoma of the pancreas is an elusive disease. We have had, in the past 5 years, a total of 12 patients with carcinoma of the head of the pancreas showing nodular pressure on the inner side of the duodenal loop on the hypotonic duodenogram. Nine of the 12 had previous gastrointestinal examinations in which results were considered normal. In the other 3 patients, the results were considered "suspicious." The introduction of hypotonic duodenography in our institution represents a distinct advance towards detection of the carcinoma. Ultrasound also promises much in this area, but it is not generally available at present. Ultrasound and hypotonic duodenography are complementary procedures and may become the 2 principal methods used for diagnosing carcinoma of the pancreas.

The cases presented demonstrate that lesions other than masses may be detected by hypotonic duodenography. A normal duodenogram will often contribute significantly in clarifying equivocal findings from a previous gastrointestinal examination.

SUMMARY

Hypotonic duodenography is a simple, rewarding procedure which all radiologists involved in gastrointestinal evaluation should be using.

Its primary application is in the detection of carcinoma of the pancreas, but it is also helpful in discovering other disease entities affecting the duodenal loop.

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REFERENCES

1. Bilbao, M. K., Frische, L. H., Rösch, J., Benson, J. A., Jr., and Dotter, C. T. Postbulbar duodenal ulcer and ring-stricture; cause and effect. *Radiology*, 1971, *100*, 27–35.
2. Gelfand, D. W., and Moskowitz, M. Massive gastric dilatation complicating hypotonic duodenography; report of three cases. *Radiology*, 1970, *97*, 637–639.
3. Goldstein, H. M., and Zboralske, F. F. Tubeless hypotonic duodenography. *J.A.M.A.*, 1969, *210*, 2086–2089.
4. Lawson, T. L., and Margulis, A. R. Simplified tube control method of hypotonic duodenography. *Radiology*, 1969, *92*, 1119–1120.
5. Little, R. R. Tuberculosis of pancreas; report of case. *New England J. Med.*, 1940, *222*, 135–137.
6. Martel, W., Scholtens, P. A., and Lim, L. W. "Tubeless" hypotonic duodenography; technique, value and limitations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 119–130.
7. Paraf, A., Ménager, C., and Texier, J. La tuberculose du pancréas et la tuberculose des ganglions de l'étage supérieur de l'abdomen. *Rev. méd. chir.*, 1966, *41*, 101–126.
8. Rubinstein, Z. J., Bank, S., Marks, I. N., and Psillos, C. Hypotonic duodenography in chronic pancreatitis. *Brit. J. Radiol.*, 1971, *44*, 142–149.
9. Tréheux, A., Fays, J., and Velut, A. Apport de la duodénographie hypotonique dans le diagnostic des ulcères post-bulbaires. *Ann. de radiol.*, 1969, *12*, 451–465.

HYPOTONIC ROENTGENOGRAPHY WITH GLUCAGON*

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INDIANAPOLIS, INDIANA

HYPOTONIC duodenography was first introduced by Porcher in 1944.[27] The temporary duodenal atony and distention induced by drugs allowed for a more definitive examination of the duodenum. This method is often the procedure of choice for detecting lesions of the duodenum and pancreas.[1,2,10,11,23,28]

Recently Obata,[26] Nishizawa *et al.*,[25] and Shirakabe *et al.*[29] reported on the use of double contrast techniques, using gas producing pills to find very small cancers of the stomach. The normal stomach shows compliant and elastic rugal folds. By distending the stomach during a double-contrast examination, a profile view of the interior of the stomach is permitted and areas of relative fixation and mucosal infiltration are exaggerated. Using these techniques Nishizawa *et al.*[25] visualized 8 of 13 cancers, 5–10 mm. in diameter. Shirakabe *et al.*,[29] in a larger series collected from several institutions, reported 62 per cent of 196 early cancers less than 1 cm. in size detected on roentgenographic examination.

Porcher[27] originally used morphine to relax the duodenum. Since then anticholinergics have been given parenterally for this purpose. The procedure appears to be relatively safe and most reports of side effects are due to the common adverse effects of anticholinergic drugs. These include transient tachycardia, blurring of vision, urinary hesitancy and retention, dryness of the mouth, headache, general malaise[3,13,22,16] and, very rarely, massive gastric dilatation.[15] Since a single dose of the anticholinergic given parenterally, sufficient to induce hypotonia of the gut, is usually much larger than that ordinarily given orally, all subjects report side effects.[16]

We theorized that if an effective hypotonic gastric and duodenal study could be consistently obtained with a drug that did not have these side effects, this would be an improvement of the diagnostic method. In clinical practice it is often the elderly patient who cannot tolerate an anticholinergic that is in need of a hypotonic gastrointestinal study.

While studying the effect of glucagon on the gallbladder,[6] we observed that the duodenum was also relaxed. This latter effect had been reported by Foa and Galansino,[14] and Lawrence,[19–21] and was confirmed by us.[7] The present investigation reports 2 double-blind crossover studies comparing the effects of gastrointestinal tonicity and motility of glucagon to atropine sulfate, propantheline bromide, and placebo.

METHOD

STUDY I

Twelve asymptomatic men ranging in age from 20 to 54 years, averaging 24.4 years, participated in the study. In these men glucagon 2 mg. was compared to

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placebo (2 ml. of diluting solution for glucagon) and to atropine sulfate 1 mg. Medication was given intramuscularly, and double-blind.

STUDY II

Twelve asymptomatic men ranging in age from 20 to 54 years, averaging 27.6 years, cooperated in the study. Glucagon 2 mg. was compared to placebo (2 ml. of diluting solution for glucagon) and to propantheline bromide 30 mg. (G. D. Searle & Co.) given intramuscularly, double-blind. The medications were coded numerically in consecutive order. All subjects were ambulatory, and except for 1 subject, were not taking any other drugs. This man was on clindamycin hydrochloride 150 mg. each morning for acne. No medication was taken for 24 hours prior to the study. The subjects in both studies received each of the 3 drugs used in that study.

TECHNIQUE

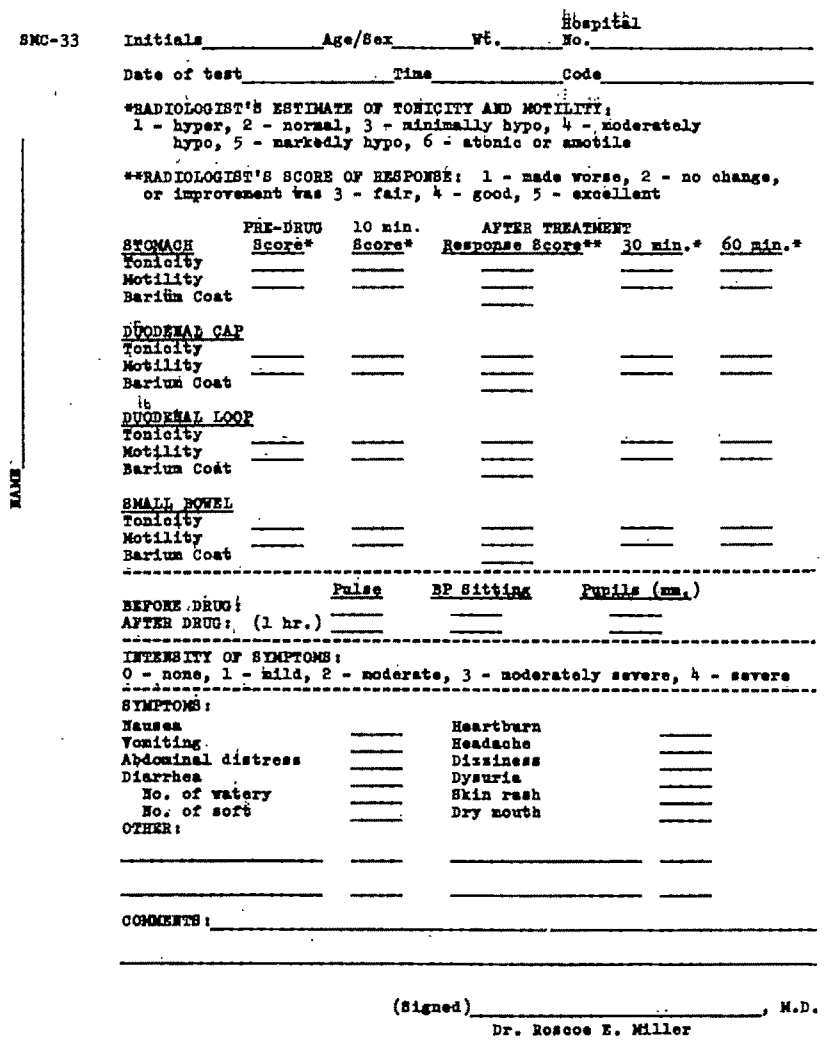
Most studies were done in the morning. However, for the few done late in the afternoon, a light liquid breakfast was allowed. No other food, fluids, or smoking were allowed until the study was completed. In the Radiology Department each subject was given one-half to one cup of barium, and the gastrointestinal tract observed fluoroscopically. Appropriate control spot roentgenograms were obtained, and medication was then given. The stomach, duodenum, and small bowel were observed at 5 and 10 minutes after dosing. At the 10 minute observation a decision was made concerning drug effect. The subject was then given barium and a teaspoonful of sodium bicarbonate, and a teaspoonful of an equal molar concentration of citric acid with lemon flavor to produce gas in the stomach. Indicated films were obtained when sufficient gas and barium were present in the stomach and small bowel. At 30 minutes, only the stomach and duodenal loop were observed for motility and tonicity; a final film was obtained at the end of 1 hour.

When examination of 6 subjects in each study had been completed (36 examinations), all the films were reviewed and a determination made concerning the effect of the drug on the tonicity and motility of the stomach, duodenal bulb, duodenal loop, and small bowel. This was repeated for the next 6 subjects (total 72 examinations). Tonicity was judged on the basis of the caliber and distensibility of the small bowel. The radiologist also determined whether examination of the upper gastrointestinal tract was enhanced by the medication and the effect, if any, of the drug on the barium coating of the gut. After each study a standard form was completed by the radiologist (Fig. 1).

The pulse, blood pressure, and pupil size were obtained just prior to and on completion of each study. Pupil size was obtained by matching the pupil to black dots 1 to 6 mm. size on a photographic paper under a standard light. The subject was questioned concerning the presence of nausea, vomiting, abdominal distress, diarrhea, heartburn, headache, dizziness, dysuria, skin rash, and dryness of the mouth on completion of the examination and the following day for any side effects that developed 4 to 6 hours after drug administration. If a positive report or spontaneous symptom was elicited, he was asked to quantitate the intensity of the symptom according to a scale of 1 to 4 (1—mild, 2—moderate, 3—moderately severe, 4—severe).

RESULTS

Since tonicity and motility scores were almost identical, only scores for tonicity will be reported. The average tonicity scores both before and at 10, 30, and 60 minutes after drug administration, the radiologist's evaluation of response, and the effect on the barium coating of the gut for the 12 subjects in Study I are presented in Table I and for the 12 subjects in Study II are presented in Table II. The distribution of the pre- and postmedication tonicity score differences at the 10 minute interval is presented graphically for both



SMC-33 Initials_____ Age/Sex_____ Wt._____ Hospital No._____

Date of test_____ Time_____ Code_____

*RADIOLOGIST'S ESTIMATE OF TONICITY AND MOTILITY:
1 - hyper, 2 - normal, 3 - minimally hypo, 4 - moderately hypo, 5 - markedly hypo, 6 - atonic or amotile

**RADIOLOGIST'S SCORE OF RESPONSE: 1 - made worse, 2 - no change, or improvement was 3 - fair, 4 - good, 5 - excellent

	PRE-DRUG	10 min.	AFTER TREATMENT		
STOMACH	Score*	Score*	Response Score**	30 min.*	60 min.*
Tonicity					
Motility					
Barium Coat					
DUODENAL CAP					
Tonicity					
Motility					
Barium Coat					
DUODENAL LOOP					
Tonicity					
Motility					
Barium Coat					
SMALL BOWEL					
Tonicity					
Motility					
Barium Coat					

	Pulse	BP Sitting	Pupils (mm.)
BEFORE DRUG:			
AFTER DRUG: (1 hr.)			

INTENSITY OF SYMPTOMS:
0 - none, 1 - mild, 2 - moderate, 3 - moderately severe, 4 - severe

SYMPTOMS:			
Nausea		Heartburn	
Vomiting		Headache	
Abdominal distress		Dizziness	
Diarrhea		Dysuria	
No. of watery		Skin rash	
No. of soft		Dry mouth	
OTHER:			

COMMENTS:_____

(Signed)_____, M.D.
Dr. Roscoe E. Miller

NAME

FIG. 1. Standard form completed for each of 72 examinations.

studies in Figure 2. Since 24 subjects each received placebo and glucagon, the results for these men were divided in half for purposes of comparison. The average difference in response for the 12 subjects in each study at 10, 30, and 60 minutes is presented graphically in Figure 3. The difference between the 10 minute tonicity score and that observed initially was added to the radiologist's score to present an over-all score for response of the gastrointestinal tract to medication (Fig. 4). The time of onset of action following the administration of medication for the 12 subjects in each study is presented in Table III.

The results of the 3 treatments were recorded for each subject and compared pairwise. Treatment preferences were then evaluated with a simple binomial test. When the results following the administration of glucagon were compared to the placebo response there was a significant ($p < 0.001$) decrease in tonicity and motility of the stomach, duodenum, and small bowel at 10 and 30 minutes. The tonicity and motility of the stomach and duodenum at 60 minutes after glucagon were still decreased in some subjects, but were not substantial enough to allow confident distinction from placebo. Small bowel tonicity and motility were also significantly ($p < 0.05$) reduced at 60 minutes after glucagon was

Table I

AVERAGE MEASURES OF EFFECTIVENESS FOR THE 12 SUBJECTS GIVEN PLACEBO, ATROPINE SULFATE 1 MG., AND GLUCAGON 2 MG. INTRAMUSCULARLY

	Before Rx T*	10 min. After Rx T*	30 min. After Rx T*	60 min. After Rx T*	Radiologist's Score T+	Barium Coating°
Placebo						
Stomach	2.0	2.3	2.3	2.3	2.3	2
Duodenal Bulb	2.0	2.6	—	2.3	2.4	2
Duodenal Loop	2.0	2.5	2.3	2.3	2.3	2
Small Intestine	2.0	2.3	—	2.2	2.3	2
Atropine Sulfate 1 mg.						
Stomach	2.0	4.0	4.6	3.9	3.2	2
Duodenal Bulb	2.3	3.8	—	4.0	3.2	2
Duodenal Loop	2.3	3.8	4.4	4.1	3.1	2
Small Intestine	2.0	4.0	—	4.0	3.4	2
Glucagon 2 mg.						
Stomach	2.0	5.7	5.3	2.7	4.7	2
Duodenal Bulb	2.1	5.7	—	2.7	4.8	2
Duodenal Loop	2.0	5.7	5.4	2.8	4.8	2
Small Intestine	2.0	4.9	—	2.8	4.1	2

(T*) tonicity scores: 1-hyper, 2-normal, 3-minimally hypo, 4-moderately hypo, 5-markedly hypo, and 6-atonic or amotile.
(T+) tonicity and coating° scores: 1-made worse, 2-no change, or improvement was 3-fair, 4-good, or 5-excellent.

Table II

AVERAGE MEASURES OF EFFECTIVENESS FOR THE 12 SUBJECTS GIVEN PLACEBO, PROPANTHELINE BROMIDE 30 MG., AND GLUCAGON 2 MG. INTRAMUSCULARLY

	Before Rx T*	10 min. After Rx T*	30 min. After Rx T*	60 min. After Rx T*	Radiologist's Score T+	Barium Coating°
Placebo						
Stomach	2.1	2.3	2.4	2.2	2.2	1.9
Duodenal Bulb	2.3	2.3	—	2.3	2.2	1.9
Duodenal Loop	2.2	2.4	2.6	2.3	2.2	1.9
Small Intestine	2.2	2.5	—	2.3	2.3	2.0
Propantheline Bromide 30 mg.						
Stomach	2.1	4.6	4.6	4.3	3.8	2.0
Duodenal Bulb	2.6	4.6	—	4.2	3.8	2.0
Duodenal Loop	2.5	4.4	4.8	4.3	3.6	2.0
Small Intestine	2.2	4.0	—	4.1	3.3	2.0
Glucagon 2 mg.						
Stomach	2.0	5.8	5.2	2.9	4.8	2.0
Duodenal Bulb	2.2	5.9	—	3.0	4.9	2.0
Duodenal Loop	2.3	5.9	5.3	2.9	4.9	2.0
Small Intestine	2.2	5.5	—	2.8	4.6	2.0

(T*) tonicity scores: 1-hyper, 2-normal, 3-minimally hypo, 4-moderately hypo, 5-markedly hypo, and 6-atonic or amotile.
(T+) tonicity and coating° scores: 1-made worse, 2-no change, or improvement was 3-fair, 4-good, or 5-excellent.

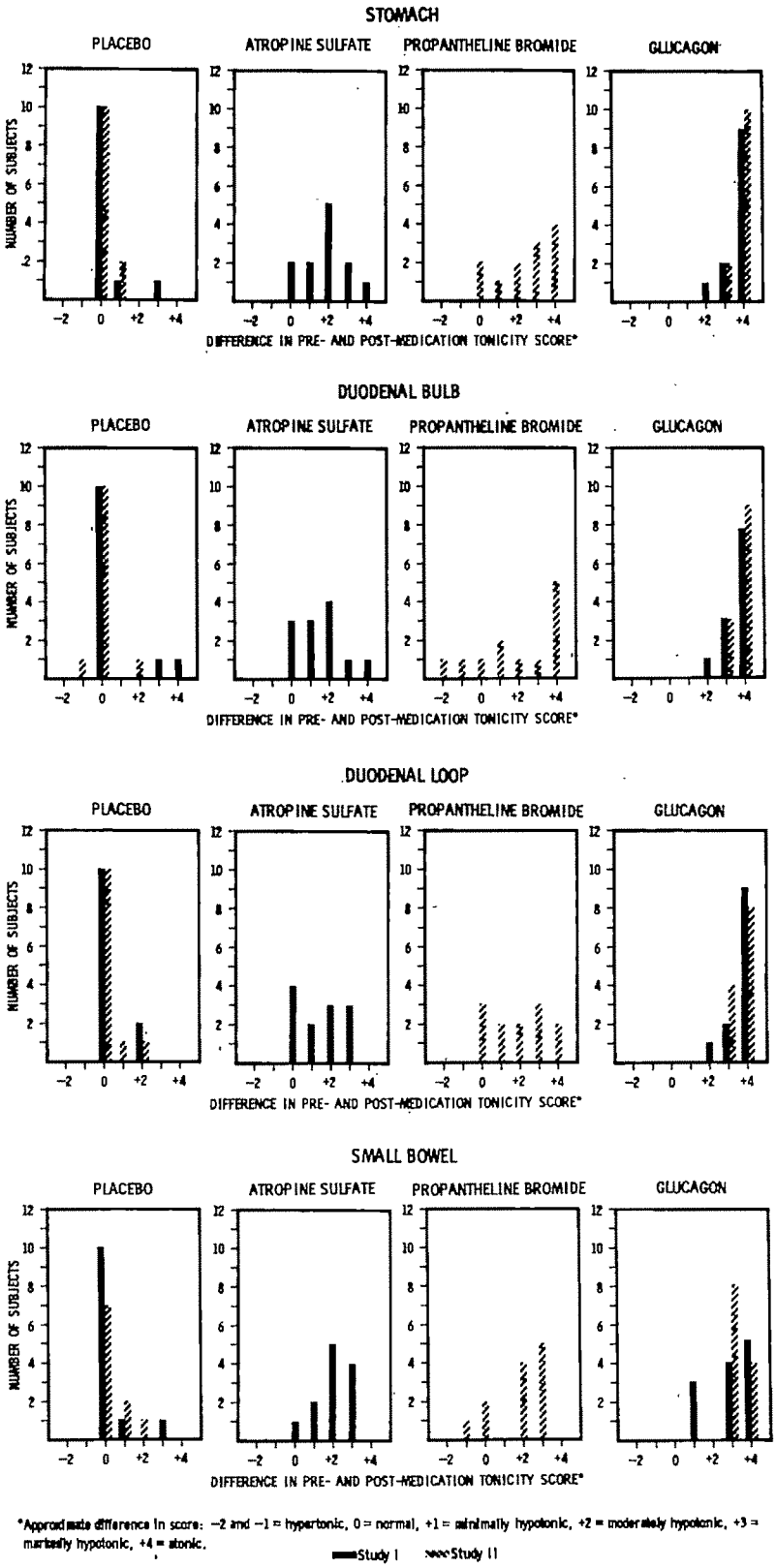
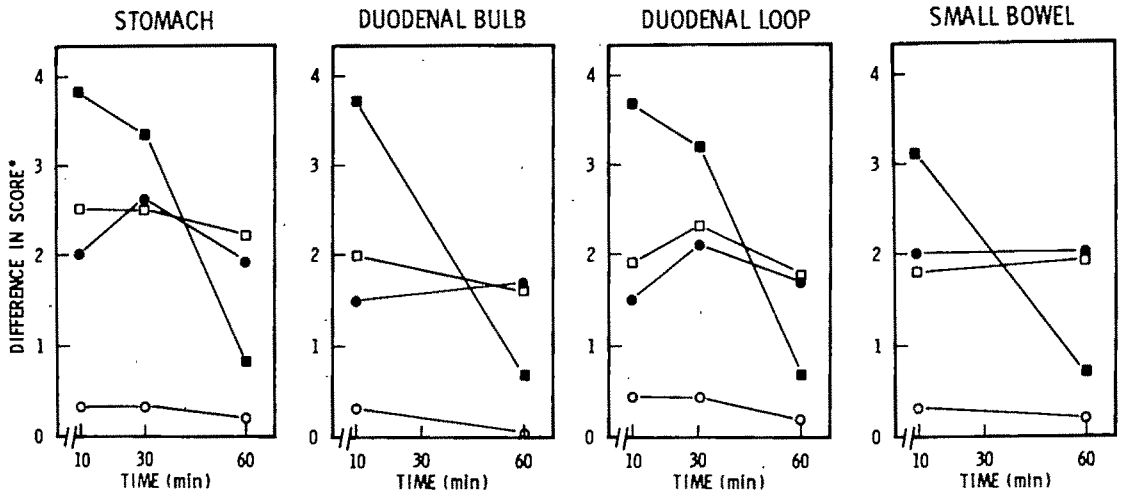


FIG. 2. Distribution of subjects according to difference between initial and 10 minute postmedication scores* for tonicity of the stomach, duodenal bulb and loop, and small bowel.



*Approximate difference in score: 0 = normal, 1 = minimally hypotonic, 2 = moderately hypotonic, 3 = markedly hypotonic, and 4 = atonic.

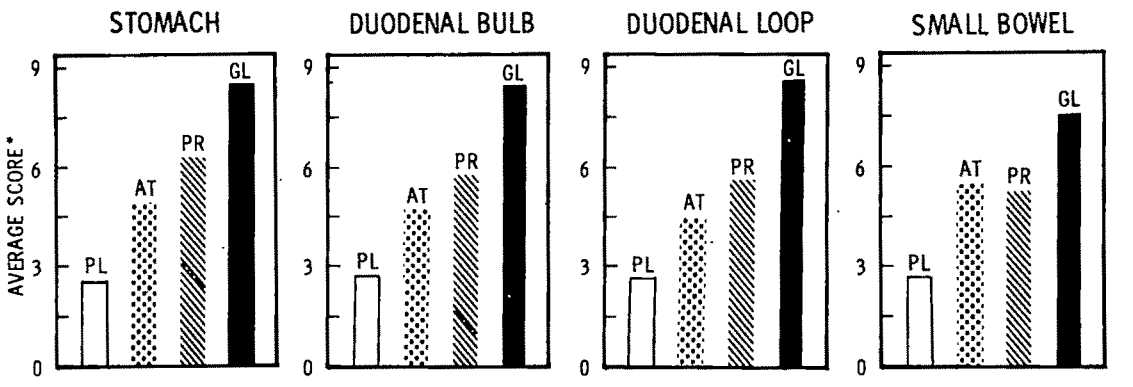
Key: Placebo (○), atropine sulfate (●), propantheline bromide (□), and glucagon (■).

FIG. 3. Average difference between pre- and postmedication tonicity score* as a function of elapsed time.

given as compared to placebo. There was a greater decrease ($p < 0.05$) in the tonicity and motility of the stomach and duodenum at the 10 minute interval after glucagon was given than following the administration of either anticholinergic. Small bowel tonicity and motility were also decreased to a greater extent with glucagon than with propantheline bromide. There was no difference observed between glucagon and atropine sulfate at 10 minutes in their effects on the small bowel.

Atropine sulfate significantly ($p < 0.05$) decreased tonicity and motility of the stomach and small bowel at 10, 30, and 60 minutes, of the duodenal bulb at 60 minutes, and of the duodenal loop at 30 and 60 minutes.

When compared to placebo, propantheline bromide decreased ($p < 0.05$) the tonicity and motility of the stomach, duodenal loop, and small bowel at 10, 30, and 60 minutes, and of the duodenal bulb at 60 minutes (Table IV). The results for the



*Radiologist's score + 10-minute tonicity − initial tonicity score.

FIG. 4. The over-all score* for the stomach, duodenal bulb and loop, and small bowel for 12 subjects in each of 2 studies after placebo (PL), atropine sulfate 1 mg. (AT), propantheline bromide 30 mg. (PR), and glucagon 2 mg. (GL).

TABLE III

TIME OF ONSET IN MINUTES OF EFFECT OF MEDICATION AS MEASURED BY EVALUATION OF THE DUODENAL LOOP

Subject	Study I			Study II		
	Placebo	*Atropine Sulfate*	*Glucagon*	*Placebo*	*Propantheline Bromide*	*Glucagon*
1	5	10	5	0	15	5
2	0	10	10	10	7	5
3	0	15	5	0	0	15
4	6	15	5	0	10	5
5	0	15	5	30	30	5
6	0	60	5	0	10	5
7	10	15	5	0	10	5
8	30	30	5	15	5	5
9	0	10	10	0	10	5
10	30	5	10	30	5	5
11	0	30	5	30	10	5
12	0	30	5	0	10	10

radiologist's score and for the over-all score were the same as those for tonicity at the 10 minute interval.

In most subjects decrease in tonicity and motility was evident approximately 5 minutes after the administration of glucagon. This varied from 5 to 30 minutes for the anticholinergics (Table III).

On each study day the pulse, blood pressure, and pupil size were measured for each subject before and 1 hour after drug administration. After the administration of 30 mg. of propantheline bromide or 1 mg. of atropine sulfate, the pulse rate was significantly ($p < 0.05$) increased at 1 hour as compared to control (Table V). No other significant changes were obtained in these 3 variables for any other treatments.

All subjects were questioned concerning other symptoms. The responses to these questions are presented in Table VI. Because some subjects reported more than one side effect the total number may be greater than 12. To combine the results for the 2 studies into 1 table, the responses for the 24 subjects given placebo and glucagon were divided in half. The figures are therefore on a "per study" basis. Following the administration of placebo, there were 2 reports of side effects.

Following atropine sulfate there were 16 reports of side effects. Eight subjects reported dryness of the mouth usually lasting approximately 4 hours, 3 reported nausea, and 1 each reported heartburn, vomiting, dizziness, sore throat, and flushing.

After the administration of propantheline bromide there were 52 reports of side effects. All subjects reported moderately severe to severe dryness of the mouth, and 10 had urinary retention that lasted 4 to 8 hours. Six had headache, 5 nausea, 3 each dizziness and dysuria, 2 each abdominal distress and flushing, and 1 each vomiting, blurred vision, sneezing, hiccups, nasal burning, drowsiness, generalized malaise, urinary dribbling, and tinnitus. Using the simple binomial test, reports of dryness of the mouth were significantly ($p < 0.001$) more frequent following the administration of propantheline bromide than following placebo.

When glucagon was given, there were 17 reports of side effects. These consisted of 7.5 reports of nausea, 4.5 of vomiting, 1.5 of dizziness, 1.5 of flushing, and 0.5 each of abdominal distress, headache, dryness of the mouth, and weakness. The nausea and vomiting reported after glucagon occurred approximately 2 hours after the drug was given and lasted approximately 5 to 10 minutes.

Table IV

RESULTS OF BINOMIAL COMPARISON

	Tonicity and Motility at:		
	10 min. p<	30 min. p<	60 min. p<
Comparisons		*Stomach*	
Glucagon to Placebo	0.001	0.001	N.S.
Atropine Sulfate to Placebo	0.05	0.01	0.01
Glucagon to Atropine Sulfate	0.01	N.S.	0.05
Glucagon to Placebo	0.001	0.001	N.S.
Propantheline Br. to Placebo	0.001	0.05	0.001
Glucagon to Propantheline Br.	0.05	N.S.	0.01
		Duodenal Bulb	
Glucagon to Placebo	0.001	—	N.S.
Atropine Sulfate to Placebo	N.S.	—	0.05
Glucagon to Atropine Sulfate	0.001	—	N.S.
Glucagon to Placebo	0.001	—	N.S.
Propantheline Br. to Placebo	N.S.	—	0.01
Glucagon to Propantheline Br.	0.05	—	N.S.
		Duodenal Loop	
Glucagon to Placebo	0.001	0.001	N.S.
Atropine Sulfate to Placebo	N.S.	0.05	0.05
Glucagon to Atropine Sulfate	0.001	0.01	N.S.
Glucagon to Placebo	0.001	0.01	N.S.
Propantheline Br. to Placebo	0.05	0.05	0.01
Glucagon to Propantheline Br.	0.01	N.S.	0.05
		Small Bowel	
Glucagon to Placebo	0.001	—	0.05
Atropine Sulfate to Placebo	0.05	—	0.001
Glucagon to Atropine Sulfate	N.S.	—	0.05
Glucagon to Placebo	0.001	—	N.S.
Propantheline Br. to Placebo	0.05	—	0.001
Glucagon to Propantheline Br.	0.01	—	0.05

Table V

THE AVERAGE PULSE, SYSTOLIC AND DIASTOLIC BLOOD PRESSURE, AND PUPILLARY SIZE BEFORE AND AT 60 MINUTES FOLLOWING THE INTRAMUSCULAR INJECTION OF PLACEBO, ATROPINE SULFATE 1 MG., PROPANTHELINE BROMIDE 30 MG., AND GLUCAGON 2 MG.

	Placebo		*Atropine Sulfate*		*Propantheline Bromide*		*Glucagon*	
	Before Rx	After Rx	Before Rx	After Rx	Before Rx	After Rx	Before Rx	After Rx
Pulse	68	70	70	90*	70	118*	70	72
Blood Pressure								
Systolic	126	127	123	128	121	129	125	125
Diastolic	82	80	78	85	79	89	83	82
Pupil Size	3.7	3.7	3.8	4.0	3.7	4.1	3.5	3.4

* Significant ($p<0.05$).

TABLE VI

FREQUENCY AND INTENSITY OF SIDE EFFECTS
TWENTY-FOUR SUBJECTS RECEIVED GLUCAGON AND PLACEBO. THE TOTAL FOR THESE 2 TREATMENTS WAS DIVIDED IN HALF IN THIS TABLE TO ALLOW COMPARABILITY ON A WITHIN STUDY BASIS

Symptoms	*Placebo*		*Atropine Sulfate*		*Propantheline Bromide*		*Glucagon*	
	No. of Reports	Total Score*	No. of Reports	Total Score*	No. of Reports	Total Score*	No. of Reports	Total Score*
Nausea			3	3	5	9	7.5	13.5
Vomiting			1	1	1	1	4.5	5
Heartburn			1	1				
Abdominal distress	0.5	0.5			2	3	0.5	0.5
Headache	1	1			6	8	0.5	0.5
Dizziness	0.5	0.5	1	1	3	3	1.5	1.5
Dysuria					3	10		
Dry mouth			8	17	12	42	0.5	1
Weakness							0.5	0.5
Flushing			1	2	2	4	1.5	3
Sore throat			1	2				
Urinary retention					10	37		
Urinary dribbling					1	3		
Tinnitus					1	3		
General malaise					1	4		
Blurred vision					1	1		
Sneezing					1	1		
Hiccups					1	1		
Nasal burning					1	3		
Drowsiness					1	1		
Total	2	2	16	27	52	134	17.0	25.5

* 0-none; 1-mild; 2-moderate; 3-moderately severe; and 4-severe.

DISCUSSION

Glucagon is a crystalline polypeptide with a molecular weight of 3,485 and is composed of 29 amino acid residues.[4] The commercially available beef and pork glucagon have exactly the same amino acid sequence as human glucagon. The hormone is normally produced in the alpha cells of the islands of Langerhans and is smaller than insulin. Because the molecule is small, it does not produce a high titer of antibodies unless it is conjugated to a large protein. Although it may be possible to develop a sensitivity to glucagon, it is rare.

Bilbao *et al.*,[3] and Stage and Banke[30] stated that they do not give anticholinergic drugs to patients in whom they may be contraindicated. This included patients with glaucoma, urinary retention, uncompensated cardiac disease, and severe angina pectoris.

Glucagon does not have the above contraindications and may be given intramuscularly to such patients with little or no difficulty. For hypotonic studies it is contraindicated, if an insulinoma[19,20] and/or pheochromocytoma[21] is suspected. In patients with an insulinoma, glucagon given intravenously may cause hypoglycemia because of its insulin releasing effect rather than induce the expected rise in blood sugar. It may possibly be used as a test for this condition. Exogenous glucagon stimulates the release of catecholamines and has been used diagnostically as a provocative agent in selected patients with pheochromocytoma. In such patients 5 mg. of phentolamine mesylate intravenously may be tried to control the blood pressure.

Jones *et al.*[17] reported that when glucagon was given to dogs there was an increased flow of bile. Dyck and Janowitz[9] reported

that in T-tube patients the flow of bile increased following treatment with glucagon. Dreiling and Janowitz,[8] and Lawrence[19] reported a decrease in gastric and pancreatic secretion after the administration of glucagon. Jones and Hall[18] reported that with continuous intravenous infusion of glucagon there was an increase in the volume of secretion from Brunner's gland pouches of the dog (maximum 1.4 ml./15 min. after 64 μg./kg./hr.). In these studies of hypotonic roentgenography there was no significant effect of any of the drugs on the barium coating of the duodenum or small bowel. Thus, if there was an increase in the secretion from Brunner's glands and an increase in the flow of bile from the liver following the administration of glucagon, it was not sufficient to affect the barium coating of the mucosa.

In *in vivo* studies endogenous glucagon has been reported to inhibit gastric motility in man[31] and dog.[24] In *in vitro* studies glucagon in a dose of as great as 1 μg./ml. had no effect on strips of antral and body muscle of the human stomach.[5] In *in vivo* studies of the cat Fasth and Hulten[12] found that glucagon administered intraarterially was followed by an immediate increase in intestinal blood flow and a somewhat delayed motility inhibition and blood pressure increase. The motility inhibition and blood pressure increase were abolished when the adrenals were excluded by encircling ligatures. The intestinal blood flow increase remained virtually the same. The authors concluded that glucagon has a direct effect on vascular smooth muscle and by a release of catecholamines from the adrenal medulla inhibits the activity of the intestinal smooth muscles.

SUMMARY AND CONCLUSION

Twelve asymptomatic subjects cooperated in each of 2 studies. In Study I glucagon was compared to placebo and to atropine sulfate 1 mg. In Study II glucagon 2 mg. was compared to placebo and to propantheline bromide 30 mg. Drugs were given intramuscularly, double-blind, and crossover.

When the subjects received glucagon, the radiologist reported that motility and tonicity were decreased and that the drug effect was significantly ($p < 0.001$) greater at 10 and 30 minutes than when placebo was given. Sixty minutes after receiving glucagon the tonicity and motility of the stomach and duodenum differed little from that seen with placebo. Small bowel motility was also significantly ($p < 0.05$) less after glucagon than after placebo.

Stomach and small bowel tonicity and motility were significantly ($p < 0.05$) decreased following atropine sulfate and propantheline bromide at 10, 30, and 60 minutes as compared to placebo. Tonicity and motility of the duodenal bulb at 60 minutes and duodenal loop at 30 and 60 minutes were also decreased ($p < 0.05$) following atropine sulfate administration. A similar effect was observed after propantheline bromide was given; the onset of action was somewhat sooner following propantheline bromide than after atropine sulfate. Glucagon was significantly ($p < 0.05$) better than either anticholinergic in the examination of the stomach, duodenal bulb, and duodenal loop.

Pulse, blood pressure, and pupil size were obtained at each session and all subjects were questioned concerning side effects. After the administration of propantheline bromide, all subjects reported dryness of the mouth, 5 experienced nausea, 6 reported headaches, and 10 had urinary retention for 4 to 6 hours. After atropine sulfate there were 8 reports of dryness of the mouth and 3 of nausea. When glucagon was given, there was an average of 7.5 reports of nausea and 4.5 reports of vomiting.

The results of this study indicate that the decrease in the tonicity and motility of the stomach, duodenum, and small bowel was more predictable and the side effects were fewer with 2 mg. of glucagon than with 1 mg. of atropine sulfate or 30 mg. of propantheline bromide.

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REFERENCES

1. BAUM, M., and HOWE, C. T. Hypotonic duodenography in diagnosis of carcinoma of pancreas and its further use when combined with percutaneous cholangiography and pancreatic scintiscanning. *Am. J. Surg.*, 1968, *115*, 519–525.
2. BILBAO, M. K., RÖSCH, J., FRISCHE, L. H., *et al.* Hypotonic duodenography in diagnosis of pancreatic disease. *Seminars in Roentgenol.*, 1968, *3*, 280–287.
3. BILBAO, M. K., FRISCHE, L. H., DOTTER, C. T., and RÖSCH, J. Hypotonic duodenography. *Radiology*, 1967, *89*, 438–443.
4. BROMER, W. Glucagon: chemistry and action. *Fortschr. Chem. Organ. Naturst.*, 1970, *28*, 429–452.
5. CAMERON, A. J., PHILLIPS, S. F., and SUMMERSKILL, W. H. J. Comparison of effects of gastrin, cholecystokinin-pancreozymin, secretin, and glucagon on human stomach muscle in vitro. *Gastroenterology*, 1970, *59*, 539–545.
6. CHERNISH, S. M., MILLER, R. E., ROSENAK, B. D., and SCHOLZ, N. E. Effect of glucagon on size of visualized human gallbladder before and after fat meal. *Gastroenterology*, 1972, *62*, 1218–1226.
7. CHERNISH, S. M., MILLER, R. E., ROSENAK, B. D., and SCHOLZ, N. E. Hypotonic duodenography with use of glucagon. *Gastroenterology*, 1972, *63*, 392–398.
8. DREILING, D. A., and JANOWITZ, H. D. Effect of glucagon on gastric secretion in man. *Gastroenterology*, 1959, *36*, 580–581.
9. DYCK, W. P., and JANOWITZ, H. D. Effect of glucagon on hepatic bile secretion in man. *Gastroenterology*, 1971, *60*, 400–404.
10. EATON, S. B., FLEISCHLI, D. J., POLLARD, J. J., NEBESAR, R. A., and POTSAID, N. S. Comparison of current radiologic approaches to diagnosis of pancreatic disease. *New England J. Med.*, 1968, *279*, 389–396.
11. EATON, S. B., BENEDICT, K. T., FERRUCCI, J. T., JR., and FLEISCHLI, D. J. Hypotonic duodenography. *Radiol. Clin. North America*, 1970, *8*, 125–137.
12. FASTH, S., and HULTEN, L. Effect of glucagon on intestinal motility and blood flow. *Acta physiol. scandinav.*, 1971, *83*, 169–173.
13. FERRUCCI, J. T., JR., and BENEDICT, K. T., JR. Anticholinergic-aided study of gastrointestinal tract. *Radiol. Clin. North America*, 1971, *9*, 23–29.
14. FOA, P., and GALANSINO, G. Glucagon: Chemistry and Function in Health and Disease. Charles C Thomas, Springfield, Ill., 1962.
15. GELFAND, D. W., and MOSKOWITZ, M. Massive gastric dilatation complicating hypotonic duodenography. *Radiology*, 1970, *97*, 637–639.
16. GOLDSTEIN, H. M., and ZBORALSKE, F. F. Tubeless hypotonic duodenography. *J.A.M.A.*, 1969, *210*, 2086–2088.
17. JONES, R. S., GEIST, R. E., and HALL, A. D. Choleretic effects of glucagon and secretin in dog. *Gastroenterology*, 1971, *60*, 64–68.
18. JONES, R. S., and HALL, A. D. Effect of glucagon on Brunner's gland secretion in dogs (34386). *Proc. Soc. Exper. Biol. & Med.*, 1969, *132*, 1151–1159.
19. LAWRENCE, A. M. Glucagon. *Ann. Rev. Med.*, 1969, *20*, 207–223.
20. LAWRENCE, A. M. Glucagon in medicine: new ideas for old hormone. *M. Clin. North America*, 1970, *54*, 183–190.
21. LAWRENCE, A. M., and FARLAND, M. Glucagon: provocative test for pheochromocytoma. *J. Lab. & Clin. Med.* (Abstr.), 1974, *64*, p. 878.
22. LIEBER, A., SCHAEFER, J. W., and BELIN, R. P. Hypotonic duodenography. *J.A.M.A.*, 1968, *203*, 425–427.
23. MARTEL, W. Technical note: hypotonic duodenography without intubation. *Radiology*, 1968, *91*, 387.
24. NECHELES, H., SPARN, J., and WALKER, L. Effect of glucagon on gastrointestinal motility. *Am. J. Gastroenterol.*, 1966, *45*, 29–34.
25. NISHIZAWA, M., ITO, T., and NOMOTO, K. X-ray demonstrability of microcarcinoma. *Stomach & Intestine*, (Tokyo), 1970, *5*, 951–959.
26. OBATA, W. G. Double-contrast technique for examination of stomach using barium sulfate with simethicone. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *115*, 275–280.
27. PORCHER, P. La stase duodénale provoquée, procédé simple, rapide et fiele d'améliorer visibilité radiologique et details de l'image du bulbe ulcereux. *Arch. Mal. App. Digest.*, 1944, *33*, 24–26. Cited by Bilbao, *et al.*[3]
28. RÖSCH, J. Roentgenologic diagnosis of pancreatic disease. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *100*, 664–672.
29. SHIRAKABE, H., KODOKORO, T., and TAKAGI, K. Statistical figures on very small gastric carcinoma under 1 cm. in diameter preoperatively diagnosed in Japan. *Stomach & Intestine*, (Tokyo), 1970, *5*, 995–998.
30. STAGE, P., and BANKE, L. Hypotonic duodenography. *Gut*, 1969, *10*, 428–432.
31. STUNKARD, A. J., VAN ITALLIE, T. B., and REIS, B. B. Mechanism of satiety: effect of glucagon on gastric hunger contractions in man. *Proc. Soc. Exper. Biol. & Med.*, 1955, *89*, 258–261.

Vol. 121, No. 2

COLLAGENOUS SPRUE*

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COLLAGENOUS sprue is a disease of unknown cause that is characterized histologically by a flat mucosa and a prominent deposit of collagen lying beneath the surface epithelium and extending into the lamina propria. No detailed description of the roentgenographic features of this disease has been reported.

Schein[13] described a patient with severe malabsorption, a histologically flat mucosal lesion, and submucosal deposit of a hyaline material, previously unreported in celiac sprue. Subsequently, Hourihane,[6] Neale,[4] and Barry *et al.*[1] described patients with extensive flat mucosa, severe malabsorption, and submucosal collagen that varied from prominent subepithelial bands to complete replacement of villi and crypts. Weinstein *et al.*[14] described a similar case and referred to the constellation of severe diarrhea and steatorrhea and a histologically flat mucosal lesion with submucosal collagen as collagenous sprue.

Three of our patients found to have collagenous sprue are added, below, to the 6 previously described in the literature.

REPORT OF CASES

CASE I. A 78 year old man was in good health until December 1966, when he first had watery, brownish-orange, greasy, foul-smelling diarrhea without blood, pus, or mucus. He had 5 to 6 bowel movements per day without associated tenesmus or abdominal pain. Subsequent attacks occurred approximately monthly and lasted 4 to 5 days.

Three years before admission (July 1968), a biopsy specimen of the small bowel was interpreted as "sprue" and the patient was placed on a gluten-free diet. Despite this diet, the pattern of his episodic diarrhea was unchanged. He lost 40 pounds in 4 years.

Laboratory data obtained in September 1971 included hemoglobin, 12.3 g./dl.; hematocrit, 36 per cent; serum B-12, 220 pg./ml.; folate, 3 ng./ml.; calcium, 7.6 and 8.3 mg./dl.; and phosphorus 3.6 mg./dl. Five hour urinary D-xylose excretion was 0.1 g. after a 25 g. dose. A biopsy of the small bowel (Fig. 1) gave findings again consistent with sprue and the patient was again placed on a gluten-free diet.

Persistent diarrhea (4 to 6 stools per day) continued while the patient was on this diet. He lost 5 more pounds in 2 months. He related that he had been drinking one-half gallon of milk per day. Milk was eliminated from his diet, and he promptly gained 2 pounds and described one "normal" stool per day. A serum calcium value of 5.9 mg./dl. and hypomagnesemia, detected at an outpatient visit, led to hospitalization on December 1, 1971. Examination of the upper gastrointestinal tract and follow-up study of the small bowel were undertaken on December 30, 1971 (Fig. 2). The stomach was normal. Abnormally thickened folds were identified in the proximal small bowel, most prominent in the proximal duodenum. No small bowel dilatation or flocculation of barium was evident. Repeat peroral small bowel biopsy specimen was interpreted as showing collagenous sprue. Review of the earlier biopsy specimens showed an increase in submucosal collagen to have been present. The patient has done relatively well on a gluten-free and milk-free diet.

CASE II. In good health until 1963, this man, at 38 years, began having attacks of cramping abdominal pain associated with severe watery diarrhea and occasional nausea and vomiting.

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FIG. 1. Case 1. Jejunal biopsy specimen, obtained September 1971, shows flat mucosa. Collagen (between arrows) has infiltrated the lamina propria extensively, and increased in amount and distribution since the initial biopsy (July 1968). (Trichrome, ×100.)

He had weight loss with each episode. He was hospitalized on many occasions since the onset of his symptoms. His stomach was normal on examination of the upper gastrointestinal tract. Small bowel examination (Fig. 3*A*) showed pronounced mucosal thickening of the duodenum and proximal jejunum with a smooth appearance of the proximal small bowel.

Because of steatorrhea in 1964, numerous small bowel biopsies were performed; histologic specimens showed a flat mucosa without villi, abnormal surface epithelial cells, and a rather dense deposit of collagen in the lamina propria.

Symptoms of steatorrhea, abdominal pain,

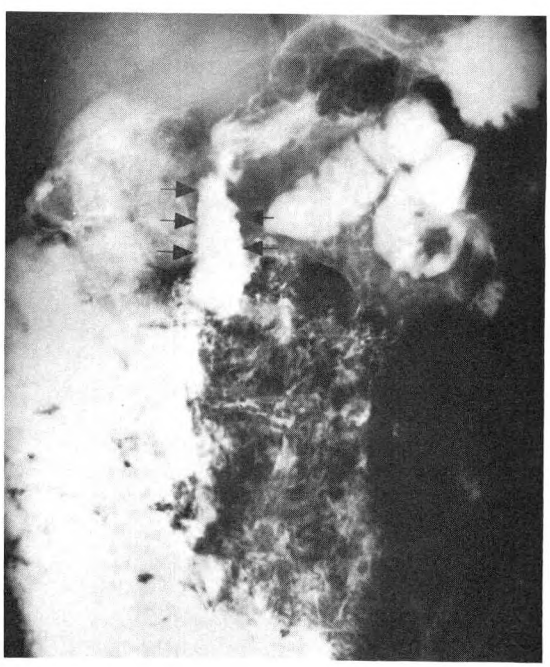


FIG. 2. Case 1. Upper gastrointestinal series (December 1971). Pronounced thickening of folds in the descending duodenum (arrows) can be seen. There is no evidence of dilatation of the small bowel, flocculation, or segmentation of the barium column. Barium in the distal small bowel appears diluted.

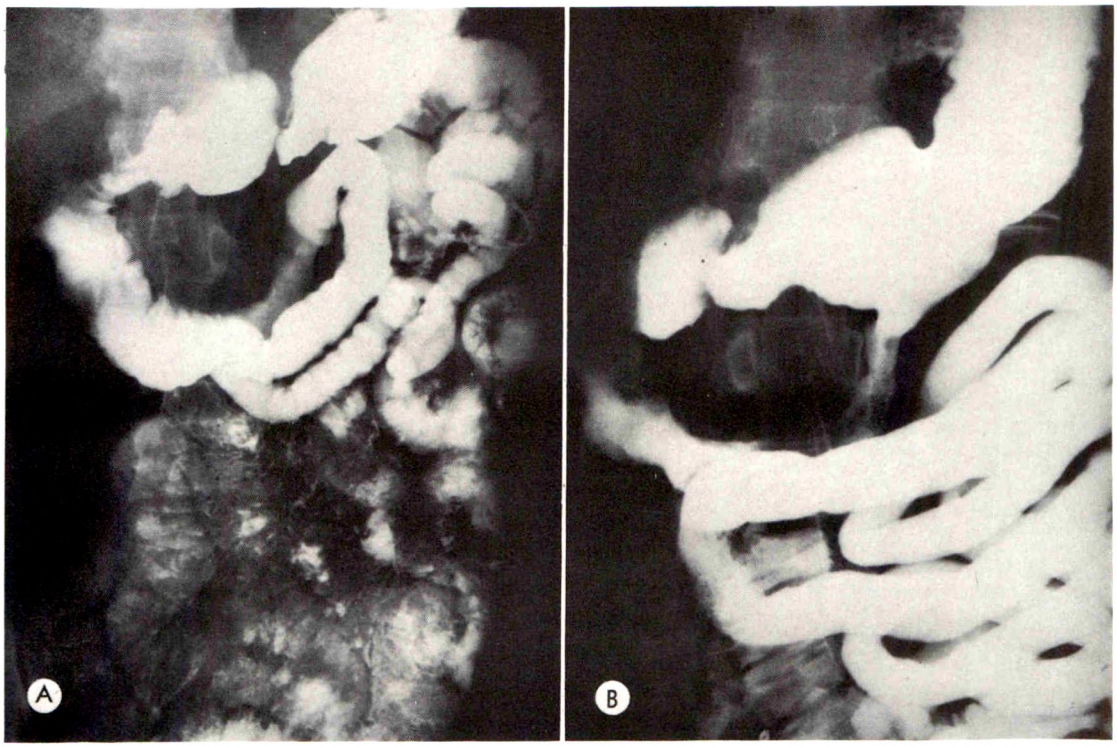


FIG. 3. Case II. (*A*) Upper gastrointestinal series reveals extensive mucosal thickening of the duodenum and proximal jejunum. Dilution of barium can be observed in the distal small bowel. No dilatation of the small bowel is evident. (*B*) Seven years later, a tubular and featureless duodenum extending to the midjejunum is evident. (Reproduced, with permission, from Sleisenger, M. H., and Fordtran, J. S. Gastrointestinal Disease: Pathophysiology, Diagnosis, Management. W. B. Saunders Company, Philadelphia, 1973, p. 878.)

and weight loss persisted despite a trial period on a gluten-free diet. In 1971, a tubular and featureless duodenum extending to the mid jejunum was noted (Fig. 3*B*).

In view of the ill-defined response to gluten withdrawal, the patient was treated intramuscularly with adrenocorticotrophic hormone (ACTH), resulting in immediate relief of his diarrhea and abdominal pain. Several days later, oral prednisone (5 mg. twice daily) was substituted for the ACTH. He was then put on Azulfidine* (500 mg. daily). Within 2 months he had gained 10 pounds. Since 1969, he has been kept on prednisone and Azulfidine. Infrequent episodes of diarrhea have been treated with Lomotil.† He had several episodes of abdominal pain, nausea, vomiting, and blood-streaked stools, which were successfully treated with an antacid regimen. He is otherwise in relatively good health.

* Salicylazosulfapyridine. Manufactured by Pharmacia Laboratories, Inc., 800 Centennial Avenue, Piscataway, N. J. 08854.

† Diphenorcylate hydrochloride. Manufactured by Searle and Company, San Juan, Puerto Rico 00936.

CASE III. This patient is a 34 year old man with diabetes mellitus who, for 18 months, was managed daily on 38 units of NPH insulin (isophane insulin suspension) and a 2,200 calorie diet. For 2 weeks before admission to the hospital, he had 4+ glycosuria.

The patient was admitted to the hospital's intensive care unit for management of ketoacidosis. Physical examination disclosed nothing abnormal. Laboratory data that were also within normal limits included values for hematocrit, sodium, potassium, CO_2, chloride, prothrombin time, partial thromboplastin time, total protein, albumin, calcium, phosphorus, creatinine, uric acid, and bilirubin. White blood cell count was 9,300/mm.^{3} with 55 per cent leukocytes. Blood sugar was 324 mg./dl. Blood urea nitrogen was 27 mg./dl. Liver function studies showed slight abnormality.

Because of 3+ to 4+ guaiac positive stools, barium contrast studies were performed. Barium enema examination gave normal findings. Examination of the upper gastrointestinal tract

(Fig. 4, *A–C*) showed the esophagus and stomach also to be normal. Pronounced mucosal thickening, however, was present in the first and second portions of the duodenum. Solitary, nodular defects were identified in the mid jejunum (Fig. 4*D*).

On the small bowel biopsy specimen a flat mucosa was seen, compatible with celiac sprue. Abnormal accumulations of collagen were seen in the lamina propria on the trichrome stained sections. Provisional diagnosis was collagenous sprue. On re-questioning, the patient described frequent, loose stools. He was placed on a gluten-free diet and responded with return of normal bowel habits.

DISCUSSION

Previous reports[1,2,4,6,13,14] have described the lack of response, or rather the ill-defined response, of collagenous sprue to a gluten-free diet. It appears that the prognosis for collagenous sprue is poorer than for celiac sprue. Although collagenous sprue is somewhat rare, these facts, in addition to the dramatic increase of collagen within the lamina propria, justify its differentiation from celiac sprue.

ROENTGENOGRAPHIC APPEARANCE

The roentgenographic features of celiac sprue have been described.[7,8] The etiologic basis for these changes is uncertain. Hypokalemia and hypoproteinemia have been implicated as contributing factors in the generalized dilatation of the jejunum and increased transit time of barium through the small bowel. Segmentation and flocculation of the barium column, as well as moulage formation, have been noted to occur with the use of USP* barium. With newer barium preparations, one rarely, if ever, sees flocculation, segmentation, or moulage formation. Although thickened folds are not a general feature of sprue, prominent, coarsened, or thickened mucosal folds may be scattered throughout the involved segments of small bowel. More frequently, the jejunal pattern is that of decreased prominence of the valvulae conniventes and small bowel dilatation. Whether this represents the flat mucosa or marked thickening is uncertain. In 25 of 30 cases of celiac sprue, Nicolette and Tully[10] have observed duodenal involvement, consisting of thickened mucosal folds in the first and second portions of the duodenum.

The roentgenographic features of collagenous sprue in the patients of Schein[13] and Barry *et al.*[1] consisted of dilatation, flocculation, and moulage formation (Table 1). In 1970, Weinstein *et al.*[14] failed to describe the characteristics presently attributable to the older barium preparations (segmentation, flocculation) and described, in collagenous sprue, only small bowel dilatation and subsequent over-all shortening of the small bowel. In Neale's[4] patient with collagenous sprue, there was marked, uniform reduction in jejunal caliber with minimal dilatation in the distal small bowel.

In our 3 cases, dilatation of the proximal intestine was not a consistent roentgenographic feature. Flocculation, segmentation, and moulage formation were absent. Abnormal intestinal appearance was localized to the proximal small bowel, most marked in the duodenum. In 2 of our cases, the first and second portions of the duodenum harbored the thickest mucosal folds. These changes were still evident, but diminished, in the remaining duodenal segments and proximal jejunum. In Case 11, initial roentgenographic evaluation revealed thickened folds in the proximal duodenum and an atrophic appearance of the proximal jejunum. The barium was diluted in the distal small bowel. Subsequent roentgenograms showed virtually total mucosal effacement, and a smooth featureless proximal bowel from the first portion of the duodenum extending to the mid jejunum. This appearance is quite similar to that described by Neale.[4]

Evidently, a characteristic roentgenographic pattern has not been established for collagenous sprue. Indeed, it is apparent from our cases that dilatation is not necessarily a feature of this entity, as it often is in celiac sprue. The only consistent roent-

* United States Pharmacopeia.

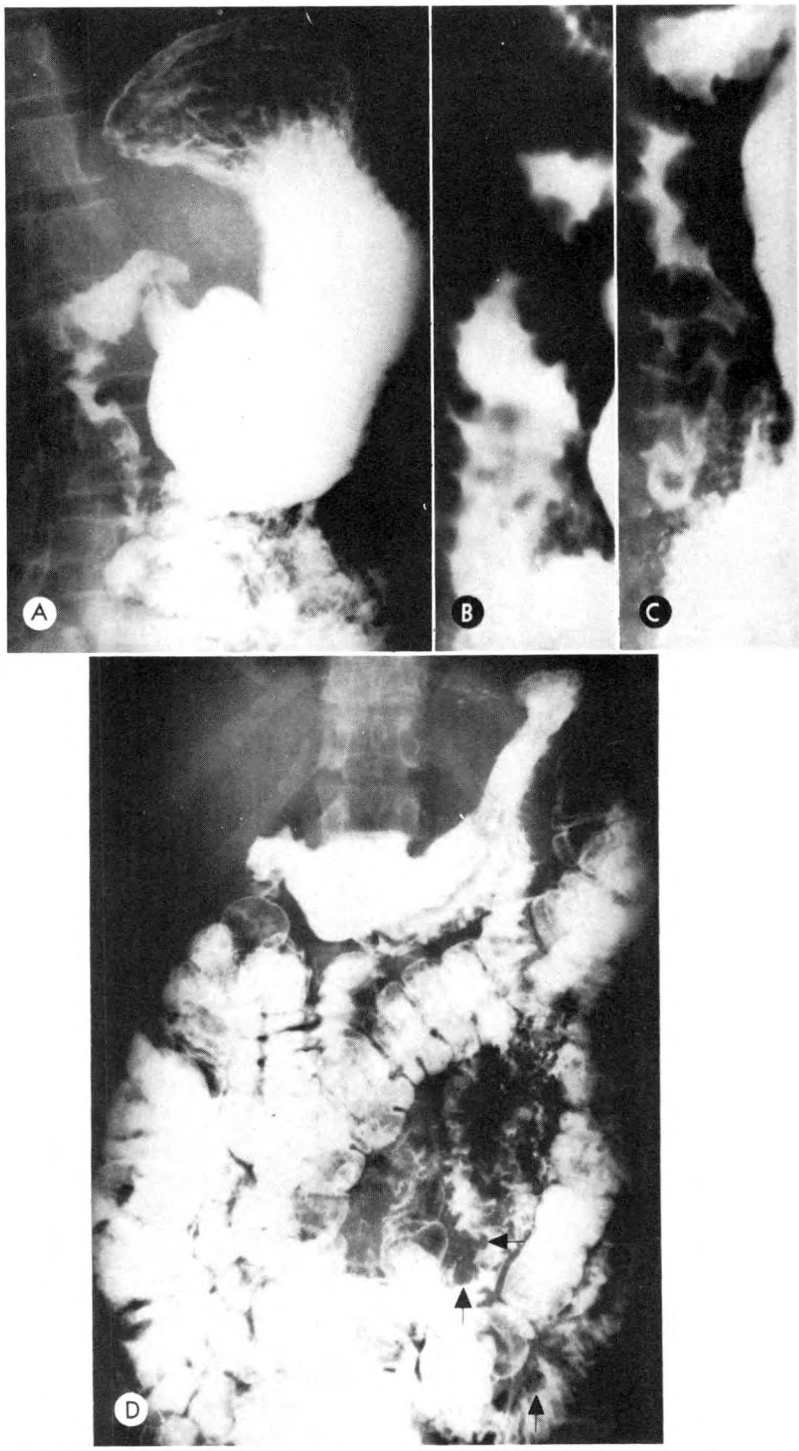


Fig. 4. Case III. (*A*) Right anterior oblique projection and (*B* and *C*) spot roentgenograms of the duodenum show pronounced mucosal thickening involving the first and second portions of the duodenum. (*D*) Solitary nodular defects are identified (arrows) in the mid jejunum.

TABLE I

FEATURES OF COLLAGENOUS SPRUE

Author(s)	Age, Sex	Physical Findings	Chemical Findings	Histologic Findings	Roentgenographic Findings
Schein, 1947[12]	15, M	Vomiting; abdominal pain; diarrhea; 18 pound weight loss	Impaired absorption of vitamin A; hypoproteinemia; hypoprothrombinemia; hypocalcemia; abnormal liver function studies	Diffuse "subtotal villous atrophy" of small bowel with broad-based, bulbous, and plump villi; hyaline deposit at apices of villi and beneath basement membrane	Puddling of barium in small bowel; moderate distention of small bowel
	71, M	Diarrhea compatible with sprue	Not reported	Villous clubbing, broad hyaline bands at tips of villi	Not reported
Hourihane, 1963[8]	Not reported	Idiopathic steatorrhea	Not reported	Small bowel abnormal from pylorus to ileocecal valve with "subtotal villous atrophy;" hyaline collagen completely replacing villi and, in some areas, crypts	Not reported
Neale, 1968[4]	50, M	Weight loss; vague abdominal pain; diarrhea	Initially, megaloblastic anemia, free gastric acid steatorrhea, response to gluten-free diet for 10 years; then, rapid deterioration; no response to gluten-free diet, milk-free diet, prednisone, or protein diet	Flat mucosa; thick layer of collagen beneath epithelium	Proximal and mid jejunum markedly and uniformly reduced in caliber, with little or no mucosal pattern; distal small bowel somewhat dilated; tubular and featureless jejunum
Barry *et al.*, 1970[1]	53, F	Weight loss; frequent loose bowel action for 2½ years	Hypocalcemia; hypoproteinemia; abnormal Schilling test; increased alkaline phosphatase steatorrhea	"Subtotal villous atrophy" pylorus to within 3 feet of ileocecal valve; well-defined band of collagen present beneath the epithelial layer in duodenum and jejunum	Marked flocculation and dilatation of jejunal loops
Weinstein *et al.*, 1970[14]	51, F	Abdominal cramps; watery stools; weight loss	Impaired absorption of vitamin A (carotene); abnormal Schilling test; hypopotassemia; hypoproteinemia; hypocalcemia; hypomagnesemia	Increasing deposits of hyaline collagenous material in the lamina propria, immediately adjacent to the surface epithelium; most of the jejunal and ileal areas devoid of villi; several areas in mid and distal small intestine were normal	Small bowel dilatation; over-all shortening of the small bowel
Pepper *et al.*, 1973	78, M	Watery, greasy, foul-smelling diarrhea	Hypocalcemia; hypomagnesemia; decreased serum carotene; hypoproteinemia	Broadened and blunted villi with increased collagen within the lamina propria	Thickened folds in duodenal region
	39, M	Frequent episodes of diarrhea; crampy abdominal pain	Record not available	Flat mucosa and spectacular deposit of collagen within the lamina propria	Smooth, featureless proximal small bowel, involving entire duodenum and several feet of jejunum
	34, M	Guaiac-positive stools; diarrhea	No chemical evidence of malabsorption	Flat mucosa with marked increase of collagen in lamina propria	Duodenal mucosal thickening; scattered nodular defects in jejunum

genographic feature, in our cases, was pronounced mucosal thickening.

HISTOLOGIC APPEARANCE

The normal jejunum (Fig. 5*A*) has tall, finger-like villi projecting from its mucosa.[10] Tall columnar epithelial cells, containing a thin brush-border, cover the villi on the luminal side. The lamina propria of normal villi contains a variable number of lymphocytes, plasma cells, and eosinophils. Collagen is inconspicuous within the lamina propria.

Paulley[11] was first to describe the histologic appearance of "idiopathic steatorrhea," and shortly thereafter identical changes were observed in celiac disease. Most authorities now agree that a flat intestinal mucosa is not specific for celiac sprue.[3,5,9,12] Diagnosis of celiac sprue can be established, however, by the presence of a histologically flat mucosa on jejunal biopsy and response to a gluten-free diet.[14] In addition, increased numbers of lymphocytes, plasma cells, and eosinophils are present within the lamina propria.[9] Trichrome staining reveals a slight, patchy increase in collagen, which is limited to the subepithelial layer (Fig. 5*B*). Surface mucosal cells are cuboidal, rather than columnar, and have disordered basal nuclei. Intestinal crypts have an increase both in depth and in the number of mitotic figures.

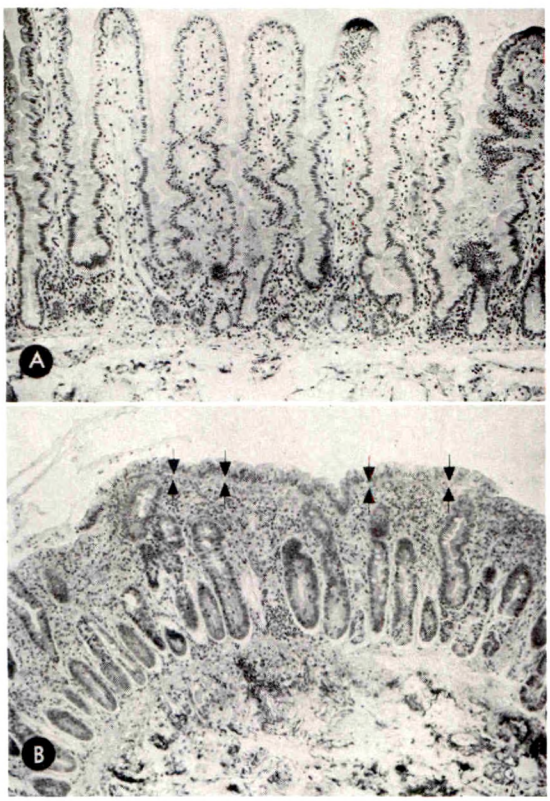


Fig. 5. (*A*) Normal jejunal mucosa. Branching intestinal villi project from the mucosa. Tall columnar epithelial cells cover the villi. The lamina propria contains a variable number of lymphocytes, plasma cells, and eosinophils. (Trichrome, ×100.) (*B*) Jejunal mucosa as seen with celiac sprue. The mucosa is flat. Increased numbers of lymphocytes, plasma cells, and eosinophils are present within the lamina propria. The collagenous band (arrows) is thin and limited to the subepithelial layer. (Trichrome, ×100.)

Collagenous sprue also has a flat mucosa histologically, but an excessive amount of collagen is present within the lamina propria. The collagen can best be seen by means of trichrome staining, where it has a distinctive, green appearance. This collagen may extend throughout the lamina propria to the muscularis mucosae and replace the normal, round cell population and even the crypts.

In Cases I and II, serial biopsy specimens of the jejunum showed increasing amounts of collagen deposit within the lamina propria. In Case I, excess collagen was not initially seen on the biopsy specimen because trichrome stains were not done. The subsequent specimens showed increasing amounts of collagen permeating the lamina propria to the muscularis mucosae. Weinstein *et al.*[14] reported similar findings in their case, in which initial biopsy disclosed normal collagen content in the lamina propria.

It is clear from the description above that the presence of excessive amounts of collagen within the lamina propria is the sole histologic factor distinguishing collagenous sprue from celiac sprue. Adequate biopsy material and proper staining techniques are, therefore, essential in establishing the diagnosis of collagenous sprue.

CLINICAL EVALUATION

The previously reported patients with collagenous sprue had refractory steatorrhea and malabsorption and ultimately died. Despite the most carefully planned gluten-free diet, symptoms persisted. In our first patient (Case I), a gluten-free diet and milk-free diet produced remission of symptoms. Our second patient (Case II) showed a prompt response to corticosteroid therapy, despite lack of response to withdrawal of gluten. In Case III, symptoms of steatorrhea were minimal and the patient has responded to withdrawal of gluten from his diet. The lack of chemical evidence of malabsorption is probably because the intestinal areas of involvement were limited.

SUMMARY

The cause of collagenous sprue is, as yet, undetermined. A patient with clinical malabsorption, histologically flat mucosa, and little or no response to gluten withdrawal is a prime suspect.

Jejunal biopsy specimens show flat mucosa with an abundant deposit of collagen in the lamina propria. Serial and multiple biopsy specimens may be required to make the diagnosis.[14] Trichrome staining is essential.

The roentgenograms demonstrate moderate to severe changes in the proximal small bowel differing somewhat from the characteristic appearance of celiac sprue. Duodenal involvement was present in each of our 3 cases and varied in appearance from mucosal thickening of the proximal small bowel to a virtually smooth, featureless, rigid pattern.

These features in patients with primary intestinal malabsorption that is partially or totally refractory upon strict exclusion of gluten from the diet should alert the radiologist to the possible presence of collagenous sprue. Differentiation of collagenous sprue from celiac sprue allows for more accurate prognosis and more definitive treatment for the patient.

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REFERENCES

1. Barry, R. E., Morris, J. S., and Read, A. E. A. Case of small-intestinal mucosal atrophy. *Gut*, 1970, *11*, 743–747.
2. Barry, R. E., Morris, J. S., and Read, A. E. Collagenous sprue. *New England J. Med.*, 1971, *284*, 1041.
3. Brandborg, L. L., Goldberg, S. B., and Breidenbach, W. C. Human coccidiosis; possible cause of malabsorption: life cycle in small-bowel mucosal biopsies as diagnostic feature. *New England J. Med.*, 1970, *283*, 1306–1313.
4. Clinicopathological Conference. Case of adult coeliac disease resistant to treatment: demstrated at Royal Postgraduate Medical School. *Brit. M. J.*, 1968, *2*, 678–684.
5. Cooke, W. T., Fone, D. J., Cox, E. V., Meynell, M. J., and Gaddie, R. Adult coeliac disease. *Gut*, 1963, *4*, 279–291.
6. Hourihane, D. O'B. Histology of intestinal biopsies. *Proc. Roy. Soc. Med.*, 1963, *56*, 1073–1077.
7. Marshak, R. H., and Eliasoph, J. Roentgen findings in malabsorption syndrome. *M. Clin. North America*, 1957, *41*, 1565–1574.
8. Marshak, R. H., and Lindner, A. E. Radiology of the Small Intestine. W. B. Saunders Company, Philadelphia, 1970.
9. McPherson, J. R. Jejunal biopsy. *M. Clin. North America*, 1970, *54*, 851–862.
10. Nicolette, C. C., and Tully, T. E. Duodenum in celiac sprue. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 248–254.
11. Paulley, J. W. Observations on aetiology of idiopathic steatorrhoea: jejunal and lymph-node biopsies. *Brit. M. J.*, 1954, *4*, 1318–1321.
12. Rubin, C. E., Eidelman, S., and Weinstein, W. M. Sprue by any other name. *Gastroenterology*, 1970, *58*, 409–413.
13. Schein, J. Syndrome of non tropical sprue with hitherto undescribed lesions of intestine. *Gastroenterology*, 1947, *8*, 438–460.
14. Weinstein, W. M., Saunders, D. R., Tytgat, G. N., and Rubin, C. E. Collagenous sprue: unrecognized type of malabsorption. *New England J. Med.*, 1970, *283*, 1297–1301.

VOL. 121, NO. 2

A ROENTGENOGRAPHIC CLASSIFICATION OF DIFFUSE DISEASES OF THE SMALL INTESTINE PRESENTING WITH MALABSORPTION*

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IN spite of classic publications by Golden,[9] Marshak[18] and many others, a simplified, cohesive organization of the diverse entities responsible for malabsorption is needed. We shall present a roentgenographic classification based on small intestine diameter and mucosal fold thickness.

This study is not intended as a review article nor as a complete description of the entities included. This presentation is intended as a preliminary one which is to be amplified not only in regard to correlating further clinical studies, but also to apply this to data processing methods for future computer analysis.

BACKGROUND

Since the first reports of a celiac or belly affliction of children described by Gee in 1888 as "infantile diarrhea of subnutrition," the medical literature has grown voluminous with descriptions of a wide variety of uncommon entities which present as a malabsorption syndrome. Only the diffuse lesions of the small intestine will be categorized in this study.

Establishing a definitive diagnosis in patients with a malabsorption syndrome is exceedingly difficult. This problem has prompted the development of numerous laboratory tests and clinical procedures. The great variety of tests available attests to their lack of specificity. During the past 20 years, the development of peroral biopsy techniques,[26] the use of colloidal barium in the small intestine roentgen examination, and fiberoptic endoscopy have permitted quite accurate gross and microscopic descriptions of the malabsorption spectrum of diseases.

The proposed classification is based on the combined information obtained from the 2 supplemental modalities of gross anatomic small intestine roentgen studies and histologic peroral biopsies.

Thus, a four-part classification to categorize diffuse small intestine diseases, based on the presence or absence of dilatation as well as thickness and regularity of the mucosal fold pattern, as identified on the roentgen examination, is presented. These findings will be correlated with the small intestine biopsy information.

RATIONALE

The advent of colloidal barium suspensions has essentially unmasked true morphologic changes of the bowel wall and mucosal fold patterns that for decades were hidden by U.S.P. barium preparations. Findings, such as segmentation, flocculation, fragmentation, and moulage formation which were originally thought to be characteristic of the malabsorption state can be produced by small amounts of intestinal secretions when U.S.P. barium is used.

The development of micropulverized flocculation-resistant preparations of barium in the 1950s permitted investigators for the first time to study true changes in the caliber of the plicae circulares and a diameter of the small intestine.[16]

The proposed classification groups all of the various diffuse lesions of the small bowel into 4 easily-recognized patterns as seen on barium examinations of the small intestine. The first pattern (Table 1) is that of normal small bowel diameter and normal-appearing mucosal folds. The second

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TABLE I

Pattern I. *Normal Small Intestine Pattern*
1. Pancreatic Insufficiency
2. Congenital Disaccharidase Deficiency
3. Lymphoma, Pseudolymphoma and Variants

Pattern II. *Dilated Small Intestine with Normal Folds*
1. Obstruction and Ileus
2. Chemical or Surgical Vagotomy
3. Diabetes and Hypokalemia
4. Sprue
5. Lymphoma
6. Scleroderma, Dermatomyositis
7. Chronic Idiopathic Intestinal Pseudo-obstruction
8. Systemic Lupus Erythematosus

Pattern III. *Thickened Regular Folds of Small Intestine with or without Dilatation*
1. Edema
2. Intestinal Lymphangiectasia
3. Amyloid
4. Lymphoma, Pseudolymphoma and Variants
5. Bleeding

Pattern IV. *Thickened Irregular or Nodular Folds with or without Dilatation*
1. Granulomatous Enterocolitis, including Tuberculosis and Histoplasmosis
2. Lymphoma, Pseudolymphoma and Variants
3. Amyloid
4. Dysgammaglobulinemias and Benign Lymphoid Hyperplasia
5. Whipple's Disease
6. Systemic Mastocytosis
7. Eosinophilic Gastroenteritis
8. Fibrocystic Disease of the Pancreas
9. Degos' Disease
10. Parasitic Infestation
11. A-beta-lipoproteinemia and A-alpha-lipoproteinemia
12. Waldenström's Macroglobulinemia
13. Zollinger-Ellison Syndrome
14. Pancreatic Cholera

pattern is a dilated small bowel with normal folds. The third pattern is that of thickened regular folds with or without dilatation, and the fourth pattern is that of irregular or nodular folds with or without dilatation.

Thickening of the mucosal folds may be produced by a number of insults to the bowel: 1. submucosal edema; 2. diffuse infiltration of the submucosa and mucosa with cells, blood or protein; 3. nodular accumulation of cells and dilated lymphatics; 4. viscous intraluminal secretions blanketing the normal mucosa, causing an apparent increase in the mucosal fold thickness; and 5. transitory thickening of the mucosal folds with no organic basis postulated to be of psychogenic origin.[7]

Dilatation may be due to functional or organic abnormality of the myenteric plexus. The recent correlation of the vasculitis in systemic lupus erythematosus which involves the muscular layers of the small intestine contributes another etiology for dilatation.[24]

Each pattern will be described and pertinent factors which permit definitive diagnosis within the category will be discussed.

DISCUSSION

PATTERN I. NORMAL ROENTGEN PATTERN OF SMALL INTESTINE

(Fig. 1 and 2)

The differential diagnosis in this category is short even though it includes the greatest proportion of patients with malabsorption.

In pancreatic insufficiency of any etiology, the hallmark in the diagnosis is a normal small bowel roentgen examination and normal small bowel biopsy. When confronted with this paradox in a patient with documented malabsorption, in the absence of hepatic disease, the prime consideration should be pancreatic insufficiency.[18]

In disaccharidase deficiency, small bowel roentgen examination and biopsy are normal; however, the addition of a challenging load of lactose to the barium meal will cause a marked increase in transit and intraluminal fluid.[13]

Disseminated lymphoma may present as an acute malabsorption syndrome. The entity sometimes called Mediterranean lymphoma found in Arabs, Sephardic Jews and other Mediterranean peoples should be included for completeness.[6] In addition, the so-called pseudolymphoma of IgA deficiency may present a similar picture. The roentgen pattern in all of these entities can vary from normal to thickened folds to nodular tumefaction. Lymphoma and its variants therefore appear in all 4 categories in the proposed classification. Biopsy is

diagnostic, showing diffuse lymphomatous infiltration of the lamina propria and submucosa.

PATTERN II. DILATED SMALL INTESTINE WITH NORMAL MUCOSAL PATTERN

Obviously the radiologist should first eliminate obstruction and ileus, but neither of these conditions present with malabsorption and need not be considered further. Chemical or surgical vagotomy, however, may present with malabsorption. A clinical history of gastric surgery or the use of anticholinergic drugs is easily obtained. Small bowel biopsy is normal in these entities.

Diabetes mellitus may on occasion be complicated by a visceral neuropathy and severe intractable diarrhea. In some cases the small bowel roentgen examination is normal; however, when complicated by hypokalemia, dilatation of the small bowel may occur. Small bowel biopsy is generally normal in diabetes but degeneration of the myenteric plexus may be demonstrated in advanced cases.[28]

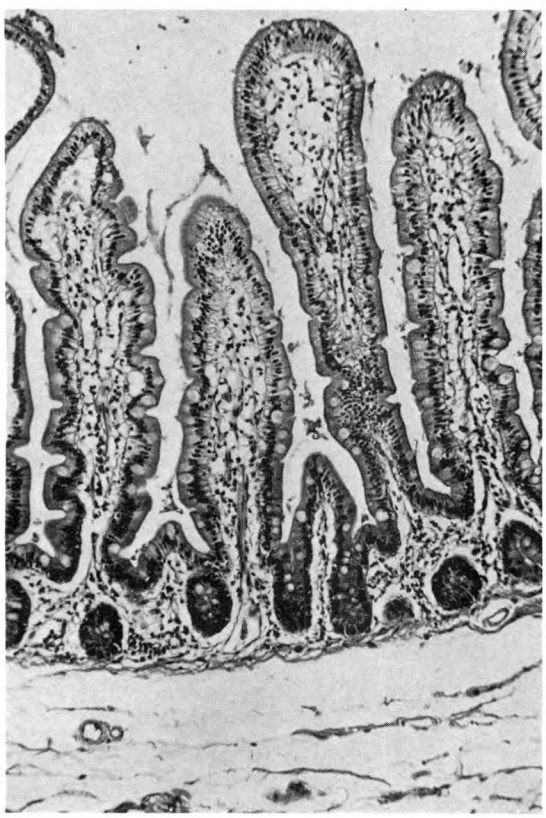


Fig. 1. A normal jejunal biopsy specimen shows tall delicate villi which are covered with columnar epithelium and surrounded at their bases by intestinal glands. The lamina propria consists mainly of connective tissue, fibroblasts and smooth muscle cells. (H. & E. 100×.)

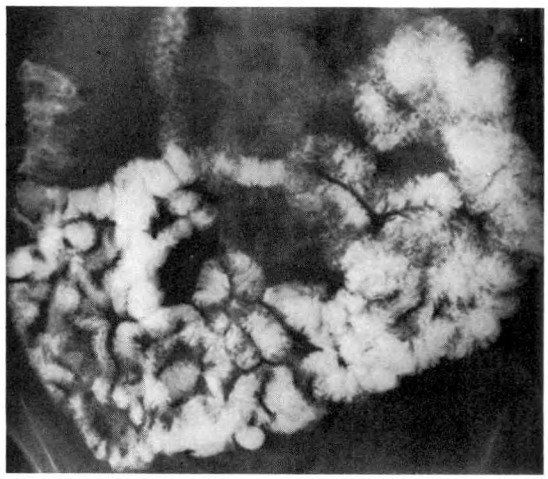


Fig. 2. A normal proximal small intestine has a diameter of approximately 2.5 cm. with mucosal folds less than 2 mm. in thickness. The distal small bowel normally has a smaller caliber with a decrease in the number of mucosal folds as compared to the proximal portion.

Sprue comprises a group of closely related diseases of the small intestine: tropical sprue, celiac sprue, collagenous sprue and dermatitis herpetiformis. The characteristic roentgenographic findings in all of these entities are thickened irregular folds in the duodenum in conjunction with a dilated small bowel with normal thin folds and episodes of transient intussusception (Fig. 3*A*). Biopsy shows villous atrophy and submucosal plasma cell infiltration (Fig. 3*B*).[20]

Diffuse intestinal lymphoma may complicate longstanding sprue. Here, the clinical course shows a sudden refractoriness to treatment and biopsy shows lymphomatous infiltrate in the submucosa. Roentgenographically, it may be indistinguishable from celiac sprue.[6]

In scleroderma and dermatomyositis, malabsorption is secondary to stasis and bacterial overgrowth. Prolonged transit,

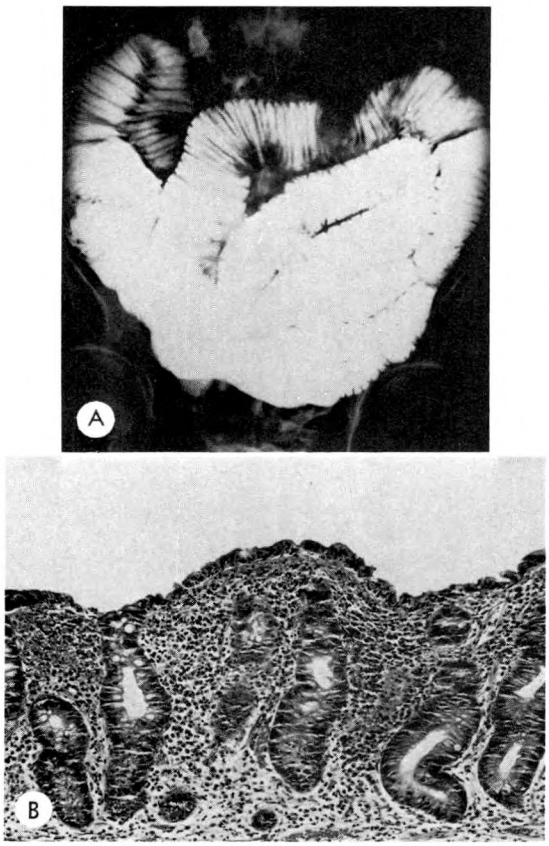


FIG. 3. (*A*) The distal small intestine in celiac sprue is dilated with normal thin folds. (*B*) Small intestinal biopsy in celiac sprue shows complete villous atrophy, deepened intestinal glands, cuboidal-shaped epithelial cells and plasma cell infiltration of the lamina propria. (H. & E. 100×.).

megaduodenum and pseudodiverticula formation are frequent roentgen findings. Biopsy shows normal villi but collagenous replacement of the muscularis and varying degrees of vasculitis.[11]

Chronic idiopathic intestinal pseudo-obstruction may be difficult to differentiate from scleroderma, but here pseudodiverticula formation is not found. Small bowel biopsy is histologically normal.[15] A vasculitis of the muscularis in systemic lupus erythematosus can apparently also result in a pseudo-obstruction picture.[24]

PATTERN III. THICKENED REGULAR FOLDS WITH OR WITHOUT DILATATION

The differential diagnosis in this category is relatively short, and by far the most common entity in this category is intestinal edema of any origin. Hypoproteinemia of any etiology may present with intestinal edema if the serum albumin level is below 1.5 grams per cent (Fig. 4). The so-called "protein-losing enteropathy" is not a single disease entity, but has been demonstrated in a variety of gastrointestinal disorders many of which are the subject of this paper.[17]

Intestinal lymphangiectasia for example is characterized by a protein loss with secondary intestinal edema. Roentgenographically, a uniform thickening of the mucosal folds is generally present with nodular defects uncommon. Biopsy will show swollen bulbous villi with dilatation of the submucosal lymphatics in addition to abundant PAS-negative macrophages.[21] Lymphangiography can be diagnostic, showing dilated lymphatics and absent abdominal lymph nodes.

Protein loss in the intestinal tract has also been described in such unrelated conditions as aganglionosis, congestive heart

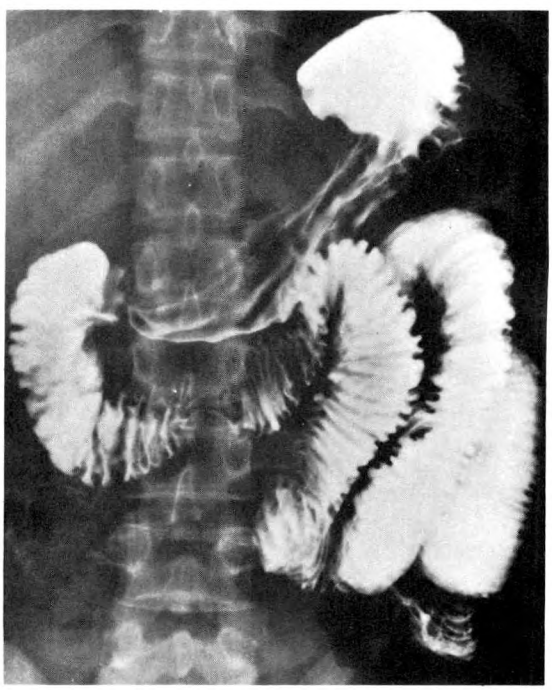


FIG. 4. The proximal small bowel in a patient with a serum albumin of 1.0 gm. per cent shows thickened regular folds.

failure, constrictive pericarditis, exudative skin conditions, familial Mediterranean fever, burns and allergy. Hypoproteinemia may also result from nephrosis or cirrhosis. Small intestinal biopsy shows a marked cell-free accumulation of fluid in the submucosa with normal villi architecture.[19]

In amyloidosis there may be diffuse immunoglobulin deposition in the small bowel with subsequent diarrhea and malabsorption.[8] Roentgenographically there is a thickening of the mucosal folds which may take on either a regular or nodular appearance, depending on the distribution of the proteinaceous deposition.[14] Biopsy shows amyloid deposits around submucosal blood vessels, in the muscularis and occasionally in the tips of the villi.

The lymphoma, pseudolymphoma and related immunoglobulin deficiency disorders may rarely present with diffuse cellular infiltration causing thickened regular folds. Confirmation will require biopsy and laboratory studies.

Intramural bleeding may cause thickened regular folds, but is not associated with malabsorption.

PATTERN IV. THICKENED IRREGULAR OR NODULAR FOLDS WITH OR WITHOUT DILATATION

Regional enteritis or transmural granulomatous enterocolitis not uncommonly presents with malabsorption. It is not the purpose of this paper to discuss this well-known entity other than to include it as a cause of malabsorption. Thickened irregular folds have been described in virtually every portion of the gastrointestinal tract. Ulceration, skip areas, mass effect, abscesses and fistulae, however, help to differentiate this entity from many others in this pattern. Small intestinal biopsy will show mucosal ulceration with submucosal inflammatory changes and granuloma formation. Tuberculosis and histoplasmosis are many times indistinguishable from transmural disease.[18]

Lymphoma again appears in this picture. Biopsy and clinical patterns, however, should be diagnostic.

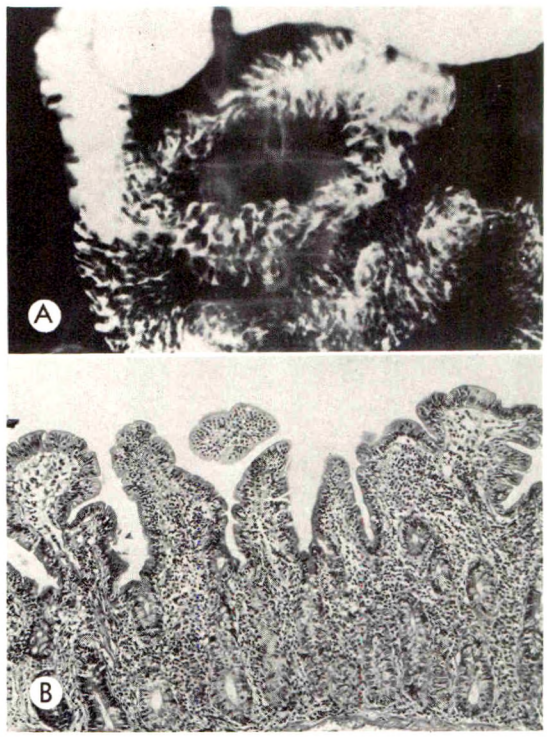


Fig. 5. (*A*) Dysgammaglobulinemia Type II shows thickened nodular folds in the proximal small intestine. The bowel caliber is normal. (*B*) Histologically, in dysgammaglobulinemia, there is partial villous atrophy and intense lymphocytic infiltration of the lamina propria and submucosa. The absence of plasma cells is characteristic. (H. & E. 100×.)

In dysgammaglobulinemia[10] and related disorders of benign lymphoid hyperplasia, nodular accumulations of lymphocytes cause a thickened nodular fold pattern (Fig. 5*A*).[27] Biopsy shows partial villous atrophy and an absence of plasma cells in the submucosa. Numerous small lymphocytes and hypertrophied lymph follicles are also characteristic (Fig. 5*B*).[26] Serum protein immunoelectrophoresis will show characteristic immunoglobulin patterns in these disorders.[27]

Whipple's disease is an uncommon cause of malabsorption and arthritis. Thickened irregular folds are more commonly found in the jejunum in this disorder.[22] Small bowel biopsy is diagnostic with swollen bulbous villi, and abundant macrophages with PAS- and gram-positive granules in the

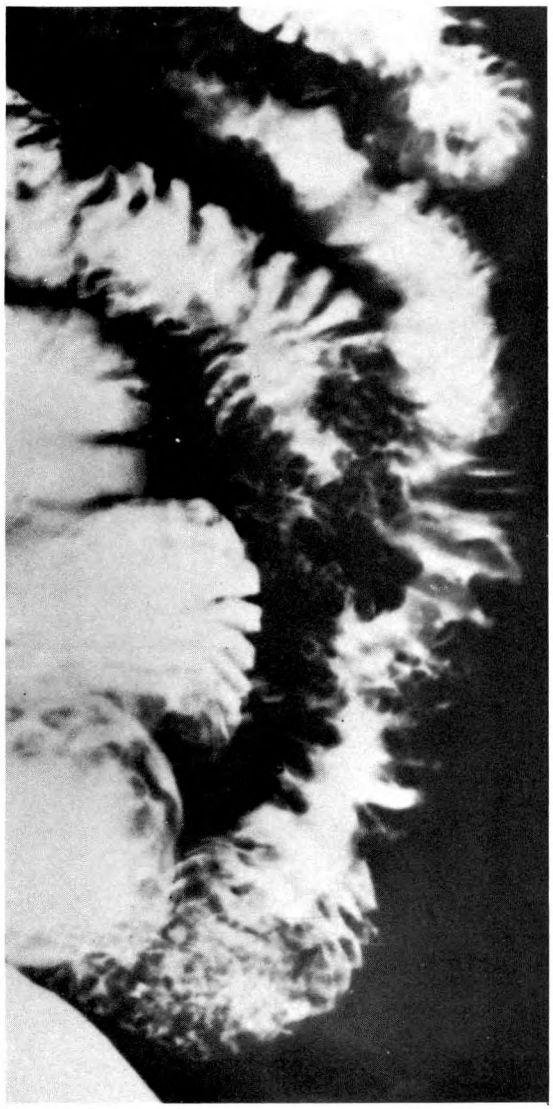


FIG. 6. In eosinophilic gastroenteritis, thickened irregular folds are confined for the most part to the proximal small intestine.

submucosa. Tiny gram-positive rod-shaped bacilli are always present in the mucosa of active cases.[26]

How frequently systemic mastocytosis affects the gastrointestinal tract and causes malabsorption is subject to serious question. Thickened nodular folds have been reported in some cases. Biopsy is reported to show nodular mast cell infiltrate of the submucosa.[5]

Eosinophilic gastroenteritis may also present with thickened nodular folds mainly in the proximal small intestine and antrum of the stomach (Fig. 6). Biopsy shows eosinophilic infiltration of various levels of the bowel wall.[2]

In fibrocystic disease, thickened folds are confined to the proximal small intestine. These are found in both symptomatic and asymptomatic individuals, and they do not necessarily correlate with the malabsorption state. Biopsy will show normal villi but an increased number of goblet cells, dilatation of the mucosal glands, gaping gland crypts and occasionally inspissated mucus in the bases of the crypts.[1]

Degos' disease or malignant atrophic papulosis on occasion presents with malabsorption. Thickened regular folds are found in the proximal small bowel secondary to fibrotic plaques in the submucosa. Biopsy shows partial villous atrophy, submucosal fibrosis and occlusive fibrosis of small arterioles.[25]

Among the parasites which may infest the human, only a few significantly alter the mucosal fold pattern of the small intestine and present with malabsorption. They are giardiasis, strongyloides and hookworm. The frequent association of giardiasis with the dysgammaglobulinemias is well documented.[27] Each causes thickened regular folds and increased intestinal secretion. Biopsy of the small intestine may demonstrate the organisms, but they are more commonly recovered from the stool.[18]

The Bassen-Körnzweig syndrome or a-beta-lipoproteinemia is a neuromuscular disorder presenting with ataxia, retinitis and steatorrhea.[23] Biopsy is characteristic with normal villi, but the mucosal cells are swollen with striking vacuolation.[26] The much rarer a-alpha-lipoproteinemia can cause this same clinical picture.

In Waldenström's macroglobulinemia, a small number of patients may present with malabsorption. Roentgenographically, the mucosal folds are thickened and nodular. Pathologically, there is edema of the mucosa and lymphangiectasia. The lymphatics are filled with a proteinaceous eosinophilic material. Large eosinophilic macrophages

and lymphocytoid plasma cell infiltration are also found. Amyloid deposits have also been described.[12]

The Zollinger-Ellison syndrome is secondary to a non-beta islet cell tumor of the pancreas. Fulminating ulcer diathesis is secondary to increased gastrin secretion by this tumor. Increased gastrin levels are best diagnosed by radioimmunoassay. Clinically, diarrhea may also supervene. Roentgenographically, in addition to the ulcers, thickened irregular folds are frequently seen in the stomach, duodenum and proximal small intestine. Small bowel biopsy is normal.[3]

Pancreatic cholera is secondary to yet another non-beta islet cell tumor of the pancreas which secretes secretin or a secretin-like substance, resulting in profound diarrhea, hypokalemia, protein loss and achlorhydria. Secretin may be demonstrated by bioassay.[4] Small bowel biopsy is normal in spite of the finding of dilatation and edematous folds.

SUMMARY

The diseases diffusely afflicting the small intestine and presenting with malabsorption are numerous and uncommon. The development of several diagnostic modalities in the past 20 years has permitted accurate clinical and pathologic descriptions of many of these entities.

A classification has been put forth based on the gross appearance of the mucosal folds and the presence or absence of intestinal dilatation as determined by small bowel roentgen examination.

It is proposed that correlation with small bowel biopsy, clinical findings and simple laboratory methods usually indicates a definitive diagnosis.

Data processing and computer analysis should be the next step in the application of this classification.

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REFERENCES

1. Berk, R. N., and Lee, F. A. Late gastrointestinal manifestations of cystic fibrosis of pancreas. *Radiology*, 1973, *106*, 377–381.
2. Burhenne, H. J., and Carbone, J. V. Eosinophilic (allergic) gastroenteritis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *96*, 332–338.
3. Christoforidis, A. J., and Nelson, S. W. Radiologic manifestations of ulcerogenic tumors of pancreas. *J.A.M.A.*, 1968, *198*, 511–516.
4. Cleator, I. G. M., Thompson, G. C., Sircus, W., and Coombes, M. Bioassay evidence of abnormal secretin-like and gastrin-like activity in tumor and blood in cases of "choleraic diarrhoea." *Gut*, 1970, *11*, 206–211.
5. Clemett, A. R., Fishbone, G., Levine, R. J., James, A. E., and Janower, M. Gastrointestinal lesions in mastocytosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 405–412.
6. Dutz, W., Asvadi, S., Sadri, S., and Kohout, E. Intestinal lymphoma and sprue: systemic approach. *Gut*, 1971, *12*, 804–810.
7. Friedman, J. Roentgen studies of effects on small intestine from emotional disturbances. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1954, *72*, 367–379.
8. Glenner, G. G., Ein, D., and Terry, W. D. Immunoglobulin origin of amyloid. *Am. J. Med.*, 1972, *52*, 141–147.
9. Golden, R., Cimmino, C. V., Collins, L., Dreyfuss, J. R., and Janower, M. L. The Digestive Tract. Williams & Wilkins Company, Baltimore, 1968.
10. Hodgson, J. R., Hoffman, H. N., II, and Huizenga, K. A. Roentgenologic features of lymphoid hyperplasia of small intestine associated with dysgammaglobulinemia. *Radiology*, 1967, *88*, 883–888.
11. Kahn, I. J., Jeffries, G. H., and Sleisenger, M. H. Malabsorption in scleroderma. *New England J. Med.*, 1966, *274*, 1339–1344.
12. Khilnani, M. T., Keller, R. J., and Cuttner, J. Macroglobulinemia and steatorrhea: roentgen and pathologic findings in intestinal tract. *Radiol. Clin. North America*, 1969, *7*, 43–55.
13. Laws, J. W., and Neale, G. Radiological diagnosis of disaccharidase deficiency. *Lancet*, 1966, *2*, 139–143.
14. Legge, D. A., Carlson, H. C., and Wollaeger, E. E. Roentgenologic appearance of systemic amyloidosis involving gastrointestinal tract. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 406–412.
15. Maldonado, J. E., Gregg, J. A., Green, P. A., and Brown, A. L., Jr. Chronic idiopathic

intestinal pseudo-obstruction. *Am. J. Med.*, 1972, *49*, 203–212.
16. MARSHAK, R. H., and ELIASOPH, J. Roentgen findings in malabsorption syndrome. *M. Clin. North America*, 1957, *41*, 1565–1574.
17. MARSHAK, R. H., KHILNANI, M., ELIASOPH, J., and WOLF, B. S. Intestinal edema. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *101*, 379–387.
18. MARSHAK, R. H., and LINDNER, A. E. Radiology of the Small Intestine. W. B. Saunders Company, Philadelphia, 1970.
19. MARSHAK, R. H., WOLF, B. S., COHEN, N., and JANOWITZ, H. D. Protein-losing disorders of gastrointestinal tract: roentgen features. *Radiology*, 1961, *77*, 893–905.
20. NICOLETTE, C. C., and TULLY, T. E. Duodenum in celiac sprue. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 248–254.
21. NUGET, F. W., ROSS, J. K., and HURXTHAL, L. M. Intestinal lymphangiectasia. *Gastroenterology*, 1964, *47*, 536–539.
22. RICE, R. P., ROUFAIL, W. M., and REEVES, R. J. Roentgen diagnosis of Whipple's disease (intestinal lipodystrophy). *Radiology*, 1967, *88*, 295–301.
23. SALT, H. B., WOLFF, O. H., LLOYD, J. K., FOSBROOKE, A. S., CAMERON, A. H., and HUBBLE, D. V. On having no beta-lipoprotein: syndrome comprising a-beta-lipoproteinaemia, acanthocytosis and steatorrhoea. *Lancet*, 1960, *2*, 325–329.
24. SHAPEERO, L. G., MYERS, A., MILLER, W. T., and OBERKIRCHER, P. B. Lupus vasculitis of small bowel. Presented at R.S.N.A., 1973, Paper Number 8.
25. STROLE, W. E., JR., CLARK, W. H., JR., and ISSELBACHER, K. J. Progressive arterial occlusive disease (Köhlmeier-Degos): frequently fatal cutaneosystemic disorder. *New England J. Med.*, 1967, *276*, 195–201.
26. TRIER, J. S. Diagnostic value of peroral biopsy of proximal small intestine. *New England J. Med.*, 1971, *285*, 1470–1473.
27. VERMESS, M., WALDMANN, T. A., and PEARSON, K. D. Radiographic manifestations of primary acquired hypogammaglobulinemia. *Radiology*, 1973, *107*, 63–69.
28. VINNIK, J. E., KERN, F., JR., and STRUTHERS, J. E., JR. Malabsorption and diarrhea of diabetes mellitus. *Gastroenterology*, 1962, *43*, 507–520.

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THOSE OTHER TYPES OF ENTEROCOLITIS*

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ARTICLES describing a great number of toxic and inflammatory conditions afflicting the gastrointestinal tract have appeared in the medical literature in the past 3 decades. Many of these descriptions have been case reports of unusual clinical conditions which the investigators have labeled as separate and new clinical entities. We shall attempt to correlate related findings in these various uncommon entities and to simplify the classification into 4 categories. The spectra of ulcerative colitis and transmural colitis have been excluded from this study. The rationale for this classification will be discussed and the roentgenographic features of each will be described.

MATERIAL AND METHOD

Several cases of uncommon types of enterocolitis have recently come to our attention. Interest in these cases has prompted a review of the literature and a retrospective analysis of autopsies and roentgenograms of various types of enterocolitis seen at the University of Minnesota and affiliated hospitals since 1954. Those patients with a diagnosis of ulcerative colitis, granulomatous enterocolitis, and specific types of inflammatory colitis, such as tuberculous and amebic, were excluded from this study. Three hundred and fifty-three autopsies with a final diagnosis of enterocolitis of various types were reviewed. One hundred and fifty records were complete and included in the study. Sixty-two of these 150 had their roentgenographic examinations of the abdomen, contrast medium studies of the gastrointestinal tract, and/or angiography during the course of their acute illness and shortly before death.

We classified all the various entities reviewed as follows: (1) pseudomembranous enterocolitis; (2) adult necrotizing enterocolitis; (3) neonatal necrotizing enterocolitis; and (4) nonocclusive ischemic enterocolitis. The results of this retrospective analysis appear in Tables I, II, III and IV.

The clinical and pathologic findings in each of the 4 categories are described in the Discussion. The significant roentgenographic findings are as follows:

Pseudomembranous enterocolitis (Fig. 1, *A–C*) usually presents with thickened mucosal folds (70 per cent) and dilated bowel (50 per cent). Pneumatosis is not infrequent (17 per cent) and toxic megacolon occurred (10 per cent). Normal roentgenograms were encountered in 10 per cent.

The most common roentgen findings in adult necrotizing enterocolitis (Fig. 2, *A–C*) are: intramural air (73 per cent), either submucosal or subserosal; air within the portal venous system (55 per cent); and pneumoperitoneum. Dilatation (73 per cent) and bowel wall edema (45 per cent) were also frequently encountered.

Neonatal necrotizing enterocolitis (Fig. 3, *A* and *B*) usually presents with pneumatosis (100 per cent), dilated bowel (60 per cent), and mucosal edema (70 per cent). Portal venous gas was present in 1 patient.

In nonocclusive ischemic enterocolitis (Fig. 4, *A–C*), the most consistent finding was that of simple dilatation, either local or generalized ileus (80 per cent), and variable amounts of mucosal edema (33 per cent).

Contrast medium examinations of the colon were performed in only 3 of the cases with pseudomembranous enterocolitis. One showed marked edema of the mucosa, another showed shaggy pseudomembrane formation and the third showed changes indistinguishable from acute ulcerative colitis. In this case, a toxic megacolon developed soon after the barium enema examination. The role of the barium enema

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TABLE I

CLINICAL FINDINGS

	Cases	Age Range	Age Average	Heart Disease	Shock or Prolonged Hypotension	Post-operative	Uremia*	Cancer	Leukemia, Lymphoma	Obstruction
Pseudomembranous enterocolitis	29	7 wk.–82 yr.	55	8	6	10	12	7	5‡	3
Adult necrotizing enterocolitis	11	8–78	47	2	0	4	6	4	2	0
Nonocclusive ischemic enterocolitis	18	20–74	55	7	11	5	16†	1	0	0

* Blood urea nitrogen >50 mg. per cent.
† All had blood urea nitrogen tests >100 mg. per cent.
‡ Five children, aged 7 weeks to 13 years, all had either lymphoma or leukemia.

TABLE II

CLINICAL FINDINGS IN NEONATAL NECROTIZING ENTEROCOLITIS
7 Patients

Cases	
Age range	5 days–1 year*
Premature	4
Hyaline membrane	3
Perforation	3
Other congenital anomalies	2†

* Child aged 1 year with agammaglobulinemia.
† One child with Down's syndrome with biliary atresia and one child with congenital narrowing of the sigmoid colon.

examination in precipitating this complication is suggested but not proven.

None of the cases of necrotizing or ischemic enterocolitis had contrast medium studies. This undoubtedly reflects the severity of their clinical condition.

Two patients with ischemic enterocolitis had angiography which demonstrated patent major vessels.

The importance of the plain film roentgenogram in the diagnosis in these conditions is self-evident.

DISCUSSION

The medical literature is replete with confusing descriptions of various specific types of enterocolitis. The majority of these descriptions have appeared in the last 3 decades. The nosology of these entities has also grown exponentially as various investigators have attempted to describe and classify unusual, clinical, and pathologic conditions afflicting the bowel and label them as specific entities. This study will attempt to group all of these various entities into 4 conditions based on clinical and

TABLE III

MICROBIOLOGY

Cultures	Organism					
	Staphylococcus	*Clostridium welchii*	*Escherichia coli*	*Pseudomonas*	*Klebsiella*	*Candida*
Pseudomembranous	6			2		2
Adult necrotizing		6	2	2	1	
Neonatal necrotizing			1	2	1	
Nonocclusive ischemic	6		1			

TABLE IV

ROENTGENOGRAPHIC FINDINGS

	No. of Cases	Dilatation of Involved Segment or Ileus	Thickened Edematous Folds	Combined Dilatation and Edema	Pneumatosis	Portal Venous Air	Toxic Megacolon	Free Air	Normal Roentgenograms	Other
Pseudomembranous enterocolitis	29	15	19	13	5	0	3	1	3	1 Dyskinetic ileus
Adult necrotizing enterocolitis	11	8	5	5	8	6	0	1	0	1 Gastric dilatation
Neonatal necrotizing enterocolitis	7	4	5	5	7	1	0	0	0	1 Ascites
Nonocclusive ischemic enterocolitis	18	15	6	6	0	0	1	0	3	1 Fluid-filled small bowel

autopsy findings. Roentgenographic and pathologic correlation together with a description of the roentgen findings will be offered for each.

(1) Pseudomembranous enterocolitis was first described by Billroth in 1867.[19] The entity has reappeared in the literature under a variety of titles: diphtheritic enterocolitis;[6] acute postoperative enterocolitis;[19] staphylococcal pseudomembranous enterocolitis;[24] and antibiotic enterocolitis.[4] Penner and Bernheim[19] reported the first large series in this country and described the pathologic findings. The intestinal tract is usually dilated and filled with liquid feces. The mucosal surface is generally lined with a friable adherent white or tan pseudomembrane that occurs either in isolated plaques or as an extensive confluent process. When the membrane is stripped off, the underlying mucosa is usually ulcerated, congested, and edematous (Fig. 1*B*).

Microscopically, the membrane consists of necrotic cellular debris, fibrin, mucus, and inflammatory cells. Occasionally, bacteria and fungi are identified[4] (Fig. 1*C*). As the disease progresses, the entire mucosa may undergo coagulative necrosis and at this stage the membrane may slough, leaving an ulcerated pyogenic surface. Thrombi and fibrinoid changes in the submucosal blood vessels have also been described.[8]

The cause of this entity has been long debated. The 3 general categories of etiologic significance are: (1) antibiotics; (2) predisposing debilitating conditions; and (3) staphylococcal infection.

The role of antibiotics is uncertain. Reports of the disease occurred before the advent of antibiotics. Nevertheless, numerous reports in the 1950s and 1960s implicated antibiotics as causal agents.[24] Recently it has been emphasized that the disease may occur in apparently normal persons who are given wide spectrum antibiotics for unrelated causes.[2] We have recently reported a reappearance of this disease in patients placed on clindamycin.[26]

The common denominator in surgery, heart disease, uremia, sepsis, leukemia, lymphoma, carcinoma, and patients on immunosuppressive therapy is debility. Menaker and Van Hagen[16] stressed the role of intestinal obstruction. Shock has also been mentioned,[19] but most observers now feel that the latter is the result of profound diarrhea and toxicity rather than the cause of the syndrome.[4]

Staphylococcal infection has also been frequently implicated.[25] Indeed, the enterotoxin may contribute to the clinical picture of toxicity, but the organism is cultured in only 25–50 per cent of cases and is certainly not the sole factor in the development of the entity. Staphylococcus was cultured in 6 of 29 cases in this study. Thus, pseudo-

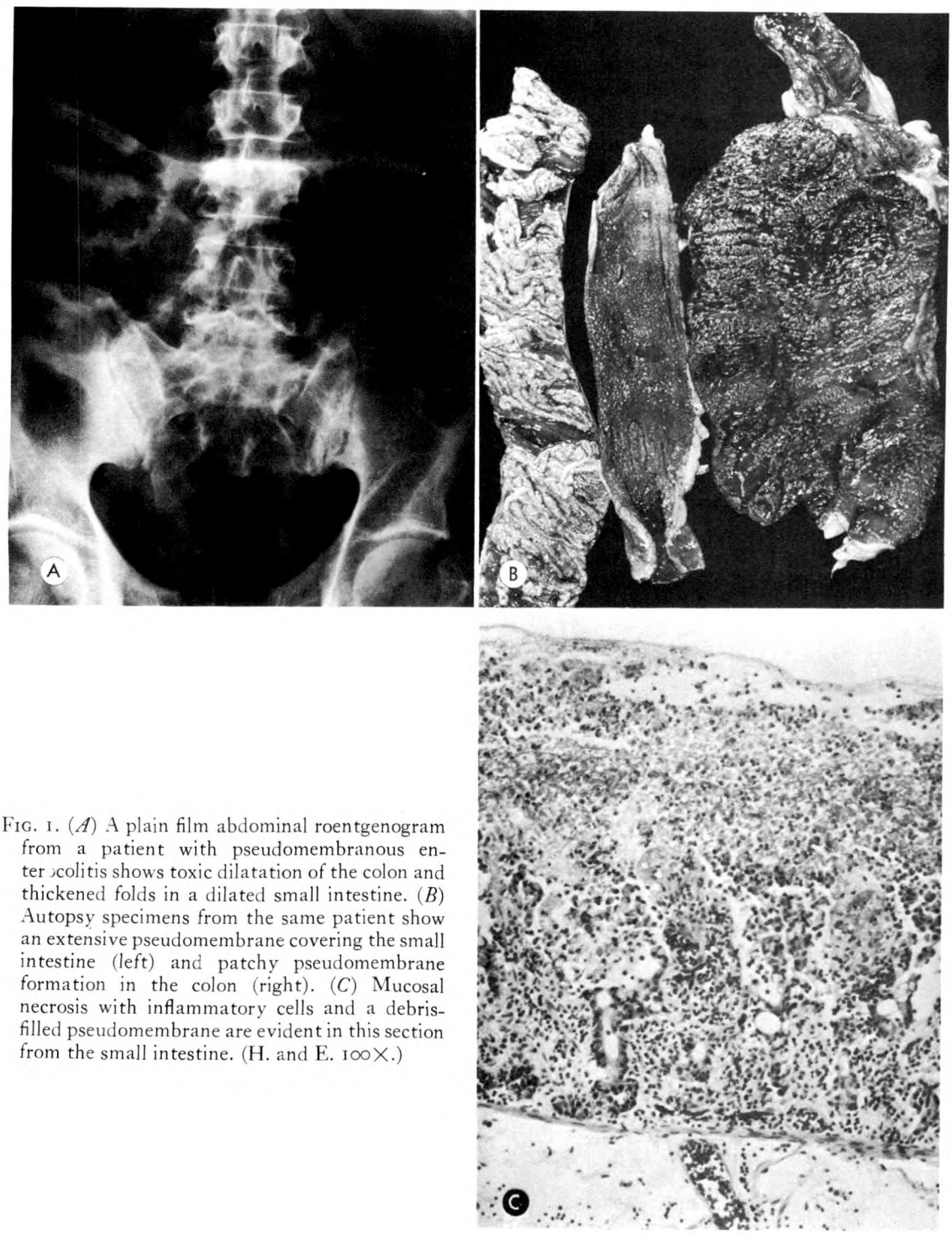


FIG. 1. (*A*) A plain film abdominal roentgenogram from a patient with pseudomembranous enterocolitis shows toxic dilatation of the colon and thickened folds in a dilated small intestine. (*B*) Autopsy specimens from the same patient show an extensive pseudomembrane covering the small intestine (left) and patchy pseudomembrane formation in the colon (right). (*C*) Mucosal necrosis with inflammatory cells and a debris-filled pseudomembrane are evident in this section from the small intestine. (H. and E. 100×.)

membranous enterocolitis is an opportunistic disease that may arise in a variety of clinical situations and no one factor is common to all.

Clinically, patients are profoundly ill with shock, dehydration, and explosive diarrhea.

The roentgenographic findings were first described by one of us[6] and recently we described the newer contrast medium and abdominal scout film roentgen findings in this entity. Similar findings were also reported by Schapiro and Newman.[22] The most consistent roentgenographic finding observed is a dilatation of the small bowel generally associated with edematous and thickened mucosal folds. The thickening of the mucosal folds may be due to the pseudomembrane covering the bowel surface (Fig. 1*A*). A shaggy appearance to the mucosa indicates partial sloughing or intermittent areas of pseudomembrane formation. Toxic megacolon was present in 3 of 29 cases in the present study. Dyskinetic ileus is frequently observed. If contrast agents are used, one can anticipate finding edema and a shaggy appearance of the mucosa and/or ulceration which is identical to inflammatory or ischemic colitis.[22,26] The findings are therefore nonspecific and roentgenographically indistinguishable from many other types of toxic or inflammatory lesions of the bowel.

Treatment is bowel rest, institution of antistaphylococcal antibiotics, pressor agents, and steroids. Despite vigorous therapy, the outcome is frequently fatal.

(2) Necrotizing enterocolitis was first described in 1947 by Jeckeln.[11] He reported a necrotizing enteritis which he called "Darmbrand," literally gut-burn as analogous to heartburn. This was later shown by Zeissler and Rassfeld-Sternberg[29] to be due to a beta toxin of *Clostridium welchii* Type F. A similar disorder was described by Murrell and Roth[18] as afflicting the jejunum in natives of New Guinea and is due to pork infected with *Clostridium welchii* Type C. This entity is referred to as pig-bel. Other terms used to describe this entity are enteritis necroticans, necrotizing colitis, necrotizing jejunitis and gangrenous enterocolitis. As one can see by these terms, various segments of the gastrointestinal tract can be involved in the process.

Pathologically, the affected bowel is demonstrated by varying degrees of ulceration and foci of patchy green-brown necrosis along its length (Fig. 2*B*). The extent of the disease is usually greater on the mucosal surface than on the serosal surface. Intramural gas bubbles may be identified grossly and perforations of the bowel are relatively frequent. The major mesenteric blood vessels supplying the afflicted portions of the bowel are patent, thereby differentiating the entity from gangrene secondary to major vessel occlusion.[14] Portal vein gas may also be identified on gross examination.

Microscopically, there is a coagulative necrosis of the mucosa and submucosa. Leukocyte infiltration is seen at the junction of the necrotic and non-necrotic regions (Fig. 2*C*). Gram positive bacilli may be identified in the necrotic tissue and gas bubbles may be identified in the submucosa. Necrosis with perforation and peritonitis may also develop.

The etiology is usually *Clostridium welchii*, *Escherichia coli* or *Klebsiella aerobacter* organisms causing gangrenous bowel in patients with predisposing debilitating conditions such as severe heart disease or prolonged hypotension. Others have emphasized the role of bowel distention and obstructing neoplasms.[14]

Clinically, necrotizing enterocolitis of the adult presents with an onset of severe abdominal pain that suggests an intra-abdominal emergency requiring immediate surgery.

Roentgenographic findings were described by Rigler and Pogue[21] and consist of intramural gas, varying degrees of dilatation, and mucosal edema. The intramural gas, submucosal and serosal, is by far the most consistent finding and is frequently complicated by gas within the confines of the portal vein. The etiology of the portal

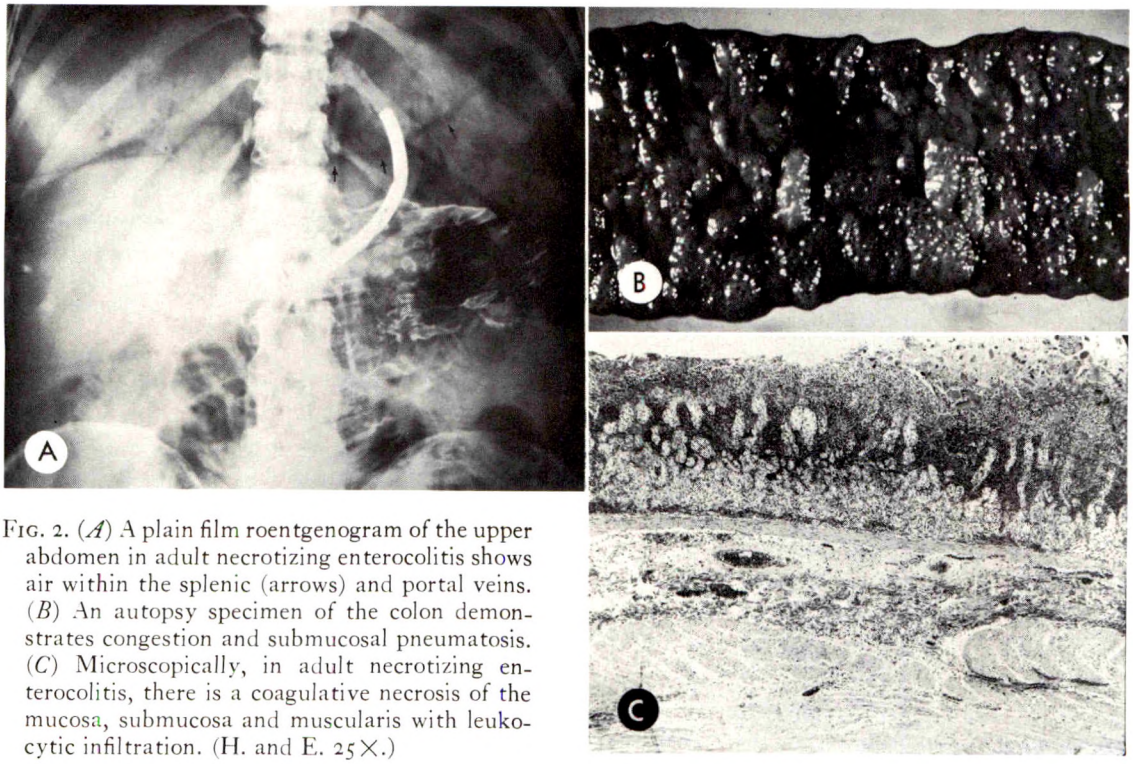


FIG. 2. (*A*) A plain film roentgenogram of the upper abdomen in adult necrotizing enterocolitis shows air within the splenic (arrows) and portal veins. (*B*) An autopsy specimen of the colon demonstrates congestion and submucosal pneumatosis. (*C*) Microscopically, in adult necrotizing enterocolitis, there is a coagulative necrosis of the mucosa, submucosa and muscularis with leukocytic infiltration. (H. and E. 25×.)

venous gas is unknown but may represent the product of gas forming organisms or mucosal necrosis with ulceration and escape of intraluminal gas into the exposed submucosal blood vessels. Roentgenographically the entity is impossible to distinguish from mesenteric arterial or venous thrombosis which may also cause intramural air and portal venous air (Fig. 2*A*).

Treatment consists of antibiotics, bowel rest, and resection of the involved segment of bowel when possible. Nearly all cases in the adult are fatal.

(3) Neonatal necrotizing enterocolitis was first convincingly characterized by Berdon and co-workers in 1964.[3] The entity is increasingly recognized as a specific disease. Pathologically, any segment of the bowel may be involved with varying degrees of ulceration and necrosis but primarily affected are the ileum, jejunum, cecum, and ascending colon. Perforations are common, and intramural air is frequently seen on gross examination.[9]

Microscopically, there is a sloughing of the surface epithelium, frequently accompanied by submucosal hemorrhages. The denuded areas may be covered by a delicate fibrinous exudate containing cellular debris and scanty inflammatory cells. Gas bubbles are often present in the submucosa (Fig. 3*B*). Fibrin thrombi are found in the submucosal and superficial muscularis vessels with occasional perivascular hemorrhages.[9] Peritonitis is a fatal complication.

Clinically, patients present from 2 to 7 days of life with dyspnea, abdominal distention, and occasional gastrointestinal bleeding and vomiting. Positive blood cultures for *Escherichia coli*, *Klebsiella pneumoniae*, and *Aerobacter aerogenes* are frequently obtained. Hyaline membrane disease and prematurity are often associated findings.[20]

The etiology is unknown. Some postulate a localized Shwartzman reaction. Others postulate pyogenic bacterial infection or infected amnionitis.[20] Perinatal hypoxia is also considered to be of etiologic significance and may be similar to the "diving reflex," in that blood is shunted away from certain areas in a response to hypoxia.[12]

Umbilical catheters deserve special attention, since their use increases the potential for sepsis and vascular thromboses.[27] In the present study, which did not attempt to encompass all of the cases of neonatal necrotizing enterocolitis seen at our institution, prematurity was a frequent finding.

Roentgenographically, submucosal accumulation of air is the common denominator present in all cases in this series despite the fact that pneumatosis by itself does not indicate necrotizing enterocolitis. Minimal dilatation associated with some edema of the mucosal folds may also be encountered (Fig. 3*A*). Portal venous gas and pneumoperitoneum are well recognized roentgenographic findings in this entity.[1] Goldstein *et al.*[7] reported a case with portal venous gas in a patient that survived.

Treatment of this entity consists of antibiotics, gastrostomy, resection of the necrotic segment, and supplementary hyperalimentation.[1]

(4) Nonocclusive ischemic enterocolitis is an entity which has been variously described in the past as uremic enterocolitis,[10] hemorrhagic enterocolitis,[28] acute postoperative enterocolitis,[19] staphylococcal enterocolitis, and terminal or preagonal enterocolitis.[15] It is now thought to be a rather specific entity caused by transient ischemia of the alimentary tract which is insufficient to cause full thickness infarction of the bowel.[15,23] The entire gastrointestinal tract or only a portion may be involved. The major blood vessels supplying the bowel are patent. Precipitating factors are numerous and they are: hypotension, splanchnic vasoconstriction, cardiac and aortic surgical operations, and intestinal obstruction. Predisposing factors include respiratory insufficiency, anemia, uremia, and a debilitated state. Schwartz *et al.*[23] have emphasized the role of congestive heart failure, shock, and especially bowel distention. The distended bowel apparently participates in a chain of events leading to progressive encroachment on the vascular integrity.

Pathologically, the affected portion of the gut in nonocclusive ischemic enterocolitis is intensely congested and frequently hemorrhagic with free blood in the lumen. Petechiae may also be identified. The serosa may be congested and discolored, or may appear quite normal. Ulcers, if present, are minute and irregularly distributed. They may encircle the entire lumen, or in the colon they may have a longitudinal orientation along the taeniae coli (Fig. 4*B*).

Microscopically, hemorrhages are found in the mucosa and submucosa. Fibrin thrombi in mucosal capillaries are characteristic of nonocclusive ischemic entero-

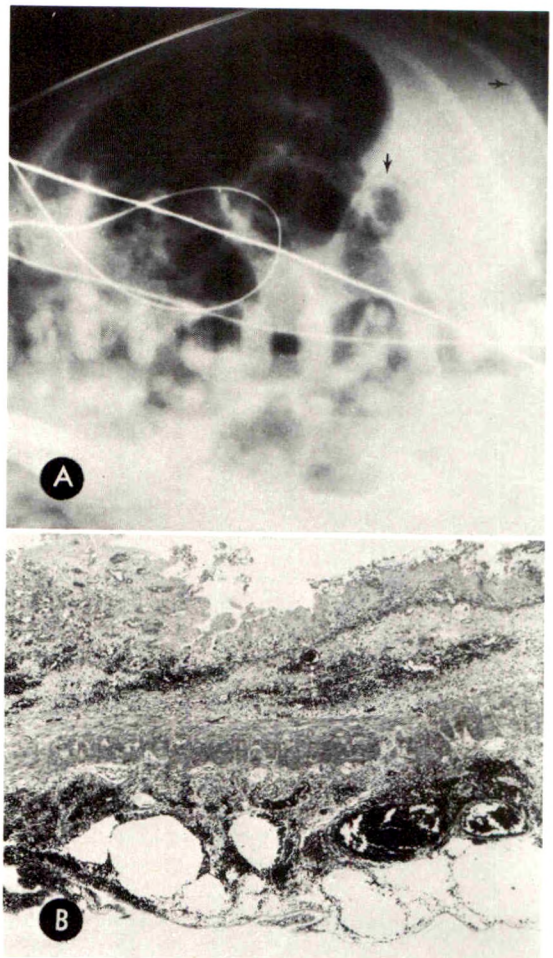


Fig. 3. (*A*) A left lateral decubitus roentgenogram from a newborn with necrotizing enterocolitis shows pneumatosis (↓), and portal venous air (→). (*B*) Microscopically there is extensive mucosal necrosis, inflammatory cell infiltrate and subserosal pneumatosis. (H. and E. 25×.)

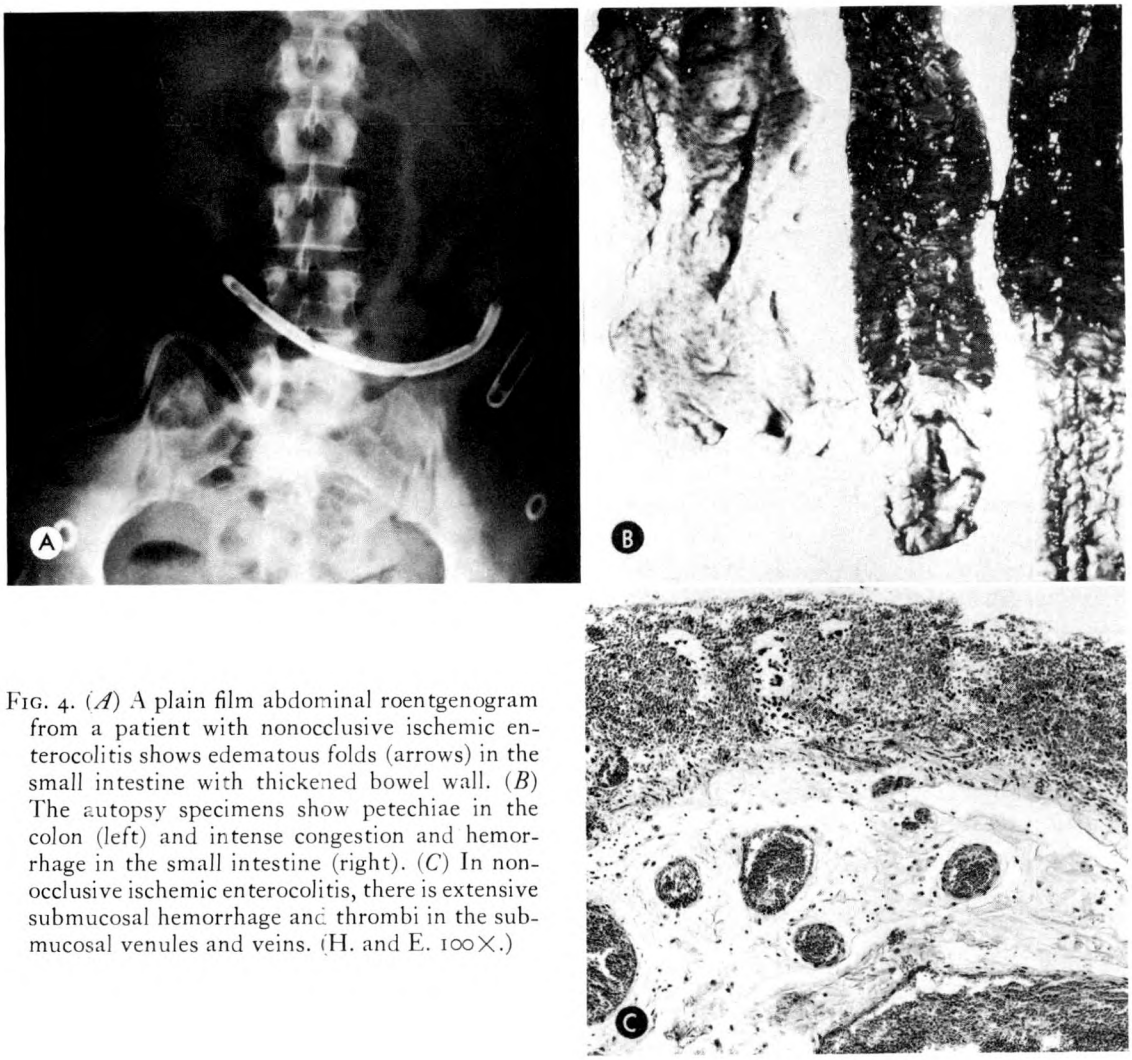


FIG. 4. (*A*) A plain film abdominal roentgenogram from a patient with nonocclusive ischemic enterocolitis shows edematous folds (arrows) in the small intestine with thickened bowel wall. (*B*) The autopsy specimens show petechiae in the colon (left) and intense congestion and hemorrhage in the small intestine (right). (*C*) In nonocclusive ischemic enterocolitis, there is extensive submucosal hemorrhage and thrombi in the submucosal venules and veins. (H. and E. 100×.)

colitis. In severe cases, they extend into the submucosal venules and veins. Sometimes there is actually necrosis of venules even when they do not contain thrombi. Apart from capillaries, the vessels affected are always veins and venules, never arteries or arterioles[14] (Fig. 4*C*). After 3 or 4 days the mucosa becomes necrotic, undergoes dissolution and ulcerates. Perforation may occur. Healing may be rapid if the disorder is mild. However, when ulceration extends through the muscularis mucosae, healing is by cicatricial granulation and at this stage one may refer to the disorder as chronic. Unsuspected retroperitoneal perforations were reported by Moss and Margulis.[17]

Clinically, patients are usually hospitalized with a severe illness presenting with shock and rectal bleeding. If the chronic form is present, symptoms of bowel obstruction may be present.

Roentgenographically, a wide spectrum of changes has been reported by Schwartz *et al.*[23] Edematous mucosal folds called pseudotumors, thumbprinting and transverse ridging are well recognized findings and are secondary to either congestion of the bowel wall and/or intramural bleeding. Small superficial mucosal ulcerations on contrast medium studies and occasionally isolated deep ulcerations have been reported. Intramural gas has been described,

but is an uncommon finding and was not present in any of the 18 cases in this series. In the present study, the paucity of roentgenographic findings was striking. In fact, the only consistent finding was a nonspecific dilatation of the colon and/or small bowel in the presence of minimal fold thickening (Fig. 4*A*). One patient in the present study had a complicating toxic megacolon.

In conclusion, we believe that most of the uncommon types of enterocolitis reported in the past 3 decades can be grouped into 4 basic categories based on clinical and autopsy findings. There is a great overlap in the roentgenologic findings in these entities which are nonspecific and reflect only insult to the bowel. As stated so eloquently by Margulis[13] in his address to the Radiological Society of North America in 1971, "It is important to realize that the colon, like the skin, has only a limited number of possible reactions to a host of harmful agents and processes and like the skin can blush, swell, ulcerate, or crack."

Certain roentgenographic signs may favor one diagnosis over others, but a more definitive diagnosis generally awaits clinical, laboratory, and more often surgical and/or autopsy verification. The differentiation of these entities from the more common types of inflammatory and ischemic lesions may be exceedingly difficult. The dramatic plain film roentgen findings in a severely ill patient with no history of preexisting inflammatory disease should at least suggest the possibility of these uncommon types of enterocolitis.

It is emphasized that even in the era of sophisticated special procedures and contrast medium studies, the plain film roentgenogram may be the only opportunity that the radiologist has to evaluate these critically ill patients, and its significance should not be underestimated.

SUMMARY

Various types of enterocolitis have been described in the past 3 decades and labeled as specific entities. We believe that these can be grouped into 4 convenient categories based on clinical and pathologic findings. The entities are discussed and the pathologic and roentgenographic features of each are described. The importance of the plain film abdominal roentgenogram is emphasized.

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REFERENCES

1. Bell, R. S., Graham, C. B., and Stevenson, J. K. Roentgenologic and clinical manifestations of neonatal necrotizing enterocolitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *112*, 123–134.
2. Benner, E. J., and Tellman, W. H. Pseudomembranous colitis as sequel to oral lincomycin therapy. *Am. J. Gastroenterol.*, 1970, *54*, 55–58.
3. Berdon, W. E., Grossman, H., Baker, D. H., Mizrahi, A., Barlow, O., and Blanc, W. A. Necrotizing enterocolitis in premature infants. *Radiology*, 1964, *83*, 879–887.
4. Durrani, K. M., and Gruhn, J. G. Pseudomembranous enterocolitis. *Am. J. Surg.*, 1963, *106*, 966–973.
5. Feinberg, S. B. Roentgen findings in severe pseudomembranous enterocolitis. *Radiology*, 1960, *74*, 778–783.
6. Finney, J. M. T. Gastroenterostomy for cicatrizing ulcer of pylorus. *Bull. Johns Hopkins Hosp.*, 1893, *3*, 53–55.
7. Goldstein, W. B., Cusmano, J. V., Gallagher, J. J., and Hemley, S. Portal vein gas: case report with survival. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *97*, 220–222.
8. Goulston, S. J. M., and McGovern, V. J. Pseudo-membranous colitis. *Gut*, 1965, *6*, 207–212.
9. Hopkins, G. B., Gould, V. E., Stevenson, J. K., and Oliver, T. K., Jr. Necrotizing enterocolitis in premature infants. *A.M.A. Am. J. Dis. Child.*, 1970, *111*, 749–752.
10. Jaffe, R. H., and Lang, D. E. Changes of digestive tract in uremia. *Arch. Int. Med.*, 1934, *53*, 851–864.
11. Jeckeln, E. Über "Darmbrand;" das pathologisch-anatomisches Bild des Darmbrandes. *Deutsche med. Wchnschr.*, 1947, *72*, 105–108.
12. Lloyd, J. R. Etiology of gastrointestinal perforations in newborn. *J. Pediat. Surg.*, 1969, *4*, 77–84.
13. Margulis, A. R. Radiology of ulcerating colitis. *Radiology*, 1972, *105*, 251–263.

14. McGOVERN, V. J. Differential diagnosis of colitis. *Pathology Annual*, 1969, *4*, 127–158.
15. McGOVERN, V. J., and GOULSTON, S. J. M. Ischaemic enterocolitis. *Gut*, 1965, *6*, 213–220.
16. MENAKER, G. J., and VAN HAGEN, F. Evaluation of distention and stasis in pathogenesis of experimental pseudomembranous enterocolitis. *Am. J. Surg.*, 1960, *99*, 292–300.
17. MOSS, A. A., and MARGULIS, A. R. Ischemic colitis with perforation. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 338–342.
18. MURRELL, T., and ROTH, L. Necrotizing jejunitis: newly discovered disease in highlands of New Guinea. *Med. J. Aust.*, 1963, *1*, 61–69.
19. PENNER, A., and BERNHEIM, A. I. Acute postoperative enterocolitis: study on pathologic nature of shock. *Arch. Path.*, 1938, *27*, 966–983.
20. POCHACZEVSKY, R., and KASSNER, E. G. Necrotizing enterocolitis of infancy. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 283–296.
21. RIGLER, L. G., and POGUE, W. L. Roentgen signs of intestinal necrosis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 402–409.
22. SCHAPIRO, R. L., and NEWMAN, A. Acute enterocolitis. *Radiology*, 1973, *108*, 263–268.
23. SCHWARTZ, S., BOLEY, S., SCHULTZ, L., and ALLEN, A. Survey of vascular diseases of small intestine. *Seminars in Roentgenol.*, 1966, *1*, 178–218.
24. SPEARE, G. S. Staphylococcus pseudomembranous enterocolitis: complication of antibiotic therapy. *Am. J. Surg.*, 1954, *88*, 523–534.
25. TAN, T., DRAKE, C. T., JACOBSON, M. J., and VAN PROHASKA, J. Experimental development of pseudomembranous enterocolitis. *Surg., Gynec. & Obst.*, 1959, *108*, 415–420.
26. TULLY, T. E., and FEINBERG, S. B. Reappearance of antibiotic induced pseudomembranous enterocolitis. To be published.
27. WILLIAMS, H. J., JARVIS, C. W., NEAL, W. A., and REYNOLDS, J. W. Vascular thromboembolism complicating umbilical artery catheterization. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *116*, 475–486.
28. WILSON, R., and QUALHEIM, R. E. Form of acute haemorrhagic enterocolitis afflicting chronically ill individuals. *Gastroenterology*, 1954, *27*, 431–444.
29. ZEISSLER, J., and RASSFELD-STERNBERG, L. Enteritis necroticans due to *Clostridium welchii* type F. *Brit. M. J.*, 1949, *1*, 267–269.

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CLINDAMYCIN (CLEOCIN) COLITIS*

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RECENTLY, clindamycin hydrochloride hydrate (Cleocin) has been reported to cause a severe protracted colitis.[3] This type of colitis may be indistinguishable clinically and roentgenographically from inflammatory or vascular bowel disease.

The purpose of this report is to describe further the clinical and roentgenographic manifestations of clindamycin-associated colitis. To our knowledge, this is the first report in detail of the roentgenologic findings of this drug-associated colitis. These findings deserve emphasis as the radiologist should include this entity in the differential diagnosis of inflammatory or vascular bowel disease.

REPORT OF CASES

CASE I. I.B. A 79 year old Caucasian female with a past history of right radical mastectomy for carcinoma of the right breast accidentally cut the first finger of her right hand. This resulted in fever and cellulitis, which was treated with oral and parenteral penicillin. Because the cellulitis failed to resolve with penicillin therapy, clindamycin, 150 mg. q.i.d., was administered. Cramping abdominal pain, fever, and diarrhea (8 to 12 bowel movements per day) without hematochezia began 1 day after the initiation of clindamycin therapy. Despite the cessation of clindamycin after 4 days and symptomatic treatment including steroid enemas, severe abdominal cramping and diarrhea persisted for 3 weeks, necessitating hospitalization. Proctoscopic examination revealed an edematous, nonfriable, hemorrhagic mucosa with cream-colored plaques scattered throughout the rectum. The barium enema roentgenogram revealed a nodular mucosal pattern throughout the entire colon and rectum without ulceration or terminal ileum involvement (Fig. 1). The patient improved steadily with treatment of nasogastric suction, intravenous fluids, and diphenoxylate hydrochloride (Lomotil). Six weeks after the administration of clindamycin, the patient was asymptomatic and the barium enema examination results were normal.

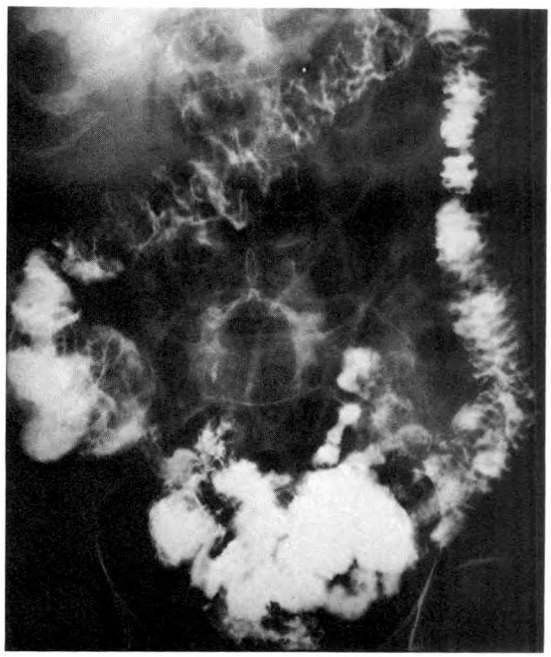


FIG. 1. Case I. Postevacuation barium enema roentgenogram reveals thickened mucosal folds with a nodular pattern throughout the entire colon.

CASE II. C.M. A 22 year old white woman was admitted to the Obstetrical Service in active labor precipitated 1 month prematurely by a diarrheal illness. Two weeks prior to delivery, the patient developed an upper respiratory tract infection for which clindamycin hydrochloride monohydrate, 150 mg. q.i.d., was prescribed for 1 week. With this regimen, the patient developed 2 loose stools per day. The upper respiratory tract symptoms abated, and the stools returned to normal with cessation of clindamycin therapy. Six days later the patient developed crampy lower abdominal pain, fever, and watery diarrhea 6 to 10 times daily. Spontaneous labor resulted in the delivery of a 2.4 kg. infant. Proctoscopic examination revealed an edematous but nonfriable rectal mu-

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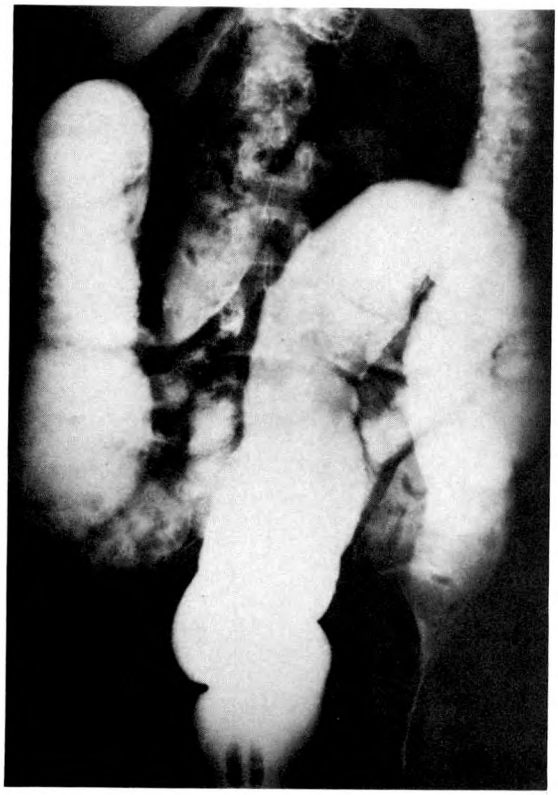


FIG. 2. Case II. Filled barium enema roentgenogram shows multiple irregular filling defects throughout the entire colon.

cosa with multiple cream-colored, plaque-like lesions. A barium enema roentgenogram later revealed an ahaustral colon containing numerous persistent irregular filling defects; multiple ulcerations were present throughout the entire colon (Fig. 2; and 3). The rectum was less severely involved, and the terminal ileum was normal. These roentgenographic findings suggested idiopathic ulcerative colitis, although ischemic or infectious colitis was considered. Rectal biopsy showed nonspecific inflammation. No pathogenic bacteria or fungi were recovered on culture. The patient showed rapid clinical improvement with the intravenous administration of corticotropin and subsequent oral prednisone therapy. One month following hospital discharge, the patient was having 1 to 2 formed bowel movement a day while taking 20 mg. of prednisone daily.

CASE III. R.A. A 61 year old white male sustained his second myocardial infarction on February 7, 1973. His hospital course was complicated by bronchitis and congestive heart failure for which he received clindamycin, digitalis, and anticoagulant therapy. Several days later he developed right-sided abdominal pain and an abdominal mass. Exploratory laparotomy revealed a rectus sheath hematoma, massive ascites, and a dilated and thickened edematous colon. Clindamycin was stopped at the time of laparotomy. The postoperative course was complicated by profuse watery diarrhea, oliguria, azotemia, and Cheyne-Stokes respirations. The patient later improved, and a barium enema roentgenogram was obtained. Diffuse ulceration from the rectum to the cecum was interpreted as compatible with ischemic or ulcerative colitis (Fig. 4). Prednisone, 50 mg. q.i.d., resulted in improvement of the diarrhea, and the patient was transferred to Brooke Army Medical Center 6 weeks after initial hospitalization. On admission, a supine abdominal roentgenogram showed a markedly dilated toxic megacolon (Fig. 5). Proctosigmoidoscopy demonstrated numerous 2–3 mm. raised, white-to-yellow, creamy plaques characteristic of clindamycin-associated colitis (Fig. 6). Cultures for fungi, ova, and parasites were negative. Rectal biopsy showed nonspecific mucosal ulceration and acute inflammation. Corticosteroids were continued, and total parenteral nutrition was initiated because of persistent

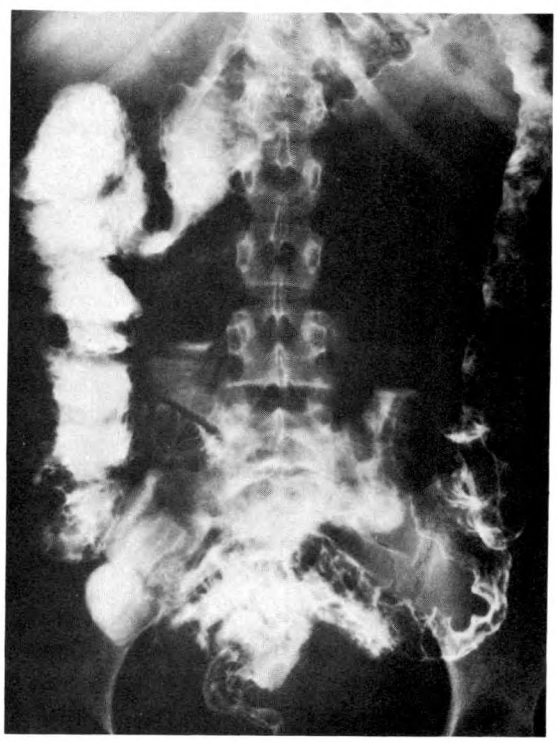


FIG. 3. Case II. Postevacuation roentgenogram shows marked irregularity and ulceration of the mucosa.

megacolon. The patient subsequently developed a *Candida* fungemia, disseminated intravascular coagulopathy, and expired on the seventeenth day after transfer to Brooke Army Medical Center. Necropsy confirmed the presence of an acute colitis with marked dilatation. Despite evidence of generalized arteriosclerosis, microscopic sections showed no evidence of colonic ischemia.

DISCUSSION

Clindamycin hydrochloride hydrate is a new synthetic antibiotic, *i.e.*, a 7 chloro-7 deoxy modification of lincomycin. Clindamycin reportedly has better absorption and fewer gastrointestinal side effects than the parent compound, lincomycin. Clindamycin is used widely for its antibacterial activity against gram-positive organisms and is the drug of choice in the treatment of anaerobic infections.[1] The reported incidence of mild diarrhea following clindamycin administration has varied from 4 per cent to 33 per cent.[4,5]



Fig. 4. Case III. Oblique roentgenogram of the colon shows the marked mucosal ulceration.

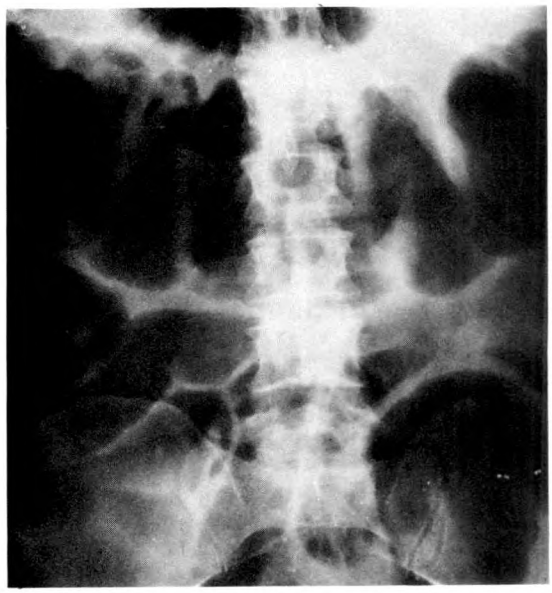


Fig. 5. Case III. Note the severely dilated megacolon which measured 13.5 cm. to the right of the spine.

In our series of 6 confirmed cases, the main clinical feature was the sudden onset of severe diarrhea and abdominal pain mimicking inflammatory or vascular bowel disease. This occurred coincident with the simultaneous oral administration or completion of clindamycin therapy. All patients had a protracted course of several weeks, requiring hospitalization despite cessation of clindamycin therapy.

Barium enema examinations performed on 6 of these patients revealed abnormalities in 3. The findings included marked mucosal changes either of a diffuse ulceration with or without intraluminal masses or a generalized mucosal nodularity. In all cases, there was total colonic involvement. Similar rectal abnormalities were present, although more subtle in appearance. An additional finding in Case III was the occurrence of toxic megacolon. Heretofore, this has been described only in ulcerative colitis, granulomatous colitis,[8] amebic colitis,[9] and pseudomembranous enterocolitis.[2]

It is readily apparent from the cases presented that the roentgenographic findings of clindamycin colitis are nonspecific. The 2 cases of diffuse colonic ulceration suggested the diagnosis of idiopathic ulcerative or ischemic colitis, although an infectious

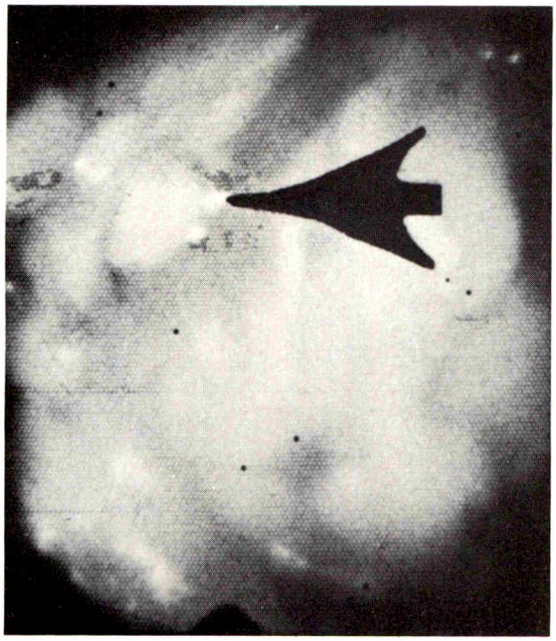


Fig. 6. Case III. Colonoscopic photograph. Characteristic 2–3 mm. diameter raised, white to cream-colored plaques are seen peripherally. These have become confluent centrally. Also noted is bleeding due to contact friability of the mucosa.

etiology was also considered. The mucosal nodularity noted in 1 case was attributed to Crohn's disease, although infiltrative diseases such as lymphosarcoma, reticulum cell sarcoma, and amyloidosis were suggested.

A case similar to these was discussed by Harkleroad and Rinaldo.[6] A 76 year old female treated with novobiocin-tetracycline capsules developed a colitis with 1 cm. creamy-white mucosal patches which eventually became confluent. Biopsy showed nonspecific inflammatory changes. Barium enema examination was consistent with a diffuse ulcerative process of the colon.

In all of our cases, proctoscopic findings of a distinctive, elevated, cream-colored, plaque-like lesion superimposed on an erythematous and friable or edematous mucosa were discovered. Biopsy of the plaque-like lesions revealed nonspecific inflammatory changes; no pathogens were obtained on culture. Treatment consisted of intravenous hydrocortisone, hydrocortisone enemas, and replacement of oral intake with intravenous fluids.

SUMMARY

Clindamycin (Cleocin) is a synthetic antibiotic used widely against gram-positive and anaerobic organisms.

In 6 patients receiving this drug, we have observed the acute onset of colitis lasting several weeks. Barium enema examinations in all patients revealed that 3 had marked mucosal abnormalities and 3 were normal. The changes included diffuse ulcerations, irregular mucosal masses, or uniform nodularity of the entire colon and rectum.

The diagnosis is established by the proctoscopic finding of unique cream-colored, plaque-like lesions superimposed on a friable or edematous mucosa.

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REFERENCES

1. Bartlett, J. G., Sutter, V. L., and Finegold, S. M. Treatment of anaerobic infections with lincomycin and clindamycin. *New England J. Med.*, 1972, *287*, 1006–1010.
2. Brown, C. H., Ferrante, W. A., and Davis, W. D., Jr. Toxic dilatation of colon complicating pseudomembranous enterocolitis. *Am. J. Digest. Dis.*, 1968, *13*, 813–821.
3. Cohen, L. E., McNeill, C. J., and Wells, R. F. Clindamycin-associated colitis. *J.A.M.A.*, 1973, *223*, 1379–1380.
4. DeHaan, R., Schellenberg, D., Vandenbosch, W. D., and Maile, M. H. Clindamycin palmitate in healthy men: general tolerance and effect on stools. *Current Therapy Res.*, 1972, *14*, 81–90.
5. Geddes, A. M., Bridgwater, F. A., Williams, D. N., Oon, J., and Grimshaw, G. J. Clinical and bacteriological studies with clindamycin. *Brit. M. J.*, 1970, *2*, 703–704.
6. Harkleroad, H. N., and Rinaldo, J. A., Jr. Acute atypical colitis after antibiotic therapy. *Gastroenterology*, 1962, *43*, 575–587.
7. Schapiro, R. L., and Newman, A. Acute enterocolitis: complication of antibiotic therapy. *Radiology*, 1973, *108*, 263–268.
8. Soll, E. L., Ferrante, W. A., and Gathright, J. B. Jr. Toxic dilatation of colon due to granulomatous colitis (Crohn's disease). *South. M. J.*, 1971, *64*, 349–353.
9. Wruble, L. D., Duckwith, J. K., Duke, D. D., and Rothschild, J. H. Toxic dilatation of colon in case of amebiasis. *New England J. Med.*, 1966, *275*, 926–928.

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INFLAMMATORY PSEUDOTUMOR OF THE SMALL INTESTINE*

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INFLAMMATORY pseudotumor[4] (eosinophilic granuloma,[6] inflammatory fibroid polyp,[3] Vaněk tumor[11]) is an uncommon non-neoplastic predominantly submucosal lesion of the gastrointestinal tract. Vaněk[11] was first to define the entity histologically, based upon 6 cases of what he termed "gastric granuloma with eosinophilic infiltration." The roentgenographic features of gastric "granuloma with eosinophils" have been described by Rigler *et al.*[7]

The gastric antrum is the most frequently reported site of inflammatory pseudotumors. Reports of extragastric locations have been rare, with the small intestine being the second most commonly reported location.

The purpose of this paper is to describe the roentgenographic findings in inflammatory pseudotumor of the small intestine. The roentgen features will be compared to those of eosinophilic gastroenteritis, a condition thought by some authors to be related to inflammatory pseudotumor.[8,10] The etiology, pathology, and clinical aspects will be considered.

REPORT OF CASES

CASE I. S.W., 177-04-83, a 49 year old female, was admitted to Columbia-Presbyterian Medical Center (CPMC) because of rectal bleeding and weight loss. There was no history of allergies. Abdominal examination was normal. Laboratory data included a hematocrit of 26 per cent, a normal leukocyte count and differential count with no eosinophils.

Upper gastrointestinal series and tubeless hypotonic duodenography revealed a 2.5 cm. sessile polypoid mass in the first portion of the duodenum (Fig. 1). Duodenoscopy revealed the mass to be soft and pliable with normal overlying mucosa.

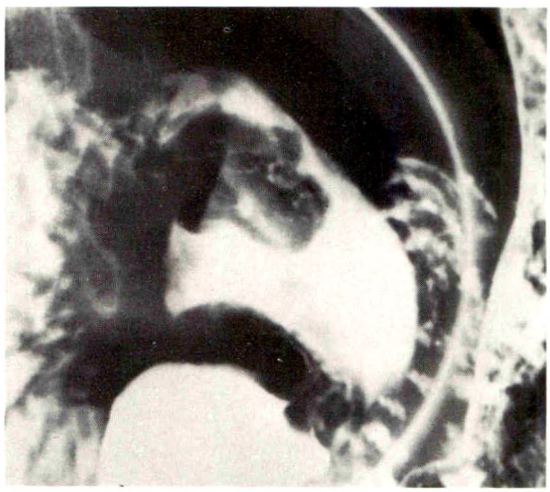


FIG. 1. Case I. Inflammatory pseudotumor of the duodenum. In the apex of the bulb, there is a 2.5 cm. sessile polypoid mass. The adjacent mucosa is normal. The roentgenographic appearance simulates that of a primary duodenal neoplasm.

At laparotomy, a 3 cm. mass was found in the first portion of the duodenum. A biopsy was taken.

The specimen was a 1.7×1.2 cm. piece of homogeneous gray tissue. Histologically, inflamed granulation tissue, containing numerous scattered eosinophils and histiocytes, was seen. The overlying mucosa was unremarkable and the adjacent Brunner glands appeared hyperplastic.

Pathologic diagnosis was inflammatory pseudotumor of the duodenum, with Brunner gland hyperplasia.

Comment. This inflammatory pseudotumor of the first portion of the duodenum (Fig. 1) simulated a benign neoplasm roentgenographically. The source of the rectal bleeding in this patient was not determined with certainty. It probably was not related to the inflammatory pseudotumor.

CASE II. E.G., 81-16-69, a 60 year old female, was admitted to CPMC for elective cholecys-

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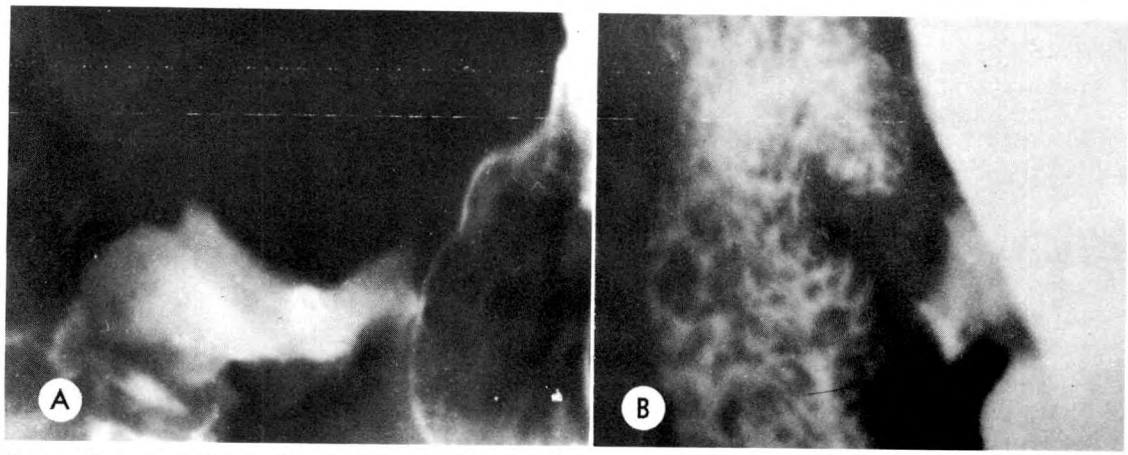


FIG. 2. Case II. Inflammatory pseudotumor of the duodenum. (*A*) Four years before, the patient had an ulcer in the duodenal bulb. (*B*) Four months prior to admission, the bulb appeared deformed.

At operation, a 1 cm. inflammatory pseudotumor was found on the greater curvature aspect; an old anterior peptic ulcer scar was also present. This case is of interest because the inflammatory pseudotumor occurred in a patient with a previous documented peptic ulcer. Such an association has been noted by other authors.

tectomy for cholelithiasis. She had had symptoms of peptic ulcer disease for 7 years. There was no history of allergies.

Four years before admission, a duodenal ulcer was documented on upper gastrointestinal series (Fig. 2*A*). Another upper gastrointestinal series 4 months prior to admission demonstrated a deformed duodenal bulb interpreted as being consistent with chronic peptic ulcer disease (Fig. 2*B*).

Physical examination and laboratory tests were normal, including a blood leukocyte count of 5,400, with a normal differential count and no eosinophils.

At cholecystectomy, the duodenal bulb exhibited an old peptic ulcer scar. A well delineated, hard, spherical 1 cm. mass was excised from the greater curvature of the bulb just distal to the pylorus.

This mass measured 0.7×0.6×0.5 cm. and was composed of loose myxoid fibrous tissue with scattered small vascular channels. Numerous inflammatory cells, predominantly eosinophils and polymorphonuclear leukocytes, were seen, but lymphocytes, histiocytes and plasma cells were also recognized.

Pathologic diagnosis was inflammatory pseudotumor of the duodenum.

Comment. This lesion, as were some of the inflammatory pseudotumors reported in the literature, as well as our Cases III and IV, was originally diagnosed pathologically as a fibroma. On the basis of current histologic criteria, the lesion is considered to be inflammatory, not neoplastic.

The development of an inflammatory pseudotumor at the site of a previous peptic ulcer suggests the possibility of a causal relationship. Animal experiments have shown that similar lesions may be produced in the stomach by intramural injection of foreign materials such as gastric juice, barium sulfate, and various commercially available antacid preparations.[5] The association of inflammatory pseudotumor and peptic ulcer in the stomach has been previously noted.[9,11]

CASE III. V. De A., 138-59-45, a 60 year old male, presented to CPMC with a 2 month history of crampy abdominal pain, vomiting, and weight loss. He denied any allergies. Twenty-seven years before, he had experienced 4 years of "asthma and sinusitis." This had not recurred.

Physical examination revealed a movable lower abdominal mass. Blood leukocyte count and differential count were normal with 1 per cent eosinophils. Hematocrit was 45 per cent, stool guaiac 3+ positive.

Abdominal roentgenograms demonstrated dilated intestinal loops with air-fluid levels consistent with a distal small bowel obstruction. Roentgenograms following instillation of barium sulfate through a Miller-Abbott tube,

showed a 5×10 cm. intussuscepting mass in the ileum (Fig. 3).

At operation, the intussuscepted ileal mass was resected. Two postoperative blood leukocyte differential counts showed 3 per cent and 1 per cent eosinophils.

The resected specimen was a 30 cm. dilated segment of ileum with an intussusception. The line of invagination was 3 cm. below the proximal margin of excision. On opening the bowel, a 9×5×4 cm. polypoid mass projected into the lumen. On microscopic examination, the superficial portions of the lesion were composed of inflamed granulation tissue. The mucosa was ulcerated. In the deeper portions of the specimen, a more hyalinized dense fibrous tissue with scattered small blood vessels was seen. Fewer inflammatory cells were identified. No smooth muscle was recognized with the trichrome stain.

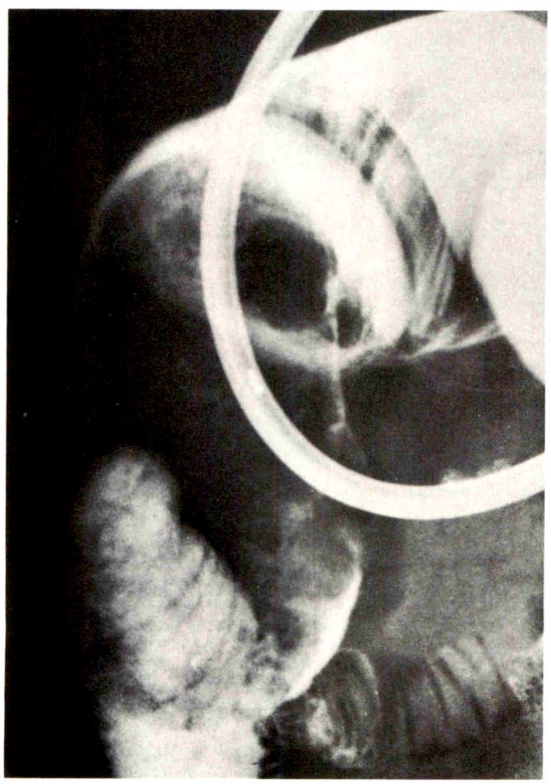


Fig. 3. Case III. Inflammatory pseudotumor of the ileum. Barium examination *via* a Miller-Abbott tube outlines a 5×10 cm. intussuscepting mass in the distal ileum. The bowel proximal to the obstructing pseudotumor is dilated. The roentgenographic appearance is similar to that seen with small bowel neoplasms.

Pathologic diagnosis was inflammatory pseudotumor of the ileum.

Comment. In this patient, a large inflammatory pseudotumor intussuscepted and caused obstruction. Most of the reported cases involving the ileum have presented in this manner.

This case also illustrates one reason why the term "eosinophilic granuloma" should probably not be used for these lesions. On histologic examination, this large mass demonstrated virtually no eosinophils.

Case IV. S.S., 44-82-90, a 58 year old female, was admitted to CPMC with a 5 month history of crampy lower abdominal pain associated with vomiting. There was no history of allergies.

On physical examination, a 10 cm. mass was palpable in the left upper quadrant, and definite peristaltic waves could be seen on the abdominal wall. Blood leukocyte count was 5,500 with a normal differential count. Blood erythrocyte count was 3.9 million/mm.^{3}. Stool for blood was negative.

Abdominal roentgenograms in the anteroposterior and erect positions revealed distended intestinal loops with air-fluid levels consistent with a distal small bowel obstruction.

At operation, a 3×4.5 cm. intussuscepting mass in the mid-ileum was found. Partial ileal resection was performed.

The resected specimen was a 22 cm. segment of ileum with an intussusception. On opening the bowel, a 4.5×3×2 cm. polypoid mass projected into the lumen. The proximal intestine demonstrated marked dilatation. On microscopic examination, the lesion was located in the submucosa. The mass was composed of fibrous tissue of variable cellularity with numerous scattered eosinophils and small vascular channels. The surface showed an ulcerated mucosa, beneath which inflamed granulation tissue was identified.

Pathologic diagnosis was inflammatory pseudotumor of the ileum.

Comment. Obstruction and intussusception are a typical clinical presentation for an ileal inflammatory pseudotumor. This case, like Cases II and III, was originally considered histologically to be a fibroma.

Case V. M.S., 95-25-88, a 62 year old male, had a 1 year history of postprandial abdominal pain. Two weeks prior to admission at CPMC, he noted the onset of vomiting and tarry stools.

An upper gastrointestinal series done at another hospital was interpreted as showing a prepyloric antral lesion compatible with gastric carcinoma. There was no history of allergies.

On physical examination, a right upper quadrant abdominal mass was felt. The hematocrit was 46 per cent. No blood leukocyte count or stool test for blood was performed.

At laparotomy, benign antral and duodenal ulcers were found. Partial gastrectomy and gastrojejunostomy were performed.

The resected specimen was the distal portion of the stomach, measuring 12 cm. along the lesser curvature and 25 cm. along the greater curvature. Distally, the attached duodenal cuff measured 2 cm. Two ulcers were recognized along the lesser curvature of the stomach and one in the duodenum. On microscopic examination, a 0.7 cm. circumscribed nodule was noted in the submucosa, forming one wall of the duodenal ulcer. This lesion was composed of inflamed granulation tissue with scattered inflammatory cells including eosinophils, lymphocytes and histiocytes.

Pathologic diagnoses were inflammatory pseudotumor of the duodenal bulb, duodenal ulcer, and 2 benign gastric antral ulcers.

Comment. As in Case II, this small inflammatory pseudotumor of the duodenal bulb was associated with peptic ulcer disease.

CASE VI. J.F., a 49 year old male, complained of intermittent crampy lower abdominal pain of 6 weeks' duration. There was no history of allergies.

Barium enema examination done prior to admission showed reflux into the terminal ileum that partially outlined a 4.5×8 cm. intraluminal mass (Fig. 4*A*). Small intestinal examination again demonstrated the mass, with dilated ileum proximally (Fig. 4*B*).

On physical examination, a tender mass was palpable in the mid-lower abdomen. Hematocrit was 41 per cent, hemoglobin 13.9 gm. per cent. Four blood leukocyte counts and differential counts were normal with 1, 2, 2, and 4 per cent eosinophils. No stool for occult blood was obtained.

At operation, a 4.5×11 cm. ileal mass approximately 30 cm. from the ileocecal valve was found. Partial ileal resection with end-to-end anastomosis was performed.

The resected specimen was a 19 cm. dilated segment of ileum. On opening the bowel, a 4.5×10 cm. mass occupied almost the entire lumen. The base of the mass extended transmurally, and presented on the serosal surface as a 4 cm. smooth grayish-white, hemispheric elevation. On microscopic examination, the mass extended from the muscularis mucosae to the serosa, replacing the muscularis propria. The mucosa was thinned and ulcerated over the central portion. Histologically, the lesion consisted mainly of inflamed granulation tissue

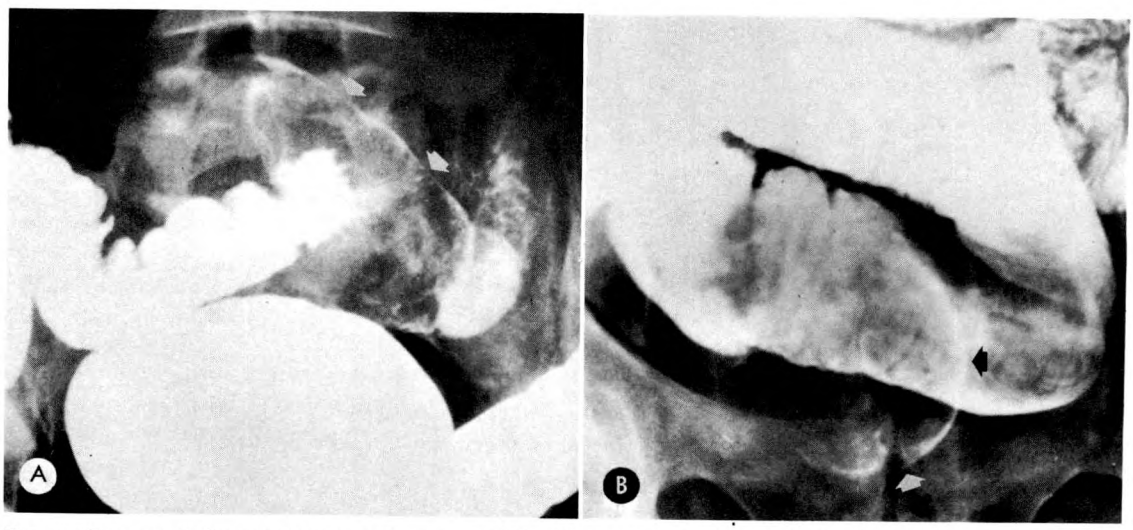


FIG. 4. Case VI. Inflammatory pseudotumor of the ileum. (*A*) Reflux of barium into the terminal ileum during barium enema examination outlines a 4.5×8.0 cm. intraluminal mass (arrows). (*B*) Small intestinal examination demonstrates the mass (arrows) with dilated ileum proximally.

with a loose fibrous stroma and numerous small blood vessels. The heavy infiltration of chronic inflammatory cells was comprised predominantly of eosinophils with a smaller number of histiocytes, polymorphonuclear leukocytes, and plasma cells.

Pathologic diagnosis was inflammatory pseudotumor of the ileum.

DISCUSSION

On the basis of current knowledge, there are 2 distinct, apparently unrelated forms of eosinophilic infiltration of the gastrointestinal tract. One, characterized by diffuse involvement of varying portions of stomach and small bowel and frequently associated with allergies and blood eosinophilia, is best termed eosinophilic gastroenteritis.[2,10] The other is a localized mass found anywhere in the gut, but most frequently in the gastric antrum; it is not accompanied by allergies or eosinophilia. For this lesion, we prefer the term inflammatory pseudotumor.[4]

The term "eosinophilic granuloma" has often been applied to both eosinophilic gastroenteritis and inflammatory pseudotumor.[6,8] The name is undesirable, however, because it fails to distinguish between these 2 quite different lesions, and because it incorrectly suggests a relationship to the more common eosinophilic granuloma of bone and other tissues. Furthermore, the number of eosinophils seen in inflammatory pseudotumor is actually quite variable; some lesions have virtually none (Case III).

On gross pathologic examination, the inflammatory pseudotumor of the small intestine usually is a well localized lesion. When small, it may have the appearance of a nodule or an area of thickening in the bowel wall. When larger, these lesions may produce polypoid masses which may lead to intussusception.

On histologic examination, these specimens basically have the appearance of inflamed granulation tissue. The fibrous stroma demonstrates variable cellularity. In areas in which the stroma has been recently formed, the fibrocytic cells have the appearance of actively proliferating fibroblasts. In other lesions, these cells appear more mature. Small blood vessels are scattered throughout and may be very prominent.

The inflammatory infiltrate is also quite variable in cellularity. In some lesions, the inflammatory cells are quite prominent; in others they are sparse. Usually present are eosinophils and histiocytes, but lymphocytes and plasma cells may also be seen. If the mucosa is ulcerated, polymorphonuclear leukocytes are also identified. In summary, histologic examination demonstrates a lesion composed of inflamed granulation tissue, with the cellularity of the fibrous tissue, the number of blood vessels and the inflammatory cell component varying, probably with the age of the lesion.

The roentgenographic features of inflammatory pseudotumor of the small intestine are those of a localized submucosal mass. Small lesions less than 1 cm. in diameter are inconspicuous and produce no (Case V) or nonspecific (Case II, Fig. 2*B*) changes on upper gastrointestinal and small intestinal series. The findings may be overshadowed by coexisting disease such as peptic ulceration or deformity.

Larger inflammatory pseudotumors simulate neoplasms (Fig. 1). They may obstruct (Fig. 4, *A* and *B*) and intussuscept (Fig. 3). Lesions originating in the gastric antrum or pylorus can prolapse into the duodenal bulb and present there as polypoid filling defects.[7] Ulceration occurs (Cases IV and VI) and has been demonstrated roentgenographically in gastric inflammatory pseudotumors.[7]

Despite certain histologic similarities between inflammatory pseudotumor and eosinophilic gastroenteritis, the roentgenologist is unlikely to confuse the two.

Eosinophilic gastroenteritis is a diffuse lesion characterized histologically by edema and eosinophilic infiltration of the bowel wall. The roentgen changes in the small intestine simulate an inflammatory process such as Crohn's disease; segments of bowel

show thickening of the wall, rigidity and luminal narrowing.[1]

The clinical manifestations of inflammatory pseudotumor are nonspecific and depend largely on the size and location of the mass. Based largely on experience with gastric inflammatory pseudotumors, Ureles *et al.*[10] concluded that a typical history is one of abdominal pain of days' to months' duration, often with ulcer-like symptoms. Not uncommonly, no symptoms are present. The more rare lesions of the jejunum and ileum have characteristically presented with intestinal obstruction. In contradistinction to eosinophilic gastroenteritis, inflammatory pseudotumor is not associated with clinical allergies and increased numbers of blood eosinophils.

Previous experience with duodenal inflammatory pseudotumors is so limited that no generalizations regarding the clinical features can be made. In our patients, the symptoms were probably due to peptic ulcer disease in one (Case V) and cholecystitis and peptic ulcer disease in a second (Case II). In the third duodenal case (Case I), there were no symptoms specifically attributable to the lesion.

The etiology of inflammatory pseudotumor is unknown. Moran and Sherman[5] produced lesions in the rabbit stomach identical to inflammatory pseudotumor by injecting various foreign substances, including meat, barium sulfate, gastric juice, and several antacid preparations used in the treatment of peptic ulcer. In a companion paper, they noted in humans the association of gastric granulomas with peptic ulcers of the stomach and duodenum. They speculated that food particles or digested smooth muscle of the gastric wall might incite the formation of the inflammatory mass.[9] Our Cases II and V of duodenal inflammatory pseudotumor associated with peptic ulceration suggest the possibility of a similar mode of origin. Despite speculation, there is no proof that inflammatory pseudotumors are caused by intestinal parasites.

SUMMARY

Inflammatory pseudotumor of the small intestine is a localized predominantly submucosal mass characterized histologically by inflamed granulation tissue with variable numbers of eosinophils. The roentgenographic features simulate those of a small bowel submucosal neoplasm. Filling defects, evidence of obstruction and intussuscepting masses may be demonstrated.

Inflammatory pseudotumor should be distinguished from another form of eosinophilic infiltration of the gut, eosinophilic gastroenteritis. These 2 lesions appear to be distinct on clinical, pathologic, and roentgenographic grounds. The use of the term "eosinophilic granuloma" to refer to either or both entities is discouraged.

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REFERENCES

1. BURHENNE, H. J., and CARBONE, J. V. Eosinophilic (allergic) gastroenteritis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 332–338.
2. EDELMAN, M. J., and MARCH, T. L. Eosinophilic gastroenteritis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 773–778.
3. HELWIG, E. B., and RANIER, A. Inflammatory fibroid polyps of stomach. *Surg., Gynec. & Obst.*, 1953, *96*, 355–367.
4. LATTES, R. and STOUT, A. P. Cited in Burkhart, C. R., and Wilkinson, R. H., Jr. Inflammatory pseudotumors of stomach: report of 2 cases. *Cancer*, 1965, *18*, 1310–1316.

5. Moran, T. J., and Sherman, F. E. Granulomas of stomach. II. Experimental production by intramural injection of foreign material including gastric juice. *Am. J. Clin. Path.*, 1954, *24*, 422–433.
6. Polayes, S. H., and Krieger, J. L. Eosinophilic granuloma of jejunum: hitherto undescribed lesion of intestines. *J.A.M.A.*, 1950, *143*, 549–551.
7. Rigler, L. G., Blank, L., and Hebbel, R. Granuloma with eosinophils: benign inflammatory fibroid polyps of stomach. *Radiology*, 1956, *66*, 169–176.
8. Salmon, P. R., and Paulley, J. W. Eosinophilic granuloma of gastro-intestinal tract. *Gut*, 1967, *8*, 8–14.
9. Sherman, F. E., and Moran, T. J. Granulomas of stomach. I. Response to injury of muscle and fibrous tissue of wall of human stomach. *Am. J. Clin. Path.*, 1954, *24*, 415–421.
10. Ureles, A. L., Alschibaja, T., Lodico, D., and Stabins, S. J. Idiopathic eosinophilic infiltration of gastrointestinal tract, diffuse and circumscribed: proposed classification and review of literature, with two additional cases. *Am. J. Med.*, 1961, *30*, 899–909.
11. Vaněk, J. Gastric submucosal granuloma with eosinophilic infiltration. *Am. J. Path.*, 1949, *25*, 397–412.

FREQUENCY OF VISUALIZATION OF THE NORMAL APPENDIX WITH THE BARIUM ENEMA EXAMINATION*

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IT is generally accepted that filling of the appendiceal lumen with barium and visualization of the entire appendix is a reliable sign of nondiseased appendix, since obstruction of its lumen is considered to be a prerequisite in the development of appendicitis.[10,11] Low pressure retrograde filling of the colon has not been shown to precipitate any complication when appendicitis is present.[8–10] Nonfilling and nonvisualization, however, have not been considered to be of diagnostic significance, unless associated changes in the cecum or terminal ileum are also present.[10]

It was our observation that most normal appendices were being filled during routine barium enema examinations performed in our Radiology Department. A review of the literature, however, did not reveal a large prospective study that demonstrated how frequently normal appendices are visualized.

Multiple roentgenographic signs have been described as either direct or indirect signs of appendicitis: sentinel loop in the right lower quadrant; small bowel obstruction; air-fluid level in the cecum; obliteration of the right psoas muscle shadow; abscess gas or appendiceal gas in the right lower quadrant; appendicolith; right lower quadrant soft tissue mass; and fluid in the right flank or in the peritoneal cavity.[1,4,5,12] The diagnosis of appendicitis, however, remains elusive clinically, as well as roentgenographically, in a significant number of instances.[6]

It is the purpose of this study to show that the normal appendix should be visualized on the barium enema examination an overwhelming majority of the time when the radiologist makes a definite effort to fill it, and that nonvisualization (except in specific instances which will be discussed later) may be considered additional strong evidence of diseased appendix.

MATERIAL AND METHOD

A prospective study was conducted on 855 consecutive patients having barium enema examinations at our institution. Of these, 310 patients had had prior appendectomies (a statistic in itself startling), and are, therefore, not included in the study. The examinations were performed by 1 of 5 attending radiologists or by a resident under his supervision. An attempt was made to visualize the appendix of each of the remaining 545 patients, ranging in age from 9 months to 92 years and comprised of 247 males and 298 females. Examination included fluoroscopy, 70 mm. and, when indicated, conventional spot films, preevacuation (Fig. 1*A*) (posteroanterior, angled, and lateral, plus any other view necessary for optimal demonstration of the appendix), and postevacuation studies (Fig. 1*B*). Because many were out-patients, and because of the large volume of other examinations performed in our department on a daily basis, delayed studies were obtained only when readily practicable. Fifteen additional patients were also excluded from the study because of nonfilling of the cecum due to either obstructing carcinoma, diverticulitis, or to some other condition.

RESULTS

Five patients had acute appendicitis proven surgically. There was, therefore, a group of 525 patients apparently without

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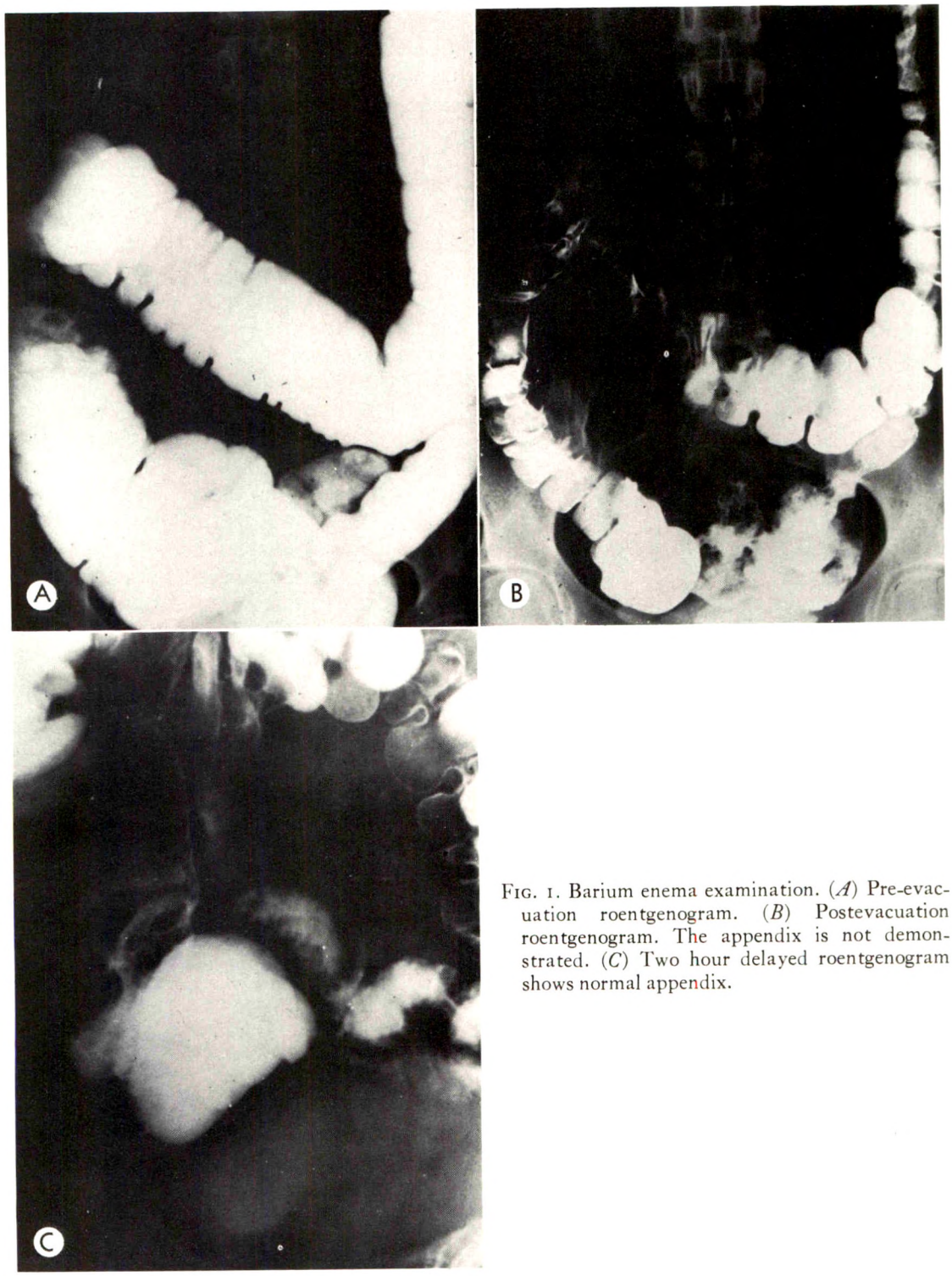


Fig. 1. Barium enema examination. (*A*) Pre-evacuation roentgenogram. (*B*) Postevacuation roentgenogram. The appendix is not demonstrated. (*C*) Two hour delayed roentgenogram shows normal appendix.

TABLE I

RESULTS OF DELAYED STUDIES

Delayed Roentgenograms	2 hr.	24 hr.
Complete visualization	10	5
Partial visualization	1	0
Nonvisualization	3*	2*
Total	14	7

* Includes 1 patient with appendicitis (2 total).

appendicitis. Of these, 430 (82 per cent) showed complete, 29 (5.5 per cent) showed partial, and 66 (12.5 per cent) showed no visualization of the appendix. Partial visualization does not exclude segmental disease,[10,11] although no patient in the study with appendicitis had either partial or complete visualization.

A delayed anteroposterior roentgenogram was obtained in 21 instances because of nonvisualization during the initial examination. Complete visualization was demonstrated in 15 cases. There was nonvisualization in 5 cases (2 with appendicitis proven surgically) and partial visualization in 1 case. Delayed roentgenograms, therefore, accounted for complete visualization of 15 of 19 normal appendices missed on the initial barium enema examination (Fig. 1*C*), with 411 of the 525 cases (78.3 per cent) being visualized immediately (Table I).



FIG. 2. There is nonfilling of the appendix in this patient with acute appendicitis. The appendix was also not demonstrated on the 2 hour delayed roentgenogram. There is questionable cecal indentation.

In 25 cases the barium enema examination was performed to rule out appendicitis: 19 showed complete visualization, thus excluding disease; none required surgery and all improved clinically, with 2 patients diagnosed roentgenographically as having terminal ileitis. In 6 cases there was nonfilling: 5 were found to have appendicitis at surgery (Fig. 2) and 1 improved clinically (no delayed roentgenogram was obtained and he was discharged from the hospital without surgery). Therefore, the correct diagnosis is assumed to have been rendered in 24 of the 25 cases when there was a clinical suspicion of appendicitis (Table II).

Thirteen patients were found to have either ulcerative colitis or Crohn's disease involving the cecum. Only 2 showed a completely visualized appendix, with partial visualization in 2 cases and nonvisualization in 9 cases. Total colectomy performed on 1 patient revealed "ulcerative colitis" involving the appendix (ulcerative appendicitis), which might explain the high rate of nonvisualization in these patients. One patient with a diagnosis of ischemic colitis had normal filling (Table III).

In 42 patients the cecum was found to be within the true pelvis (Fig. 3, *A* and *B*). Visualization was complete in 19, partial in 2, and absent in 21 (Table IV).

TABLE II

EXAMINATION PERFORMED TO EXCLUDE APPENDICITIS

Complete visualization	
No appendicitis	19
Nonvisualization	
No appendicitis	1
Nonvisualization	
Appendicitis	5
Total	25

Table III

ULCERATIVE COLITIS OR CROHN'S DISEASE INVOLVING CECUM

Complete visualization	2
Partial visualization	2
Nonvisualization	9
Total	13

Table IV

PELVIC CECUM

Complete visualization	19
Partial visualization	2
Nonvisualization	21
Total	42

In 1 instance of nonvisualization and in 2 instances of partial visualization, the pathologic specimens revealed obliterative fibrosis (elective surgery for reasons other than appendicitis had been performed). We could find no mention of its true incidence in our review of the literature, although it is probably no more than 5 per cent.

DISCUSSION

A prospective study of 530 consecutive barium enema examinations on patients without prior appendectomies is presented. Over-all, 82 per cent demonstrated complete filling and visualization, thus virtually excluding appendiceal disease. There was a high rate of nonvisualization when the cecum was found to be within the true pelvis or when either ulcerative colitis or Crohn's disease involving the right side of the colon was present. If the 55 patients in these categories are excluded, comprising 31 of the 66 cases of nonvisualization in patients with appendicitis, the statistics are

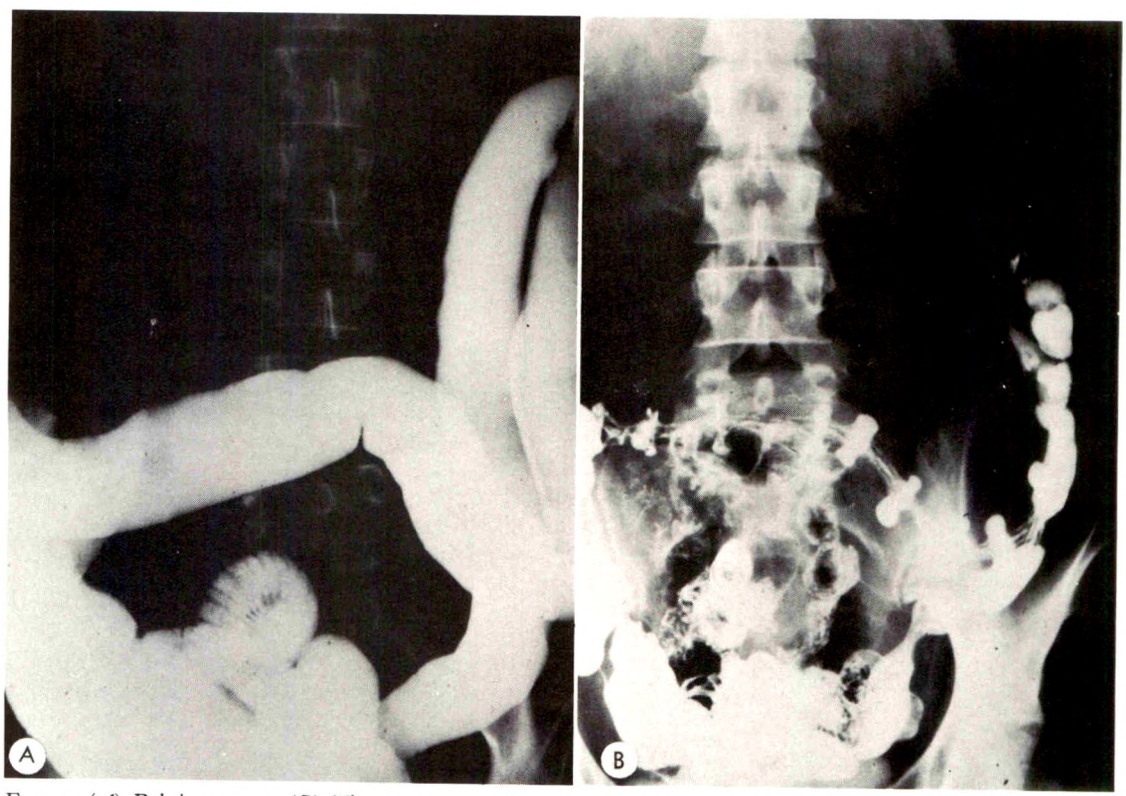


Fig. 3. (*A*) Pelvic cecum. (*B*) The postevacuation study still does not demonstrate the appendix in this asymptomatic patient. Note the overlap of the cecum by the sigmoid colon and terminal ileum.

TABLE V

OVER-ALL RESULTS

	Normal Appendix		Minus Instances of Pelvic Cecum and Colitis	
	No.	Per Cent	No.	Per Cent
Complete visualization	430	82*	410	87†
Partial visualization	29	5.5	25	5.5
Nonvisualization	66	12.5	35	7.5
Total	525		470	

* Includes 15 of 19 patients with delayed roentgenograms.
† Includes 14 of 17 patients with delayed roentgenograms.

improved considerably—83 per cent are visualized immediately and 87 per cent with the delayed roentgenograms (Table v). These results compare favorably to those of other diagnostic roentgenographic procedures such as oral cholecystography, in which 85 per cent of normal gallbladders show satisfactory visualization during the initial examination with 15 per cent requiring a repeat study because of absent or poor visualization.[2,3] The value of the delayed roentgenogram is demonstrated by the 21 cases in which they were obtained; an additional three-fourths of the appendices not visualized immediately were completely filled.

We believe that a barium enema examination is indicated in any patient suspected of having acute appendicitis, but whose clinical diagnosis is not definite or is in doubt. We were correct in 24 of 25 patients suspected of possibly having appendicitis; however, the examination can be considered incomplete in the remaining patient since no delayed roentgenogram had been obtained. No complication occurred in any of our patients related to the examination. This substantiates reports in the literature which indicate that retrograde low pressure filling of the colon has not been shown to precipitate any complication in the presence of acute appendicitis.[8–10]

CONCLUSION

The authors demonstrated an over-all 82 per cent incidence of complete filling and visualization of normal appendices with the barium enema examination. Since an additional 15 of 19 normal appendices (not seen immediately) were demonstrated with delayed roentgenograms, these studies should be included whenever possible to complete the examination. Under these circumstances, completely filled normal appendices should be demonstrated well over 90 to 95 per cent of the time.

Therefore, it can be seen that nonfilling of the appendix is strong corroborative evidence of appendicitis. When either the presence of a pelvic cecum or ulcerative disease of the colon (or any other condition which obscures demonstration or prevents filling of the cecum) is shown, nonvisualization cannot be considered significant, although complete filling would still be a valuable finding in ruling out disease.

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REFERENCES

1. BENEVENTANO, T. C., SCHEIN, C. J., and JACOBSON, H. G. Roentgen aspects of some appendiceal abnormalities. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 344–360.
2. BERK, R. N. Consecutive dose phenomenon in oral cholecystography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *110*, 230–234.
3. BERK, R. N. Editorial. Problem of impaired first-dose visualization of gallbladder. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 186–188.
4. FIGIEL, K. S., and FIGIEL, S. J. Barium examination of cecum in appendicitis. *Acta radiol.*, 1962, *57*, 469–480.
5. FRIMANN-DAHL, J. Roentgen Examinations in Acute Abdominal Diseases. Charles C Thomas, Publisher, Springfield, Ill., 1951, pp. 242.
6. GRAHAM, A. D., and JACOBSON, H. F. Incidence of radiographic findings in acute appendicitis

compared to 200 normal abdomens. *Military Med.*, 1966, *131*, 272–276.

7. Mayson, P. B., Jr., and Rosenthal, S. J. Roentgen findings in delayed diagnosis of appendicitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 347–350.
8. Noveroske, R. J. Intracolonic pressures during barium enema examinations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *91*, 852–863.
9. Seaman, W. B., and Wells, J. Complications of barium enema. *Gastroenterology*, 1965, *48*, 728–737.
10. Soter, C. S. Contribution of radiologist to diagnosis of acute appendiceal disease. *Med. Radiog. & Photog.*, 1969, *45*, 2–15.
11. Soter, C. S. Use of barium in diagnosis of acute appendiceal disease. *Clin. Radiol.*, 1968, *19*, 410–415.
12. Soteropoulos, C., and Gilmore, J. H. Roentgen diagnosis of acute appendicitis. *Radiology*, 1958, *71*, 246–256.

ORAL EXAMINATION OF THE COLON*

"THE COLONIC COCKTAIL"

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THE radiologic study of the colon has been traditionally performed by means of a barium enema. Double contrast techniques, in particular, have proven to be valuable in the evaluation of colonic pathology.[10,11] However, barium enema examination may, in certain instances, not be feasible and/or may be contraindicated.[1]

An alternate radiologic method of colonic examination which is intended to complement an unsuccessful or incomplete barium enema study is described in this paper. The method consists of oral administration of a special contrast mixture containing barium sulfate, sorbitol and water soluble contrast medium.

In the past, follow-through examinations of the colon were unsatisfactory, since small bowel transit time associated with the commonly used barium mixtures was extremely variable. By the time the mixture finally reached the colon it had often dried, fragmented and generally failed to produce an adequate mucosal coating of the colon. Most of these shortcomings have been eliminated with the new technique herein described and a reliable, good quality visualization of the entire colon has been obtained in the large majority of cases.

METHOD

The usual preparation for conventional barium enema examination is adhered to. A fasting state is additionally required for the upper gastrointestinal series.

A preliminary roentgenogram of the abdomen is necessary in order to ascertain adequacy of preparation as well as to detect or document large bowel obstruction.

A routine gastrointestinal series may be performed if not done previously. During, or at the end of fluoroscopy, 2 full cups of the "colonic cocktail," *i.e.*, a special barium preparation* containing 3.5 per cent sorbitol mixed with 40 ml. of water soluble contrast medium,† and further diluted to a 43 per cent w/w suspension, are given with a third cup administered prior to overhead roentgenography.

Roentgenograms of the abdomen at 30 and 60 minutes may be obtained in order to provide a supplementary screening study of the small bowel (Fig. 1).

The patient is requested to return 4 hours after completion of the upper gastrointestinal series with instructions to refrain from moving the bowels. Liberal intake of fluids is allowed but no solid food permitted. At 4 hours, the entire colon is usually filled with fluid barium, and the stomach and small bowel are often almost empty (Fig. 2, Table 1). A double contrast study is then performed by means of gas insufflation through the rectum. Multiple appropriate views are taken. Erect (Fig. 3), anteroposterior (Fig. 4), both obliques, lateral and tilted views of the rectum may be utilized as required.

RESULTS

Over 100 oral examinations of the colon were done, usually in association with routine upper gastrointestinal series. A small number of patients had had double contrast studies of the stomach.[14] In more than 90 per cent of the cases, the barium had reached the cecum at 1 hour. The en-

* "Ultra-R" barium containing 3.5 per cent sorbitol and special suspending agents supplied in "EZ-GI" cups by EZ-Em Co.

† "Oral Hypaque Liquid" or "Gastrografin."

* From The City Hospital Center at Elmhurst, Mount Sinai Hospital Services, Mount Sinai School of Medicine, City University of New York, New York.



FIG. 1. One hour roentgenogram. Barium reaches the colon in 1 hour in over 90 per cent of patients. The entire colon is filled in this patient—a frequent finding.

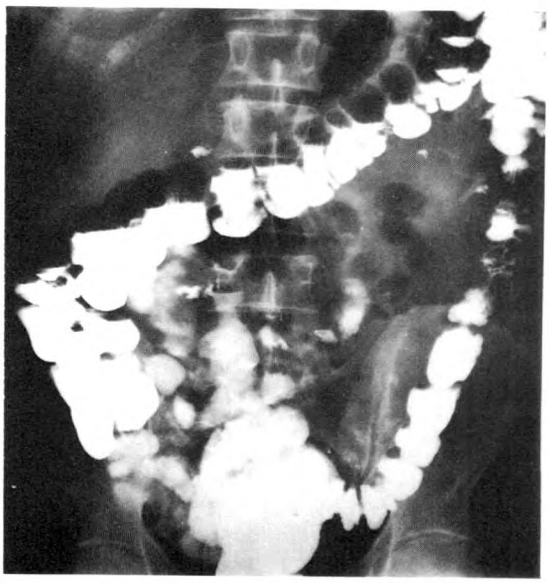


FIG. 2. Four hour erect roentgenogram. The barium remains in liquid state and fills the entire bowel. Note minimal residual in the stomach and small bowel.

tire colon was often completely filled at 1 hour (Fig. 1).

The entire colon was visualized (Fig. 2) in 89 of 100 patients at 4 hours (Table 1). Only in 11 patients did the entire colon fail to fill. Small bowel transit of the barium was not completed at 4 hours in only 1 patient.

The small bowel showed minimal or no barium residual in 74 out of 100 patients. Significant barium residual in the small bowel was seen in only 26 patients.

The stomach showed significant barium residual in only 28 out of 100 patients. In 72 patients, there was no or only minimal residual of barium in the stomach (Table 1).

In many of those patients whose colon was not completely visualized at 4 hours, it was discovered that they had ignored our instructions and evacuated prior to the 4 hour roentgenogram. The urge to empty the bowel was quite frequent. If evacuation occurred immediately prior to the 4 hour roentgenogram, an excellent mucosal coating of the entire colon was often obtained (Fig. 5).

In some patients in whom excessive small bowel residual barium was noted at 4 hours (Table 1), repeat examination at 5 hours showed further significant emptying of the

TABLE I

RESULTS OF 100 FOLLOW-THROUGH STUDIES
4 HOUR ROENTGENOGRAMS

Colon Filled		Small Bowel Residual		Gastric Residual	
Entirely	Partially	Significant	Minimal—None	Significant	Minimal—None
89	11	26	74	28	72

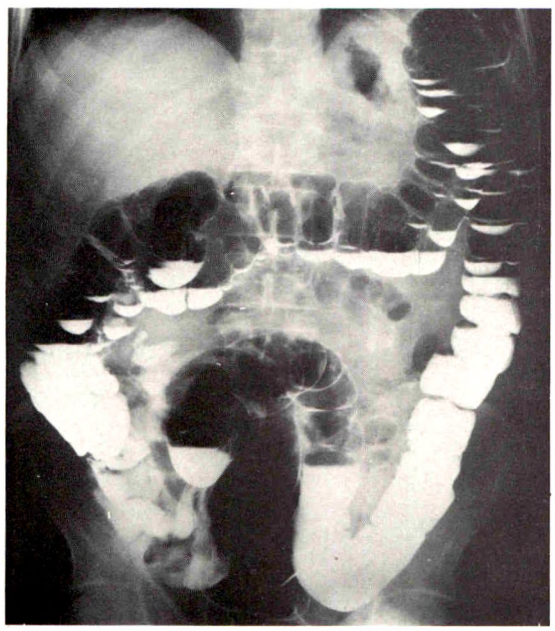


FIG. 3. Erect roentgenogram. Double contrast phase of the oral examination of the colon is easily obtained at 4 hours solely by rectal gas insufflation. Note air-fluid levels confirming liquid state of barium suspension.

small bowel as well as unobscured view of the colon.

In practically all patients the barium remained in a liquid state and an erect view would invariably show intracolonic air-fluid levels (Fig. 2; and 3). There was often a significant amount of gas in the colon at 4 hours (Fig. 2).

A good double contrast study of the colon was obtained by simply insufflating the colon with gas *via* the rectum, since the entire colon was already filled with liquid barium. The quality of these studies often approached those obtained with conventional double contrast barium enema techniques (Fig. 3; 4; and 6*B*).

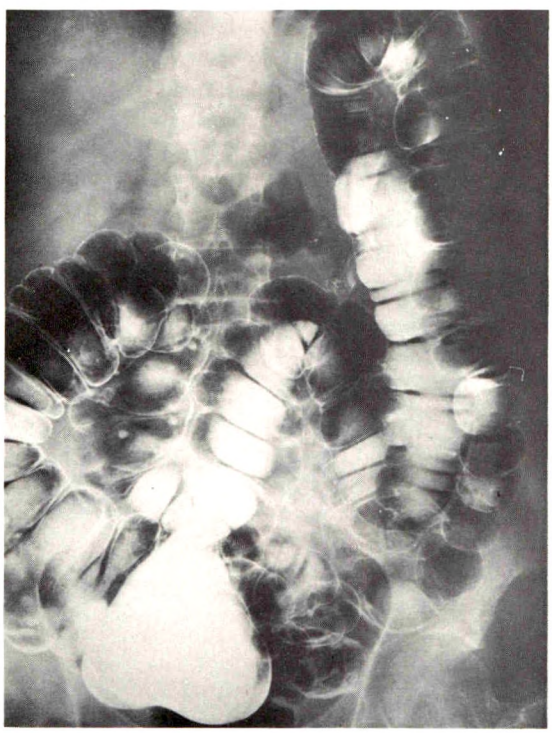


FIG. 4. Oral colonic examination. Double contrast phase 4 hours after oral administration of barium. Note that the quality of the study approaches that obtained with conventional double contrast barium enemas.

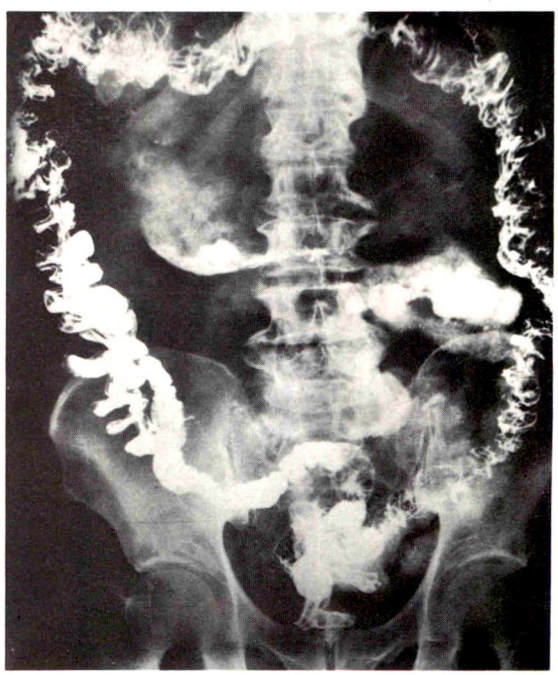


FIG. 5. Postevacuation roentgenogram obtained with the oral method. Note excellent mucosal pattern of the colon.

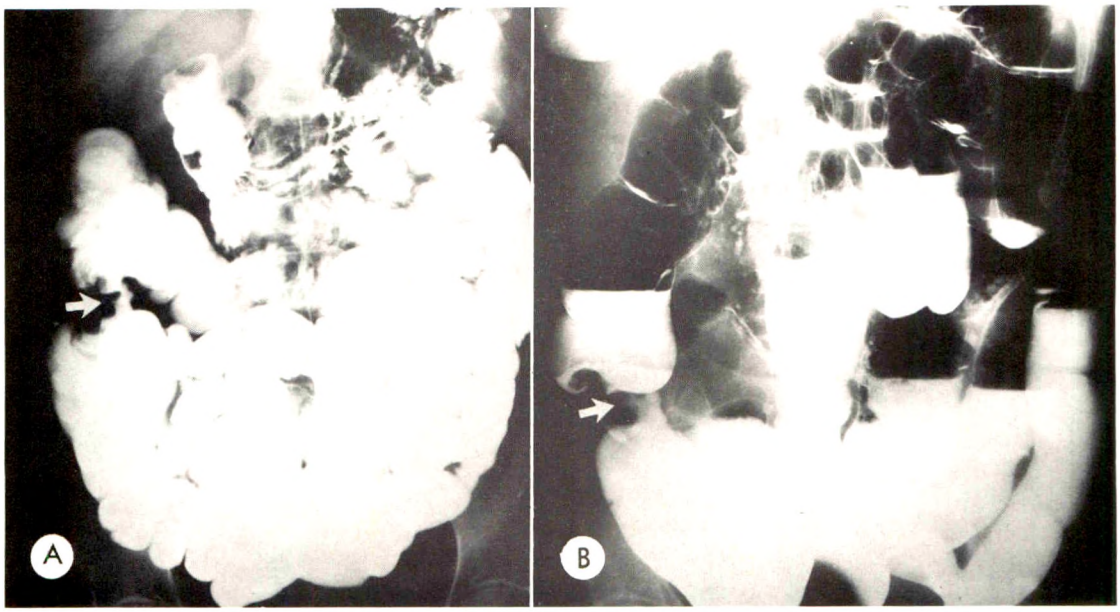


Fig. 6. Carcinoma of the ascending colon. Oral study. (*A*) The lesion is visualized at 1 hour (arrow). (*B*) It is clearly outlined at 4 hours, following rectal gas insufflation (arrow).

DISCUSSION

In order to accomplish a reliable follow-through examination of the colon 2 prerequisites had to be satisfied. The first prerequisite was to use a barium mixture which would remain in the liquid state for several hours after having reached the colon. The second prerequisite was to accelerate the small bowel transit time in order to promptly fill the entire large bowel and to empty the stomach and small bowel in order to afford an unobstructed view of the colon.

A special barium mixture containing 3.5 per cent sorbitol was found to fulfill the first prerequisite. Sorbitol was selected since it had been shown that it improves the coating quality of the barium preparations, reduces its viscosity if added to sodium citrate[2] and has accelerating influence on small bowel transit.[15,16] The accelerating effect of sorbitol on the small bowel transit may be due to its hypertonic action since it is not absorbed from man's gastrointestinal tract.[15,16] It therefore has a tendency to draw body fluids into the colon.

The second prerequisite, reliable acceleration of the small bowel transit, was accomplished with the addition of 40 ml. of water soluble contrast material to each cup of barium suspension, the patient being given a total of 3 cups of barium during the examination. There was no appreciable change in the appearance or coating ability of the barium preparation. The bitter taste of the water soluble contrast medium was almost completely concealed by the flavored barium used.

Water soluble contrast medium was shown to have a shorter transit time than barium in animal experiments.[8] This was thought to be due to its hyperosmolarity and its action as a saline type of cathartic.[8] The addition of 30 ml. of water soluble contrast medium to the barium preparation was also shown[3] to significantly accelerate the small bowel transit time. Nevertheless, it was also noted that even when only 10 ml. of water soluble contrast medium was used and the mixture rendered isotonic, the transit time was still significantly accelerated.[3] In this study, 40 ml. was required to accomplish the best results (Table 1).

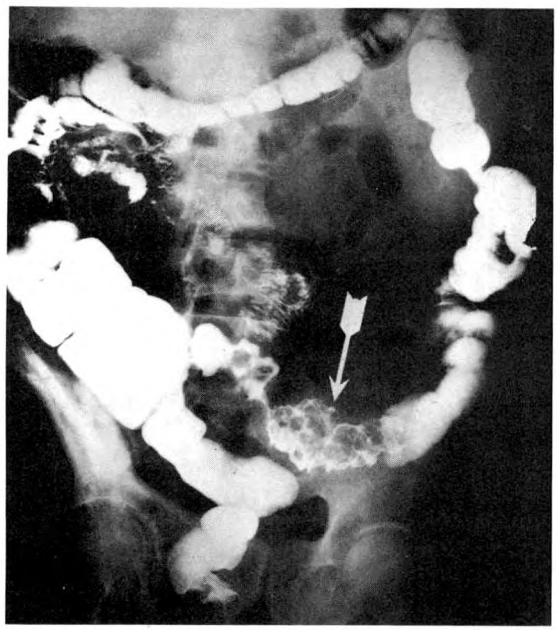


FIG. 7. Oral examination of the colon confirms the presence of a sigmoid villous adenocarcinoma (arrow) diagnosed by previous barium enema without need to repeat it.

Other agents which have been shown to possess an accelerating effect on the small bowel transit time were also considered. Among them were the subcutaneous administration of neostigmine,[6] intravenous injection of cholecystokinin,[9] and oral administration of metoclopramide.[5] The combination of a special barium sorbitol formulation and a water soluble contrast agent was selected, since these constituents were readily available and precluded the need to use any parenteral or oral medication.

Preliminary cleansing of the colon was required, since it was demonstrated[7] that it aided rapid small bowel barium transit. In addition, it improved the quality of the follow-through study of the colon.

INDICATIONS AND ADVANTAGES OF THE ORAL METHOD

The most important indication for the oral method is to provide an alternate means of examination of the colon in weak and elderly patients in whom previous contrast enema studies were incomplete or unsuccessful (Fig. 8, *A* and *B*; 9; and 10) despite the use of improved rectal catheters and balloons.[12]

The oral examination of the colon also served as a highly useful and expeditious confirmatory study of colonic pathology (Fig. 6, *A* and *B*; 7; 9; 10; and 11) following a conventional contrast enema study. The confirmatory oral colonic study may be combined with and will not cause the post-

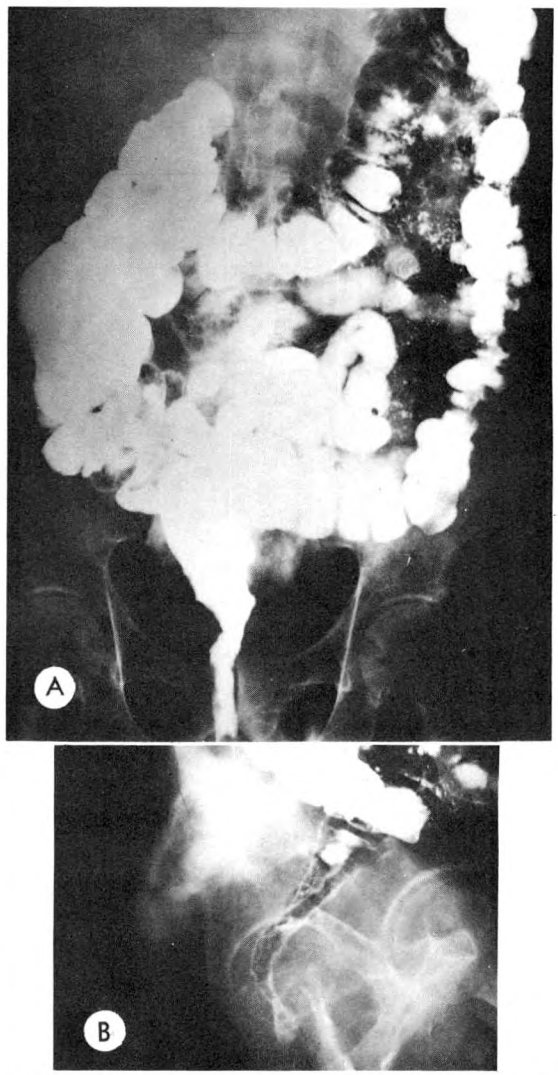


FIG. 8. Oral examination of the colon. Note marked rectal narrowing by invasive prostatic carcinoma. Previous barium enema examination was unsuccessful due to inability to insert the rectal catheter. (*A*) The entire colon is visualized at 4 hours. (*B*) Note osteoblastic sacral metastases.

ponement of any prescheduled upper gastrointestinal series.

Oral examinations using the above described method should be considered where contraindications for the conventional barium enema study exist, *i.e.*, diverticulitis, a friable but nonobstructive rectal lesion, or severely inflamed hemorrhoids and elderly patients with severe heart disease.[1]

It may be more convenient to perform in colostomy patients, particularly those with tight colostomy apertures. The introduction of special colostomy catheters[13] is, therefore, obviated.

Meticulous colonic preparation was found not to be as crucial in the oral method as in the conventional barium enema examination. This was due to the fact that small amounts of retained feces, if present, often gradually mixed evenly with the liquid barium progressing from above without causing disturbing shadows. Retained barium from a previous study also readily mixed with the oral barium and did not, as a rule, interfere with the study.



Fig. 9. Oral exmination of the colon. A previous barium enema study suggested a cecal lesion but was discontinued due to inability of the patient to retain the enema. The cecal cancer is clearly seen (arrow).

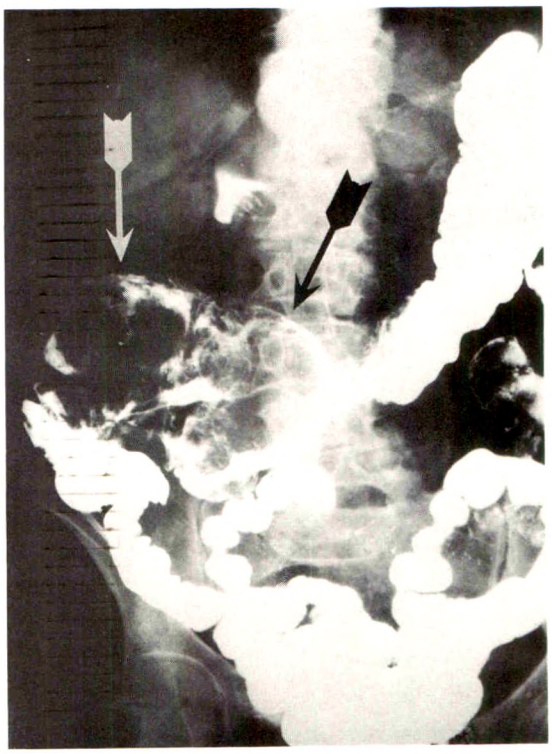


Fig. 10. Oral examination of the colon. Note 2 large villous adenocarcinomas in the transverse colon (arrows). Routine barium enema study failed to disclose the second separate lesion due to poor retention.

CONTRAINDICATIONS AND DISADVANTAGES

Oral examinations are not always reliable since they may not show the colon in its entirety at all times and they should not supplant conventional barium enemas as the examination of choice for suspected colonic pathology.

The oral method is contraindicated when an obstructive lesion of the colon is suspected, since retained barium proximal to the lesion may aggravate an existing obstruction. This pitfall can be avoided by obtaining routine preliminary roentgenograms of the abdomen on all cases and/or by clinical evaluation of each patient prior to the study.

The oral method may be contraindicated when the patient is dehydrated since the



FIG. 11. Oral examination of the colon. An adenomatous polyp in the descending colon, which was first suspected on the basis of a previous conventional barium enema study, is confirmed on the oral examination (arrow).

contrast medium preparation used is hypertonic.[3] This is especially true in infants and young children.[4]

The oral method does not have the benefit of controlled fluoroscopic monitoring during colonic filling and requires 4 hours from the time of interval barium administration to the completion of the study.

SUMMARY

A new method of radiologic colonic examination is described which consists of the oral administration of a "Colonic Cocktail" which consists of a special contrast preparation containing barium sulfate, sorbitol and water soluble contrast medium.

The chief characteristics of this barium preparation are rapid progression through the small bowel and the retention of its liquid state within the colon for many hours. This will permit a good double contrast examination of the colon in most cases, solely by rectal gas insufflation and appropriate filming 4 hours after the oral administration of barium.

The merits and indications of the method are illustrated and discussed.

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REFERENCES

1. EASTWOOD, G. L. ECG abnormalities associated with barium enema. *J.A.M.A.*, 1972, *219*, 719–721.
2. EMBRING, G., and MATTSSON, O. Barium contrast agents. *Acta radiol.* (Diag.), 1968, *7*, 245–256.
3. GOLDSTEIN, H. M., POOLE, G. J., ROSENQUIST, C. J., FRIEDLAND, G. W., and ZBORALSKE, E. F. Comparison of methods for acceleration of small intestinal radiographic examination. *Radiology*, 1971, *98*, 519–423.
4. HARRIS, P. D., NEUHAUSER, E. B. D., and GERTH, R. Osmotic effect of water soluble contrast media on circulating plasma volume. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 694–698.
5. KREEL, L. Use of oral metoclopramide in barium meal and follow-through examination. *Brit. J. Radiol.*, 1970, *43*, 31–35.
6. MARGULIS, A. R., and MANDELSTAM, P. Use of parenteral neostigmine in roentgen study of small bowel. *Radiology*, 1961, *76*, 223–229.
7. NICE, C. M. Roentgenographic pattern and motility in small bowel studies. *Radiology*, 1963, *80*, 39–45.
8. NOONAN, C. D., and MARGULIS, A. R. Small bowel transit time of water soluble iodinated contrast medium and barium sulfate in cats with simulated surgical acute abdomen. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *110*, 334–337.
9. PARKER, J. G., and BENEVENTANO, T. C. Acceleration of small bowel contrast study by cholecystokinin. *Gastroenterology*, 1970, *58*, 679–684.
10. POCHACZEVSKY, R., SHERMAN, R. S., and MEYERS, P. H. Disposable kit for barium enemas. *Radiology*, 1961, *77*, 831–833.
11. POCHACZEVSKY, R., and SHERMAN, R. S. New technique for roentgenologic examination of colon. AM. J. ROENTGENOL., RAD. THERAPY &

Nuclear Med., 1963, *89*, 787–796.
12. Pochaczevsky, R., and Meyers, P. H. Safe, self-retaining, disposable rectal catheter for barium enemas with sealed insufflation bulb and movable external retention cushion. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 359–361.
13. Pochaczevsky, R., and Meyers, P. H. New, disposable catheter for selective guided barium enemas: contrast examinations in patients with colostomies, rectal lesions and fistulas and for pediatric colon studies. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 392–395.
14. Pochaczevsky, R. Bubbly barium: carbonated cocktail for double-contrast examination of stomach. *Radiology*, 1973, *107*, 465–466.
15. Porcher, P., and Caroli, J. Un accélerateur inattendu du transit intestinal (grêle et côlon). *Arch. mal. app. digest.*, 1957, *46*, 663–665.
16. Reid-Smith, A. Use of sorbitol in investigation of small bowel. *J. Canad. A. Radiologists*, 1970, *21*, 35–38.

THE WATER DOUBLE CONTRAST BARIUM ENEMA STUDY*

A NEW METHOD OF ROENTGEN EXAMINATION OF THE COLON

By RUBEM PÓCHACZEVSKY, M.D.
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DOUBLE contrast examinations of the colon usually employ air or carbon dioxide[10,18] as the contrast agent, while tannic acid has been traditionally employed to enhance colonic evacuation.

A new method of double contrast colonic examination substituting water instillation for gas insufflation has been developed which uniformly yields reliable postevacuation roentgenograms without the use of tannic acid.

APPARATUS

The apparatus consists of a disposable enema kit*[9,10] composed of 2 enema bags connected by means of a "Y" shaped adaptor (Fig. 1). One enema bag is prepackaged with 300 gm. of barium sulfate. The other is empty. Both are then filled with water through individual self-sealing apertures. Approximately 300 ml. of water is added to the bag containing prepackaged barium sulfate, which is then kneaded until the barium suspension is thoroughly mixed. A 50 per cent w/w suspension is thus obtained. The other bag is filled with 2,000 ml. of tap water. The flow from each bag is controlled by clamps.

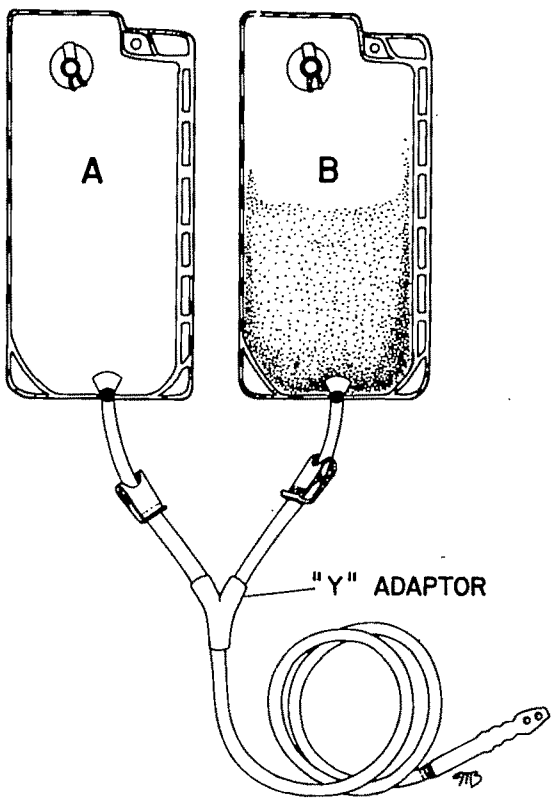


FIG. 1. Disposable double bag enema kit. Both bags can be filled with water through self-sealing apertures. Bag "A" is empty and is filled with 2,000 ml. of tap water prior to the examination. Bag "B" is prepackaged with 300 gm. of barium sulfate. The barium suspension is prepared by adding 300 ml. of water to bag "B," followed by kneading and mixing. The bags are connected by means of a "Y" shaped adaptor. The flow from each bag is controlled by clamps.

TECHNIQUE

The examination is initiated by releasing the clamp of the barium filled bag (Fig. 1) and allowing the colon to fill up to the splenic flexure (Fig. 2*A*). Appropriate fluoroscopic spot roentgenograms and overhead roentgenograms are then taken. Complete colonic filling is not necessary in this initial step.

The examination is continued by releasing the second clamp leading to the water filled bag. The fluoroscopist then appreciates a gradual decrease in the density of the left hemicolon (Fig. 2, *B* and *C*), as the barium column progresses to outline the entire right hemicolon. Thus, at the early

* Manufactured by EZ-EM Co.

* From The City Hospital Center at Elmhurst, The Mount Sinai Hospital Services, Mount Sinai School of Medicine, City University of New York.

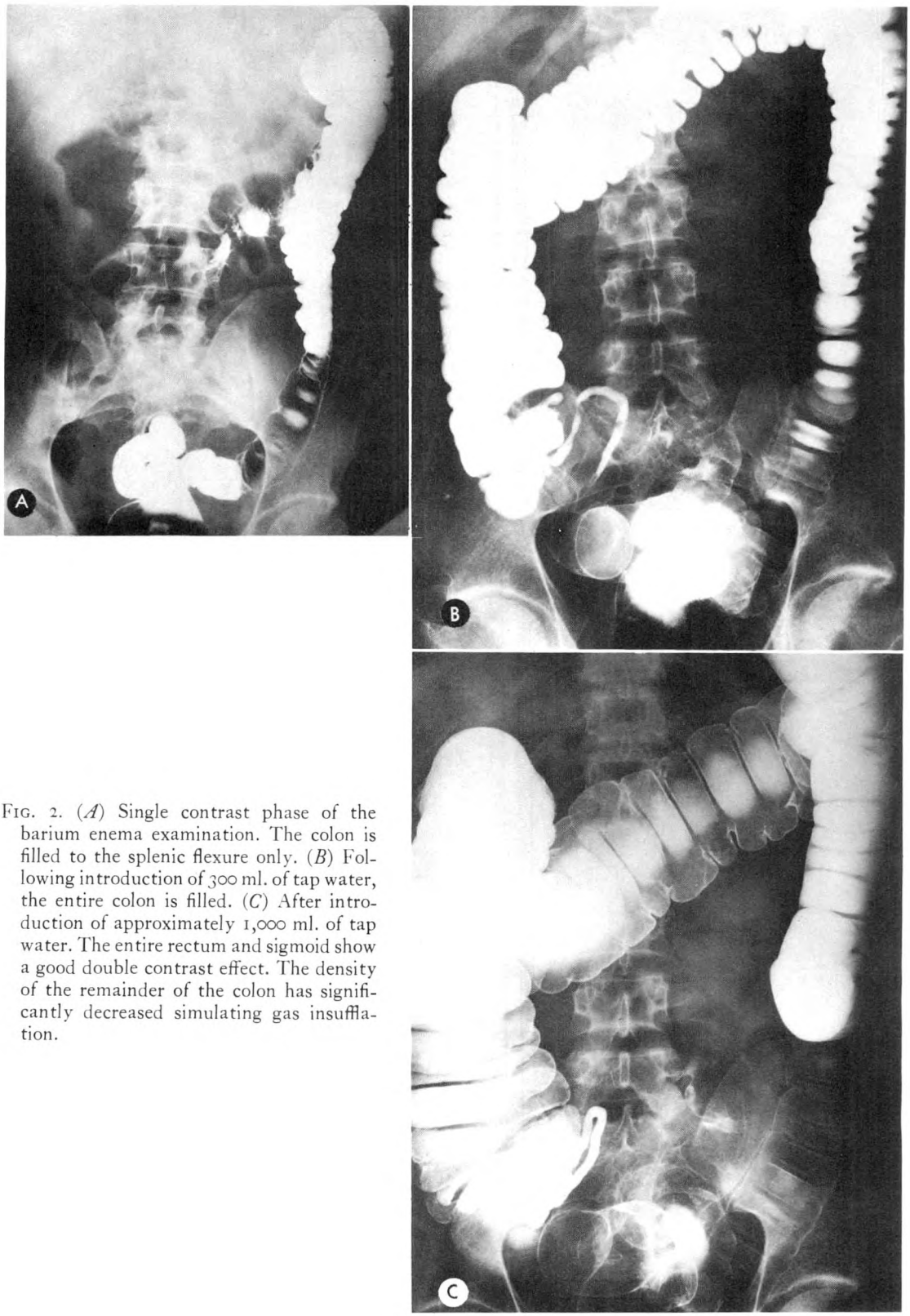


Fig. 2. (*A*) Single contrast phase of the barium enema examination. The colon is filled to the splenic flexure only. (*B*) Following introduction of 300 ml. of tap water, the entire colon is filled. (*C*) After introduction of approximately 1,000 ml. of tap water. The entire rectum and sigmoid show a good double contrast effect. The density of the remainder of the colon has significantly decreased simulating gas insufflation.

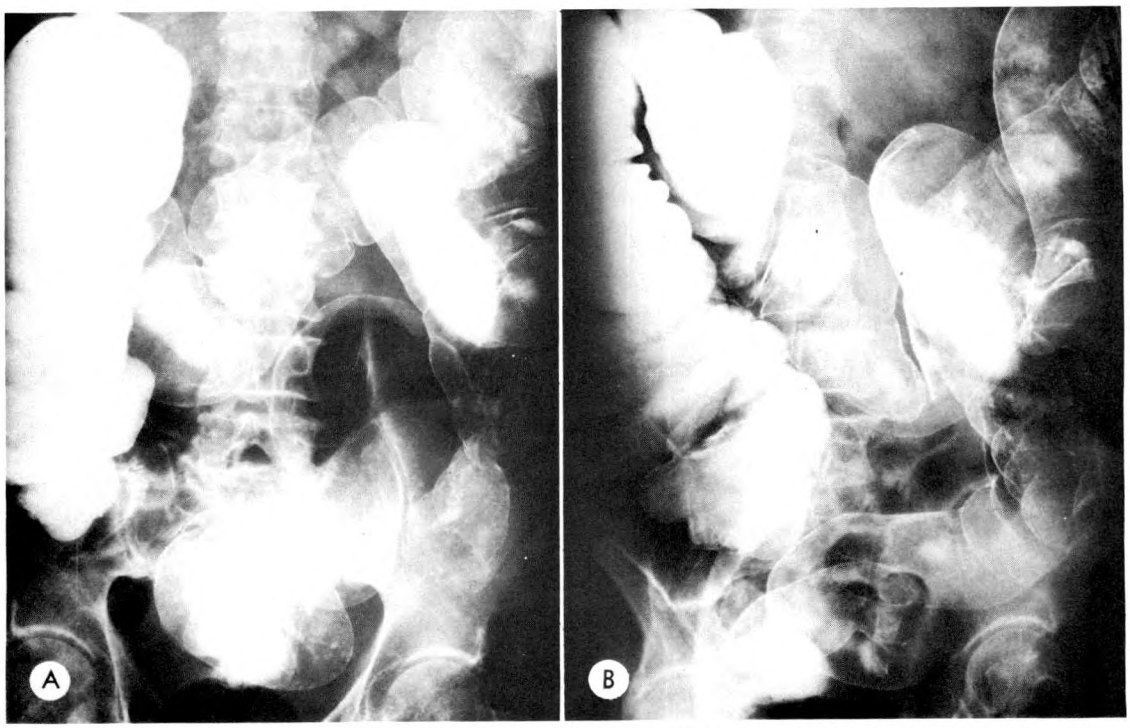


FIG. 3. (*A*) Anteroposterior and (*B*) oblique views. Water double contrast of the entire colon after introduction of approximately 1,500 ml. of tap water. Note that a "see-through" effect is obtained despite the redundant colonic loops.

phase of the study, a simultaneous single contrast of the proximal colon and double contrast of the distal colon will be obtained (Fig. 2*C*). If the examination is continued, a steady decrease in density will allow for a double contrast study of the entire colon (Fig. 3, *A* and *B*). After appropriate spot and overhead roentgenograms are taken, the colon is syphoned by lowering the bag and allowing the barium and water mixture to reflux into it.[9,10] After completion of emptying, usually within 1 minute, a postevacuation roentgenogram is taken (Fig. 4, *A* and *B*), and the examination is completed.

RESULTS

One hundred cases were studied by this method. Depending on the clinical problem or the radiologist's preference, either a complete or a selective double contrast study of the colon was obtained using the double bag water contrast technique. Patients were selected at random. A variety of pathologic processes were clearly demonstrated including carcinomas (Fig. 5, *A* and *B*; 6; and 7), polyps, ulcerative colitis, diverticulosis (Fig. 8, *A* and *B*), diverticulitis and pericolonic abscess (Fig. 9, *A* and *B*).

Postevacuation roentgenograms obtained by the syphoning technique[9,10] showed adequate emptying of more than 90 per cent of the colon (Fig. 4, *A* and *B*; and 8*B*) in 41 of 100 patients (Table 1). Adequate emptying of 70 to 90 per cent of the colon was noted in 24 of 100 patients (Fig. 5*B*). Thus, more than 70 per cent colonic emptying

»»→

FIG. 5. Polypoid constricting carcinoma of the ascending colon. (*A*) Water double contrast roentgenogram. The lesion is clearly demonstrated (arrow). The sigmoid allows a "see-through" effect due to the water contrast and does not obscure the cecum. (*B*) Postevacuation roentgenogram following passive syphoning. Note good mucosal coating of the colon. The lesion in the ascending colon is well delineated (arrow).

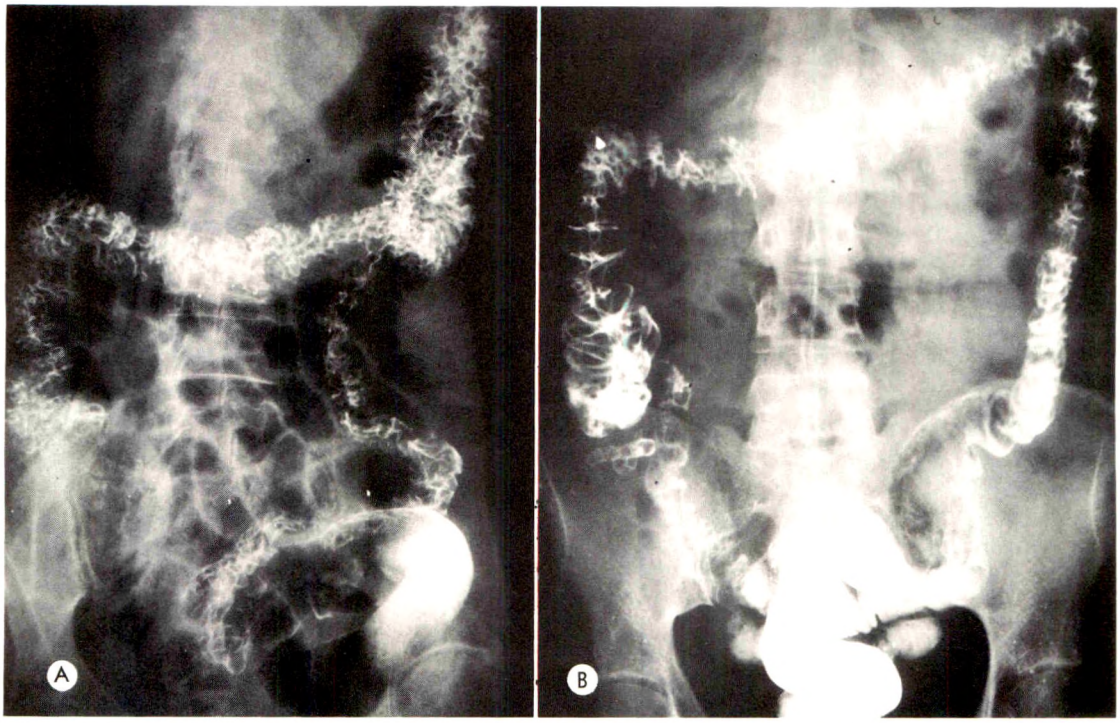
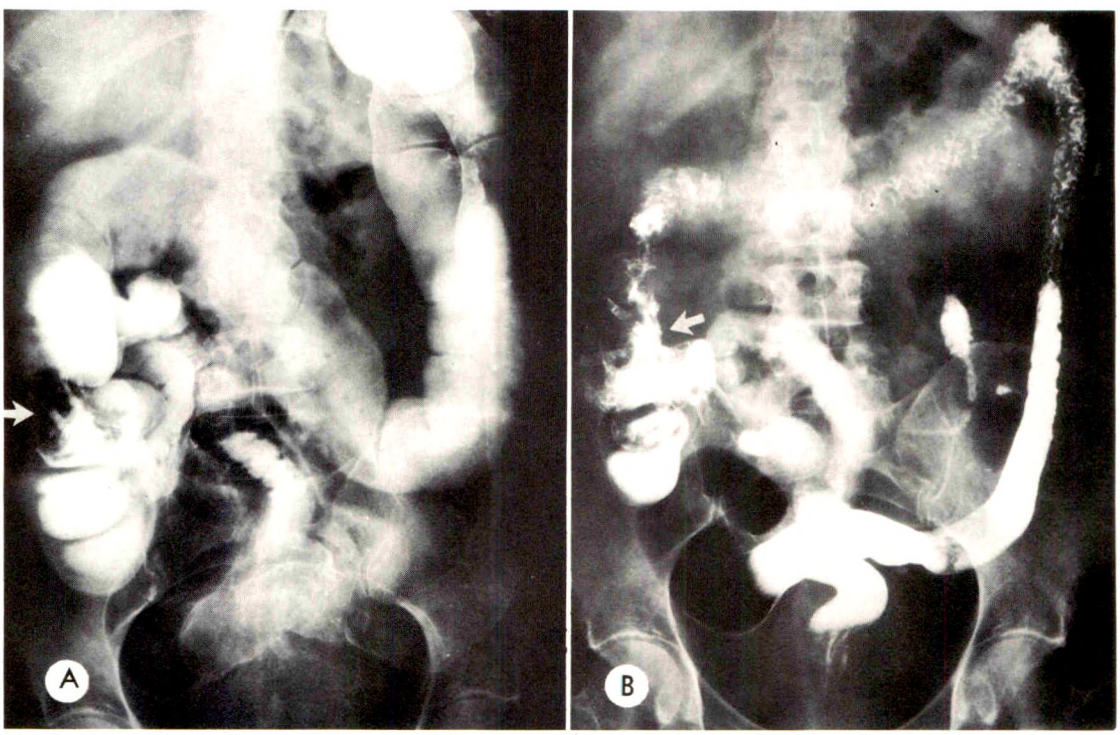


Fig. 4. (*A* and *B*) Postevacuation roentgenograms of the colon in 2 patients following passive syphoning. These roentgenograms were taken *after* completion of the double contrast phase of the study. Note more than 90 per cent colonic emptying as well as the excellent mucosal coating.



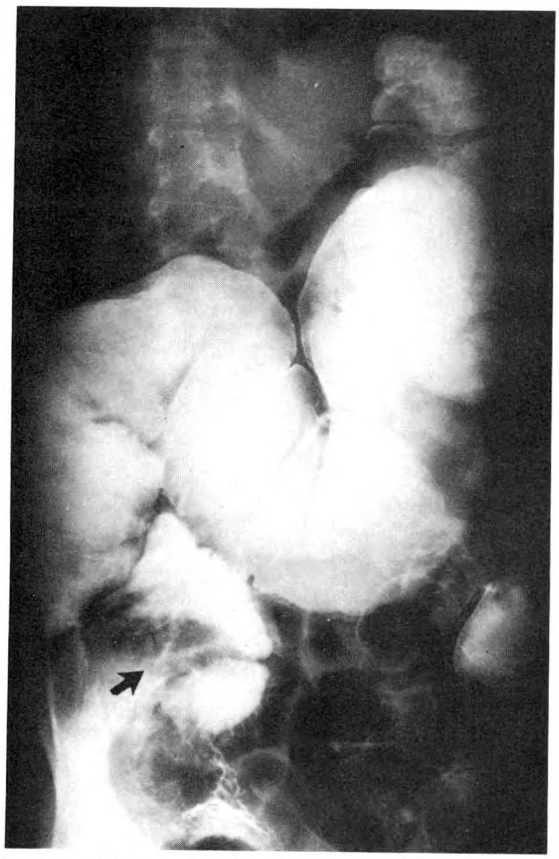


FIG. 6. Water double contrast study. Polypoid carcinoma of the cecum (arrow). The sigmoid is rendered lucent by the water contrast and, although it overlaps the cecum in this view, does not obscure the lesion.

was noted in almost two-thirds of the cases. Adequate emptying of 50 to 70 per cent of the full length of the displayed colon was seen in 12 of 100 patients. Less than 50 per cent emptying of the colon (Fig. 9*B*) was noted in only 23 of 100 patients, or less than one-fourth of the cases. However, a good double contrast effect was often obtained and excellent colonic mucosal coating was noted even in those segments which were not entirely emptied on the postevacuation roentgenograms (Fig. 9*B*). Conversely, complete colonic evacuation such as would prevent adequate visualization of the coated mucosa was almost never observed with the syphoning method.

DISCUSSION

The use of double cans, *i.e.*, one containing water and the other a barium suspension, for obtaining variable colonic contrast has not been utilized in the United States, but has been described in the foreign literature.[1,4,14,15] In the latter reports, a water suspension of methylcellulose was used for selective visualization of the colonic segments.[4,15] Methylcellulose was added for its reputed antispasmodic effect,[1] and enhancement of colonic coating.[14]

The present technique differs from those previously described in that, in addition to a controlled variable contrast study, a complete double contrast examination of the colon may be obtained. This is accomplished by means of an adaptation of the author's previously described closed system, one-stage technique utilizing prepackaged disposable kits.[9,10] The single contrast initial phase of the currently reported method yields an opaque column of barium of the desired density (Fig. 2, *A* and *B*). An immediate double contrast study is obtained by simply allowing water to enter the colon. Despite the introduction of water, mucosal coating persists and the resultant study may simulate a routine

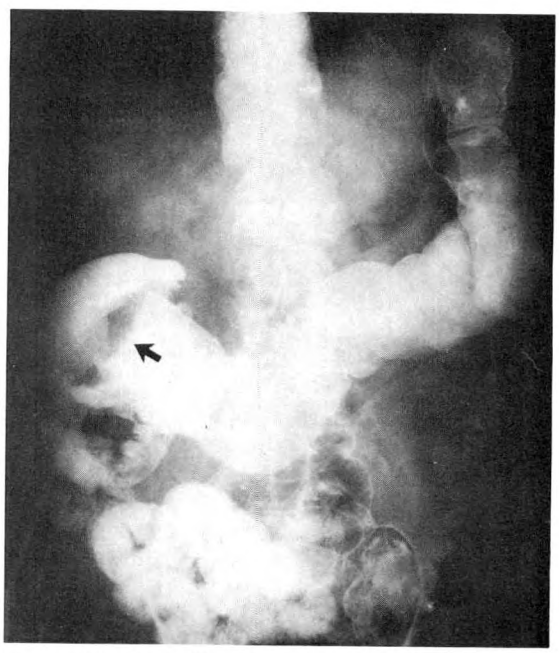


FIG. 7. Water double contrast study. Note polypoid carcinoma of hepatic flexure (arrow).

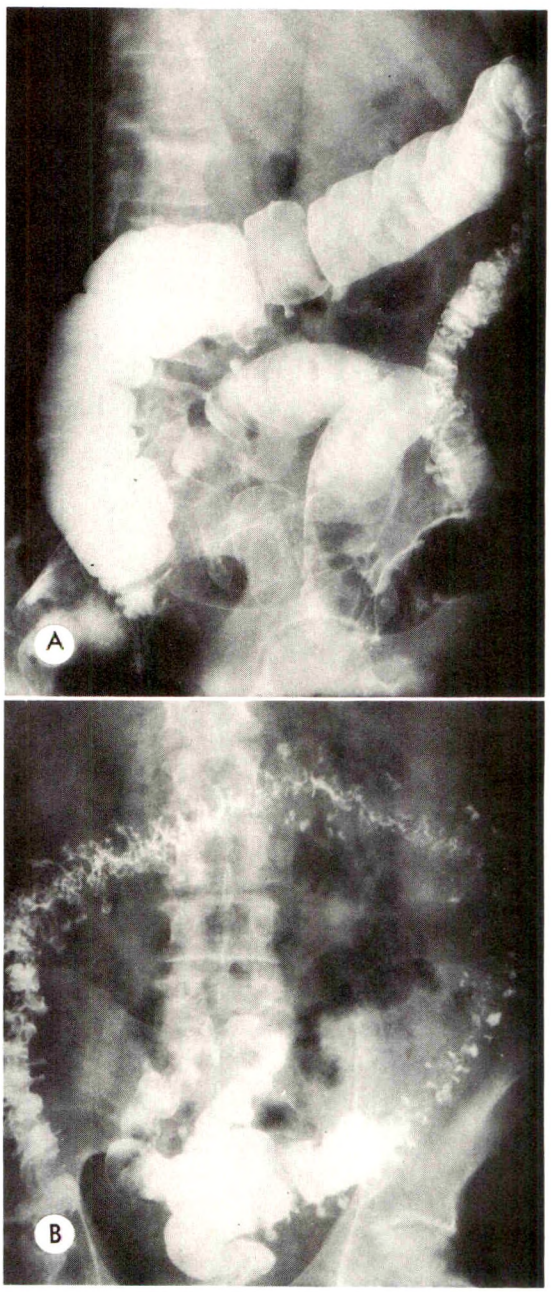


Fig. 8. Inguinal hernia of the cecum (*A*) Selective double contrast roentgenogram of the sigmoid. Left posterior oblique view. Despite abnormally low position of the cecum, it is not obscured by the overlapping sigmoid rendered radiolucent by water content. Scattered diverticula are also outlined. (*B*) Postevacuation roentgenogram of the colon following passive syphoning. Note excellent mucosal coating and good colonic emptying. The diverticula are again clearly seen.

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double contrast examination obtained by rectal gas insufflation (Fig. 3, *A* and *B*). Methylcellulose was not used, since it did not significantly add to the quality of the examination.

Water has proved to be an admirable double contrast agent, since it immediately mixes with the colonic barium. The resulting density, however, is significantly higher than that of plain water, and water density lesions such as polypoid masses have been clearly defined (Fig. 5, *A* and *B*; 6; and 7).

A postevacuation roentgenogram which permits an evaluation of a well coated colonic mucosa, is an important goal. This, however, is often not achieved, despite addition of tannic acid to the barium enema.[2,6] Janower[5] reported that even if 2 packets containing 2.5 mg. each of tannic acid were added to the enema, the mucosal coating was of good or intermediate quality in only 72 per cent of cases. The percentage dropped appreciably if only 1 packet was used. A good mucosal coating was not obtained in a significant number of his cases due to almost complete colonic evacuation and consequent insufficient barium residual.

With the currently described technique, good colonic evacuation by means of passive colonic syphoning[9,10] was obtained in most cases following the double contrast phase (Fig. 4, *A* and *B*; 5*B*; and 8*B*), despite the fact that no tannic acid was used (Table 1). A possible explanation for the excellent colonic evacuation obtained by syphoning may be related to inherent colonic elasticity and recoil following distention with water, as well as to the larger bore of the currently used enema kit tubing.*

ADVANTAGES AND INDICATIONS

The new technique provides an alternate method of double contrast examination of the colon. It obviates the additional step of gas insufflation and eliminates the neces-

* Manufactured by EZ-EM Co.

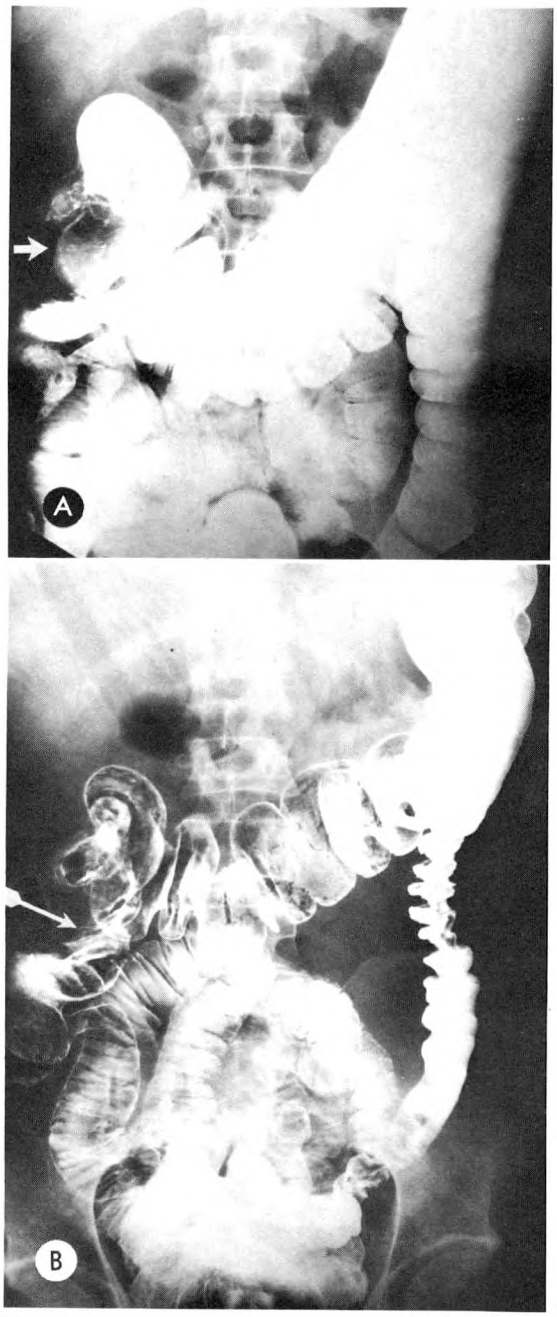


FIG. 9. Pericolonic abscess in the hepatic flexure secondary to perforation, presumably from localized diverticulitis. (*A*) Water double contrast study. Note large mass lesion with narrowing of the hepatic flexure (arrow). (*B*) Postevacuation roentgenogram following passive syphoning. Despite partial colonic evacuation in this case, a good mucosal coating and double contrast effect was obtained. Note pleating of the colonic wall at the hepatic flexure (arrow) indicating the extrinsic nature of the mass.

TABLE I

RESULTS OF SYPHONING OF THE COLON FOLLOWING DOUBLE BAG WATER CONTRAST TECHNIQUE IN 100 PATIENTS

Degree of Colonic Emptying	No. of Patients
More than 90%	41
70% to 90%	24
50% to 70%	12
Less than 50% emptying	23
Total	100

sity of preloading enema bags with gas.[10]

Since the study can be done in a single stage, utilizing a closed system,[9,10] there is no need for the patient to leave the roentgenographic table in order to evacuate. Evacuation is effectively accomplished by means of passive syphoning (Table 1). The latter additionally decreases chances of barium spillage and considerably shortens examination time. Since evacuation takes place *after* the double contrast phase of the study, the completeness of evacuation does not influence the double contrast phase as in routine double contrast studies.

Tannic acid is unnecessary with this technique; therefore, its expense, since 2 packets may be required,[5] as well as its potential hazards, are avoided.

The method permits a variable selective contrast technique of the colon. Thus, if the sigmoid is redundant or a cecal lesion is suspected, it is possible to significantly decrease sigmoid density and thereby afford an unobscured view of the cecum by allowing water to enter the sigmoid (Fig. 2*C*; 3, *A* and *B*; 5; *A* and *B*; 6; and 8*A*). A "see-through" effect is also obtained with redundant colonic loops (Fig. 3, *A* and *B*).

The technique is particularly useful in elderly patients with poor sphincter control, who have difficulty in retaining enemas despite special rectal catheters.[11] In the event of minor barium spillage prior to desired colonic filling, additional water can be introduced which will invariably advance the barium column towards the cecum.

Another useful feature of this technique is that it can be connected to special colostomy catheters[12] in order to provide good double contrast studies in patients with colostomies.

Due to its variable contrast feature, it may be possible to obtain a low density barium distribution throughout the colon (Fig. 3, *A* and *B*; and 5*A*), thereby reaping the benefits obtained only with high kilovoltage and/or low density barium[3] even though a standard barium density may be achieved on the initial roentgenograms (Fig. 2, *A* and *B*).

DISADVANTAGES

The double contrast effect obtained with the current technique may approach or simulate a routine double contrast colon study. However, the contrast may not be as sharp as that achieved with conventional techniques. Since the entire colon is filled, air-fluid levels cannot, as a rule, be obtained with erect or lateral decubitus views.

Another disadvantage of this technique is its tendency to produce excessive reflux into the terminal ileum, particularly if a double contrast study of the entire colon is pursued. Ileal reflux may be avoided by filling the colon solely to the splenic flexure in the initial phase of the study (Fig. 2*A*).

SUMMARY

A new technique for double contrast examination of the colon utilizing water instead of gas is described.

The study is carried out by means of a disposable prepackaged double bag enema kit utilizing a one-stage, closed system. Reliable postevacuation roentgenograms of the colon are achieved simply by passive syphoning and without the use of tannic acid.

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REFERENCES

1. Clifton, V. K., and Robertson, A. I. Methyl cellulose double contrast barium enema. *Med. J. Aus.*, 1968, *2*, 899–900.
2. Diner, W. C., Dodd, D., and Riggs, O. E. Value of bisacodyl tannex (clysodrast) in radiological examination of colon. *Radiology*, 1972, *104*, 49–51.
3. Figiel, S. J., Figiel, L. S., and Rush, D. K. Study of colon by use of high-kilovoltage spot-compression technique. *J.A.M.A.*, 1958, *166*, 1269–1274.
4. Fraser, A. M. Variable contrast barium enema. *Brit. J. Radiol.*, 1955, *28*, 698–700.
5. Janower, M. L. Efficacy study of use of clysodrast in barium enemas. *Radiology*, 1973, *106*, 704.
6. Lasser, E. C., and Gerende, L. J. Efficacy study of clysodrast. *Radiology*, 1966, *87*, 649–654.
7. Lucke, H. H., Hodge, K. E., and Patt, N. L. Fatal liver damage after barium enemas containing tannic acid. *Canad. M. A. J.*, 1963, *89*, 1111–1114.
8. McAlister, W. H., Anderson, M. S., Bloomberg, G. R., and Margulis, A. R. Lethal effects of tannic acid in barium enema: report of three fatalities and experimental studies. *Radiology*, 1963, *80*, 765–773.
9. Pochaczevsky, R., Sherman, R. S., and Meyers, P. H. Disposable kit for barium enemas. *Radiology*, 1961, *77*, 831–833.
10. Pochaczevsky, R., and Sherman, R. S. New technique for roentgenologic examination of colon. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 787–796.
11. Pochaczevsky, R., and Meyers, P. H. Safe, self-retaining, disposable rectal catheter for barium enemas with sealed insufflation bulb and movable external retention cushion. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 359–361.
12. Pochaczevsky, R., and Meyers, P. H. New, disposable catheter for selective guided barium enemas: contrast examinations in patients with colostomies, rectal lesions and fistulas and for pediatric colon studies. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 392–395.
13. Pochaczevsky, R. Oral examination of colon: colonic cocktail. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 318–325.
14. Sinclair, D. J., and Buist, T. A. S. Water contrast barium enema technique using methyl cellulose. *Brit. J. Radiol.*, 1966, *39*, 228–230.
15. Sinclair, D. J. Variable contrast examination of colon. *J. Canad. A. Radiologists*, 1971, *22*, 36–39.

GIANT DUODENAL DIVERTICULA*

By JACK R. MILLARD, M.D., FRED M. H. ZITER, JR., M.D., *and* WILLIAM P. SLOVER, M.D.
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DUODENAL diverticula are common incidental findings on barium examinations of the upper gastrointestinal tract, and their usual appearance has been well described previously.[1] Occasionally, they may become very large and present diagnostic difficulties on plain abdominal roentgenograms.

Four patients with "giant" duodenal diverticula are reported to illustrate their modes of presentation.

REPORT OF CASES

CASE I. O.A., a 69 year old woman, was first seen at Hartford Hospital in 1960 at which time a retroperitoneal fibrosarcoma was removed. In January, 1971, the patient was re-admitted with complaints of episodic nausea. A gastrointestinal series showed an 11 cm. diverticulum arising laterally from the fourth portion of the duodenum (Fig. 1*A*). A side-to-side duodenojejunostomy was performed, as it was felt that the diverticulum was causing partial and intermittent obstruction of the duodenum.

A follow-up examination obtained 10 months later, because of symptoms of high intestinal obstruction, showed that the diverticulum had increased in size to 20 cm. and lay in the right lower quadrant (Fig. 1, *B* and *C*).

CASE II. J.G., a 64 year old woman, was admitted in January, 1968, for hemorrhoidal bleeding. The patient gave a history of intermittent diarrhea for 2 years which was controlled with tincture of opium. A roentgenogram preliminary to a barium enema examination showed an abnormal air-fluid collection in the right upper quadrant (Fig. 2*A*). The subsequent upper gastrointestinal series demonstrated a 10 cm. diverticulum located laterally in the third portion of the duodenum (Fig. 2*B*). A follow-up examination 1½ years later showed no change in the appearance of the diverticulum.

CASE III. I.G., a 70 year old woman with a known duodenal diverticulum for more than 20 years was admitted in August, 1970, with a history of fatigue, nausea and vomiting. Roentgenograms obtained during a barium enema examination showed a loculated gas collection in the right upper quadrant (Fig. 3*A*). A subsequent upper gastrointestinal series demonstrated the presence of a large septate duodenal diverticulum arising from the third portion of the duodenum (Fig. 3*B*). Further work-up revealed the presence of pancreatic carcinoma. The patient was treated with chemotherapy.

CASE IV. E.C., an 88 year old woman, was admitted to Hartford Hospital in October, 1972, with symptoms of fatigue, nausea, anxiety and cancerophobia. Physical examination revealed a mass in the right lower quadrant. Subsequent tests proved this mass to be a low-lying right kidney. An upper gastrointestinal series showed a large duodenal diverticulum arising from the third portion of the duodenum (Fig. 4, *A* and *B*). All additional tests were within normal limits.

DISCUSSION

Diverticula of the duodenum are incidental findings in 1–5 per cent of barium examinations of the upper gastrointestinal tract and in approximately 20 per cent of autopsies.[5] The so-called "primary" or congenital diverticula have no muscularis propria and are found most frequently in the second and third portions of the duodenum. "Secondary" or acquired diverticula (pseudodiverticula) contain all layers of the duodenal wall and are usually sequelae of inflammatory disease. They are most frequent in the first portion of the duodenum. In one series, 88 per cent of diverticula were located on the medial wall of the duodenum, 4 per cent laterally and 8 per cent posteriorly.[7] Most diverticula are asymptomatic, but several complications have been reported.[2–4,6–9] These include

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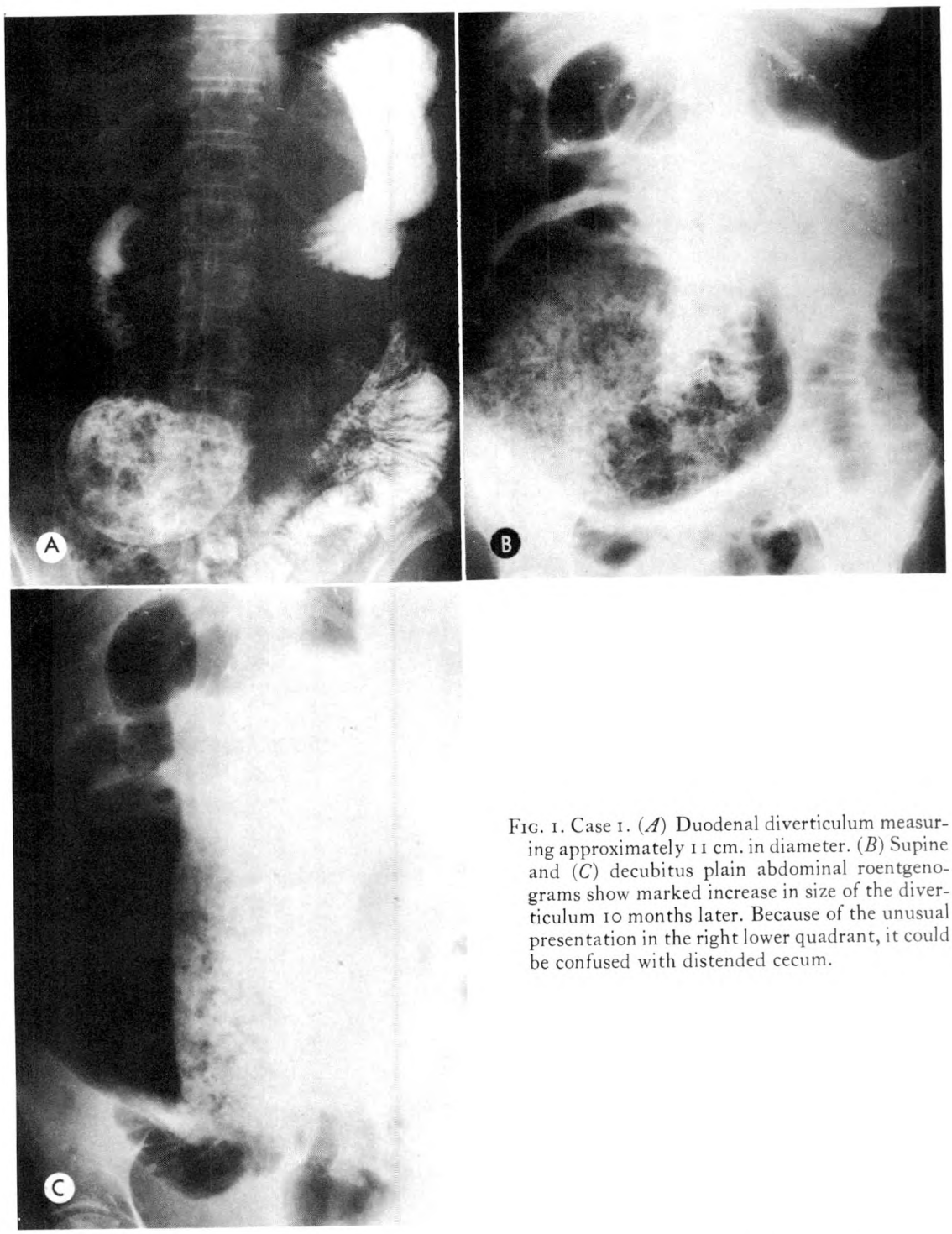


Fig. 1. Case 1. (*A*) Duodenal diverticulum measuring approximately 11 cm. in diameter. (*B*) Supine and (*C*) decubitus plain abdominal roentgenograms show marked increase in size of the diverticulum 10 months later. Because of the unusual presentation in the right lower quadrant, it could be confused with distended cecum.

small bowel obstruction from pressure phenomena, hemorrhage from ulceration,[9] perforation and fistula formation.[3] Occasionally, the common bile duct may insert into the diverticulum with subsequent biliary tract obstruction.[7]

Our cases demonstrate additional important points concerning these diverticula.

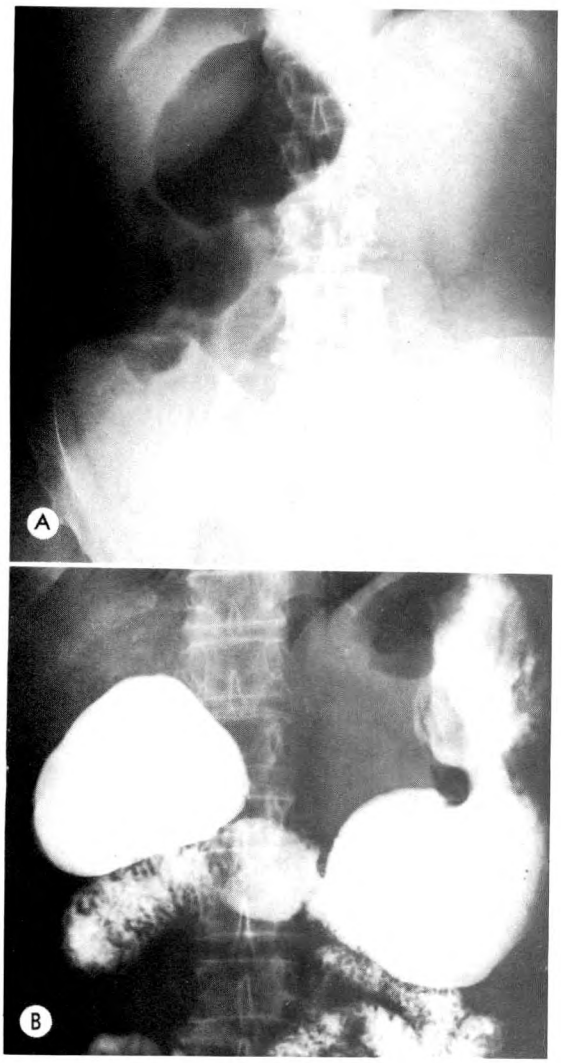
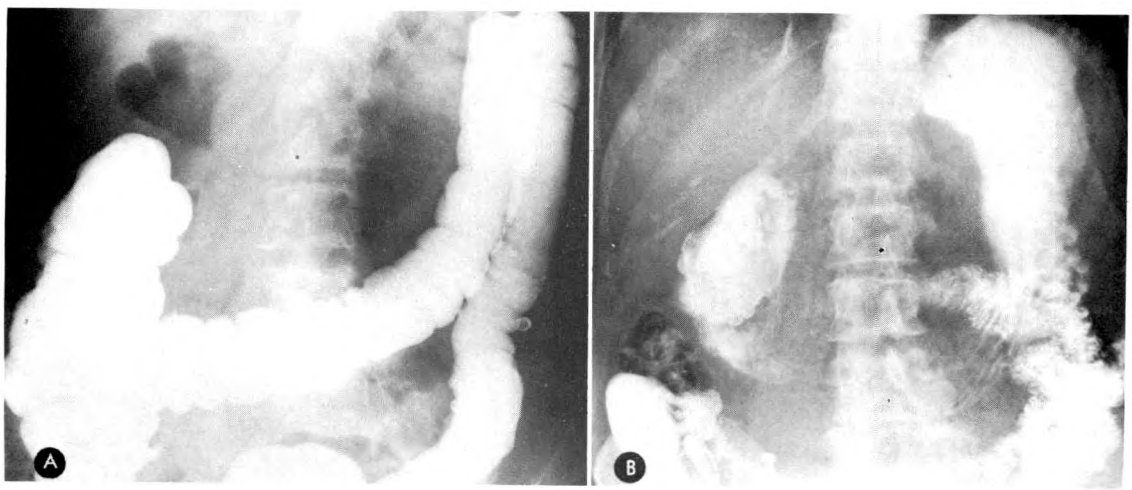


FIG. 2. Case II. (*A*) Smooth, gas-filled collection in the right upper quadrant. (*B*) Gastrointestinal series shows presence of a 10 cm. smooth walled lateral diverticulum of the duodenum.

First, they may become very large and present as confusing collections of air and/or fluid on plain roentgenograms of the abdomen. These collections may be in sites other than the right upper quadrant (Case I). Such a diverticulum may then be incorrectly interpreted as an abscess, dilated cecum, colonic diverticulum or pseudocyst of the pancreas. As shown in Case I, the giant duodenal diverticulum can also enlarge greatly in the interval between examinations.

Case III demonstrates that oblique roentgenograms may be of value by demonstrating the "neck" of the diverticulum, thereby offering a clue to the correct diagnosis.

Case IV demonstrates that such diverticula need not be smooth-walled, but may present as multilocular collections of gas. This appearance may be confused with an abscess, especially since the diverticulum may lie near Morrison's pouch.



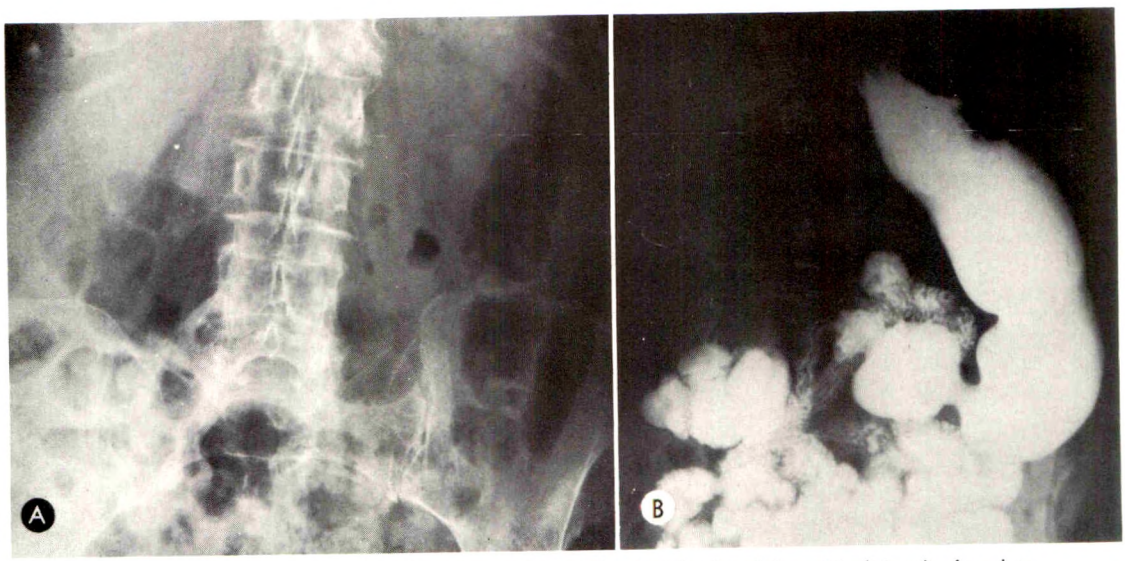


FIG. 4. Case IV. (*A*) Multiloculated gas-filled collection in the right mid-abdominal region. (*B*) Septate duodenal diverticulum shown on gastrointestinal series.

As noted previously, the great majority of duodenal diverticula occur on the medial aspect of the duodenum. In all of our cases, the giant diverticula arose laterally. Since medial diverticula are frequently surrounded by pancreatic tissue, gross enlargement of the diverticula would seem unlikely. However, it appears possible that the lack of such containment of a laterally-occurring diverticulum would account for their occasional large size.

SUMMARY

Four patients with giant duodenal diverticula are reported.

The appearance of these diverticula on plain roentgenograms of the abdomen is discussed, as is a possible reason for their large size.

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REFERENCES

1. ACKERMANN, W. Diverticula and variations of duodenum. *Ann. Surg.*, 1943, *117*, 403–413.
2. CAVENAGH, J. E., JR. Enteroliths and perforation of duodenal diverticula. *A.M.A. Arch. Surg.*, 1970, *100*, 614–618.
3. GALLIVAN, G. J., and PAINTER, R. W. Benign duodenocolic fistula. *Hartford Hospital Bull.*, 1968, *23*, 86–98.
4. JULER, G. L., LIST, J. W., STEMMER, E. A., and CONNOLLY, J. E. Perforating duodenal diverticulitis. *A.M.A. Arch. Surg.*, 1969, *99*, 572–578.
5. MARGULIS, A. R., and BURHENNE, H. J. Editors. Alimentary Tract Roentgenology. C. V. Mosby Company, St. Louis, 1967, p. 110.
6. MCSHERRY, C. W., and GLENN, F. Biliary tract obstruction and duodenal diverticula. *Surg., Gynec. & Obst.*, 1970, *130*, 829–836.
7. MUNNELL, E. R., and PRESTON, W. J. Complications of duodenal diverticula. *A.M.A. Arch. Surg.*, 1966, *92*, 152–156.
8. NEILL, S. A., and THOMPSON, N. W. Complications of duodenal diverticula and their management. *Surg., Gynec. & Obst.*, 1965, *120*, 1251–1258.
9. WOLFE, R. D., and PERAL, M. J. Acute perforation of duodenal diverticulum with roentgenographic demonstration of localized retroperitoneal emphysema. *Radiology*, 1972, *104*, 301–302.

FIG. 3. Case III. (*A*) Diverticulum seen as gas-filled structure on a barium enema roentgenogram. Oblique roentgenogram showed the "neck" of the diverticulum. (*B*) Gastrointestinal series confirms the presence of a septate diverticulum arising laterally from the third portion of the duodenum.

GIANT SIGMOID DIVERTICULUM*

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THE roentgenologic appearance of a giant air cyst arising from the sigmoid colon was originally described in 1953 by Hughes and Green[4] and since then has only been sporadically recorded in the literature. It is difficult to understand this unusually low incidence since the lesion is always found in association with diverticular disease in the elderly—a common enough entity in this age group.

During the past 2 years we have collected 5 cases (Table 1). All 5 patients underwent surgery. The roentgenologic findings, differential diagnosis, and pathologic features encountered form the basis of this report; they further support the view that these giant air cysts represent another manifestation of diverticulitis.

REPORT OF CASES

CASE II. H.P., a 64 year old male, was admitted to the hospital because of a 2 month history of crampy gas pain in his right side. He suffered from chronic constipation for many years but in the 2 months preceding this hospital admission noticed a change in his bowel habits. At this time he experienced repeated attacks of pain and diarrhea which lasted for 2 to 3 hours at a time. Physical examination of the abdomen was unremarkable. Sigmoidoscopy was performed and demonstrated a narrowing approximately 18 cm. from the anal verge. The colonic mucosa was intact and appeared normal to the site of the constriction and a mucosal biopsy was also interpreted as normal.

A supine roentgenogram of the abdomen revealed a large, oval 8.0 cm. gas filled cystic mass in the left iliac fossa. Barium study of the colon demonstrated a constriction at the level of the rectosigmoid colon. The mucosa was coarse, irregular and compressed (Fig. 2*A*). A large sac filled with barium was present in the area of the constriction. This corresponded to the air filled cyst seen on the scout roentgenogram. The remainder of the colon was normal.

Resection of the diverticulum and the adjacent colon was performed.

Pathologic study showed that the cystic mass measured 7.5 cm. in diameter and had a 1.0 cm. thick wall (Fig. 2*B*). The neck connecting the diverticulum to the colon was too small to be identified macroscopically and was detected only after careful microscopic study. No identifiable mucosa was present and the cyst wall was composed of granulation and fibrous tissues (Fig. 2*C*). The diagnosis was attributed to diverticular perforation with abscess formation.

CASE IV. L.T., a 79 year old white male, was admitted to the hospital for the first time because of the recent onset of melena. No pain was associated with the melena nor was there any history of constipation or diarrhea. His past history was also unremarkable apart from a previous appendectomy. On physical examination a nontender mobile mass measuring 10 cm. in diameter was palpated in the left lower quadrant.

The diagnosis of diverticular disease of the colon with superimposed carcinoma was considered and a barium enema examination performed. On this examination, a large oval lucency was noticed in the left lower quadrant. This arose from the sigmoid colon and filled with barium. Other small diverticula were present and the mucosa in this region had a coarsened edematous appearance. The remainder of the colon was normal.

The patient underwent surgery and a large cystic mass together with 25 cm. of adjacent sigmoid colon was resected.

On pathologic examination the round mass communicated with the lumen of the colon *via* a small 0.5 cm. opening. The diverticulum was filled with some fecal material and reddish brown hemorrhagic fluid. The wall was thick

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Table I

SUMMARY OF CASES

Case	Sex	Age	Pain	Mass	Melena	Nausea and Vomiting	Diarrhea	Location	Roentgenographic Size of Tumor (cm.)
I. (*Fig. 1, A and B*)	F	65	+ (recent)	not palpable	+	−	−	Sigmoid	22.0
II. (*Fig. 2, A–C*)	M	64	+ (2 months)	not palpable	−	−	+	Sigmoid	8.0
III. (*Fig. 3, A and B*)	M	69	−	palpable	−	−	−	Sigmoid	12.0
IV. (*Fig. 5*)	M	79	−	palpable	+	−	−	Sigmoid	7.0
V. (*Fig. 4*)	F	71	+ (1 month)	palpable	+	−	−	Sigmoid	6.0

and measured 0.8 cm. (Fig. 5). On histologic examination only fibrous and granulation tissues were present and the diagnosis of diverticulitis of the colon was established.

CASE MATERIAL AND CLINICAL PRESENTATION

Our 5 cases comprised 3 males and 2 females, whose ages ranged from 64 to 79 years. Pain was a variable presenting symptom in our series and surprisingly was absent in 2 patients. When present it was either of very recent origin or had persisted for several weeks or months. None of our patients complained of nausea or vomiting, a frequent presentation in the review of Asch *et al.*[1] In 1 patient, the mass was totally unsuspected and was a chance discovery on routine physical examination prior to hemorrhoidectomy (Case III) and in 2 others, the mass was felt when attention was drawn to the abdomen because of other symptoms. Blood mixed with stool is commonly found,[7] but was the presenting symptom in only 2 of our patients. Change of bowel habit with diarrhea was encountered in one.

DISCUSSION

Although the clinical presentation in many aspects may be nonspecific, the mass is easily identified on abdominal roentgenologic studies as a large, well circumscribed radiolucent round to oval cystic structure within the lower abdomen (Fig. 1, *A* and *B*).[2] It is almost always related to the sigmoid colon. Multiple projections including a right or left decubitus view may be required to best demonstrate this. The wall of the cyst appears smooth and regular. This feature is important in distinguishing it from other gas-containing structures that may occur in this location. The margin of an acute abscess is ill defined and less regular and the abscess often presents with a bubbly appearance on the plain abdominal roentgenogram. The irregular mixture of gas and pus within the abscess accounts for this appearance. A roentgenogram taken in an upright or decubitus position may, however, demonstrate an air-fluid level. Although emphysematous cystitis could resemble a giant colonic cyst, its location arising centrally in the pelvis is distinctive and if necessary intravenous pyelography will confirm the diagnosis.

Occasionally, pneumatosis intestinalis may cause some confusion in the differential diagnosis. However, the size of the gas

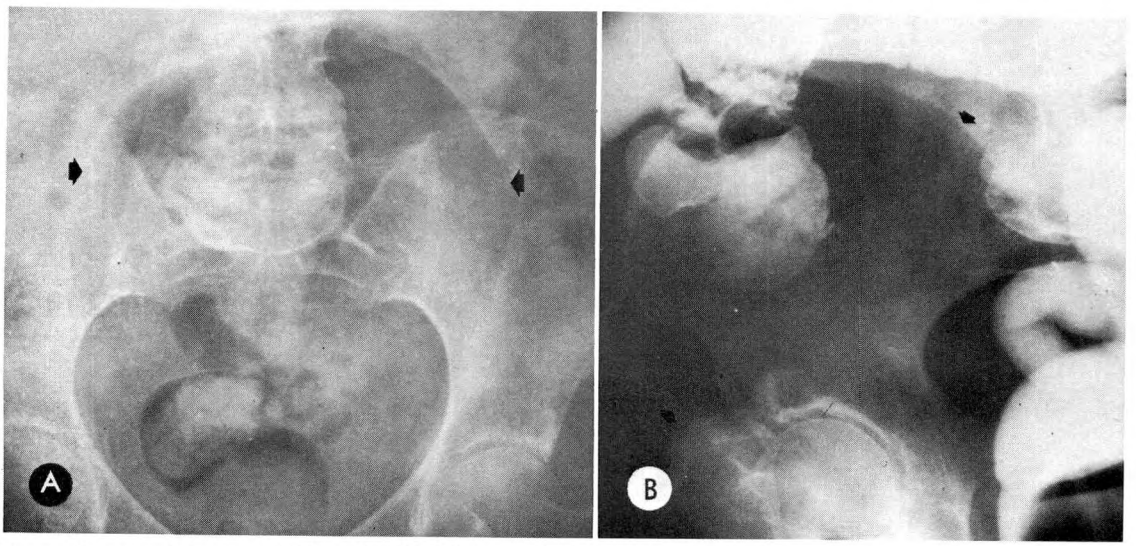


FIG. 1. Case 1. A 65 year old female with history of pain and melena.
(*A*) A large air-containing cyst is present within the pelvis (arrows). The wall of the cyst is smooth and regular and because of its location could be confused with emphysematous cystitis. (*B*) During the barium enema examination only minimal filling of the cyst occurred (arrows) (this is not apparent on this film). Surprisingly, this mass was not palpated clinically.

cysts in this condition average 1–3 cm. in diameter and never attain the proportion of the single giant sigmoid cyst. Moreover, the multiplicity and distribution of the cysts throughout the colon are characteristic. Although lipoma may have a superficially similar radiographic density, it is not as lucent nor necessarily restricted to the left lower quadrant.

The giant cyst usually fills with barium during the enema examination except when the diameter of the neck is too narrow to permit entry of the contrast material (Fig. 2; 3; and 4). Gastrografin could perhaps be used in this circumstance, although opacification of the cysts was accomplished in all our cases by the use of barium alone. Filling of an exceptionally large cyst may not always be apparent, as at times little barium may enter the cyst. The segment of colon from which the giant air cyst arises is always deformed by diverticular disease and may be simultaneously compressed by the cyst (Fig. 2). Both these factors serve to narrow the lumen and the resulting appearance may be confused with a carcinoma. Following evacuation, barium may be retained within the cyst for variable periods of time.

It was originally thought that these giant cysts represented congenital duplications. However, this concept is no longer acceptable since these giant air cysts demonstrate an entirely different histologic picture and are found only in the elderly.

One case of a giant cyst has been described in a location other than the sigmoid colon.[6] This was situated in the transverse colon and was similarly associated with diverticular disease. All other cases including our series were confined to the sigmoid colon. When examined pathologically, the cyst wall is thick, measuring up to 1 cm. (Fig. 2*B*; and 5). Histologically this is composed predominantly of dense granulation and fibrous tissues (Fig. 2*C*). Occasionally, residual portions of serosa and mucosa may be detected microscopically, but muscle tissue is not normally encountered. These features corroborate the contention that the giant cyst represents the end result of an acquired pseudo-diverticulum that has undergone superimposed infection, abscess formation, and healing.[3] The original mucosal and serosal tissues are destroyed and replaced by granulation tissue with result-

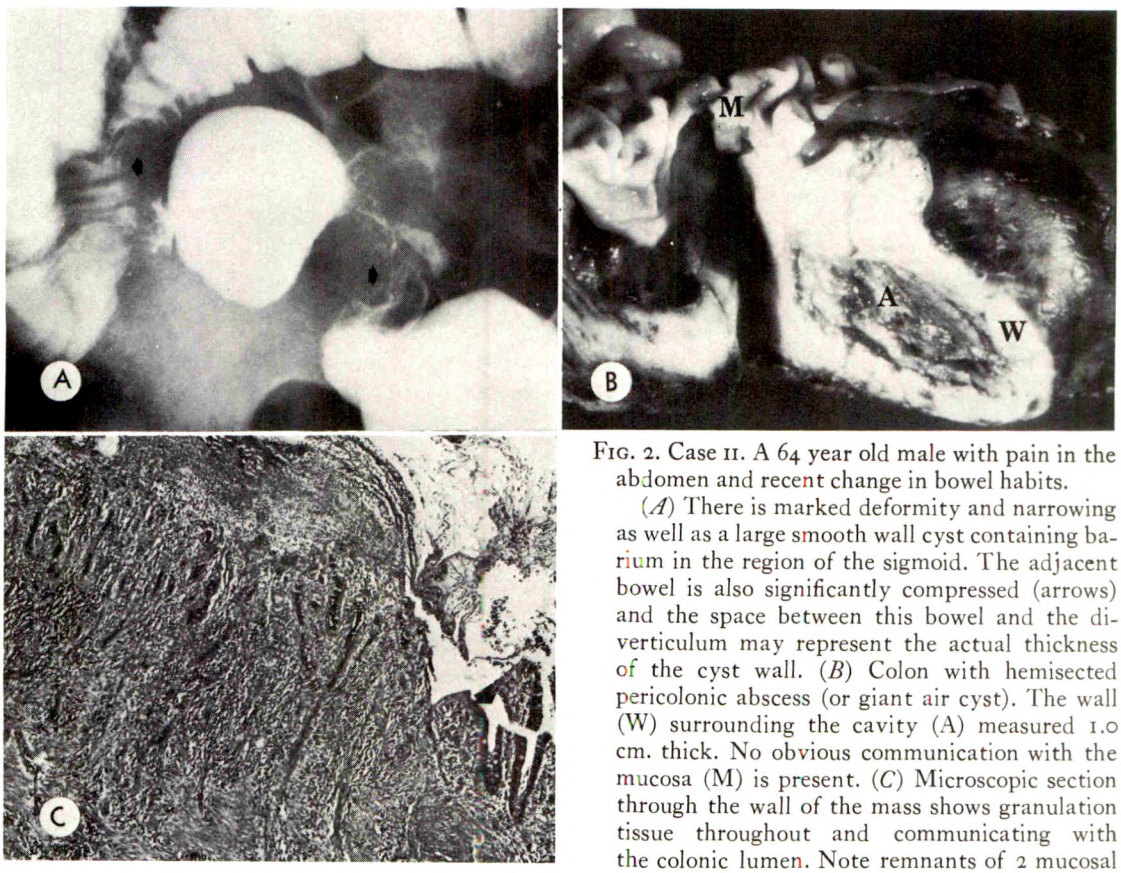


Fig. 2. Case 11. A 64 year old male with pain in the abdomen and recent change in bowel habits.

(*A*) There is marked deformity and narrowing as well as a large smooth wall cyst containing barium in the region of the sigmoid. The adjacent bowel is also significantly compressed (arrows) and the space between this bowel and the diverticulum may represent the actual thickness of the cyst wall. (*B*) Colon with hemisected pericolonic abscess (or giant air cyst). The wall (W) surrounding the cavity (A) measured 1.0 cm. thick. No obvious communication with the mucosa (M) is present. (*C*) Microscopic section through the wall of the mass shows granulation tissue throughout and communicating with the colonic lumen. Note remnants of 2 mucosal glands (arrow in lower right).

ing fibrosis. However, the relative paucity of clinical symptoms noted in all our cases and in some reported in the literature, suggests that this abscess is quite slow in development and frequently undergoes a chronic subclinical course. This is quite different from the more dramatic presentation of acute diverticulitis with perforation, although this too may occasionally heal as a walled off cystic structure.[5]

Duplication cysts are composed of all the elements of normal bowel wall. However, it is possible that complicating infection could cause a similar pathologic appearance and in some cases, recorded as giant diverticular cysts, remnants of smooth muscle tissue have been detected microscopically. These cysts in all probability were duplications.[1]

It has also been suggested that these large cysts represent hyperinflated diverticula caused by the act of straining. A "ball valve" action is thought to occur at the neck of the diverticulum resulting in air-trapping. If this were the only functioning mechanism, one would anticipate a high incidence of giant diverticula. In addition, the inner lining should then consist of normal mucosa and the wall should be thin. Straining, nevertheless, probably accounts for the variations in the amount of gas contained within the cysts. The larger cysts no doubt have very narrow connecting necks with an effective "ball valve" mechanism.

It is important to recognize these giant cysts as another complication of diverticular disease since it allows one to more easily recognize the changes occurring in the adjacent bowel. All too often the associated colonic narrowing and deformities have been interpreted as carcinoma. Although carcinoma may also occur in this area, the

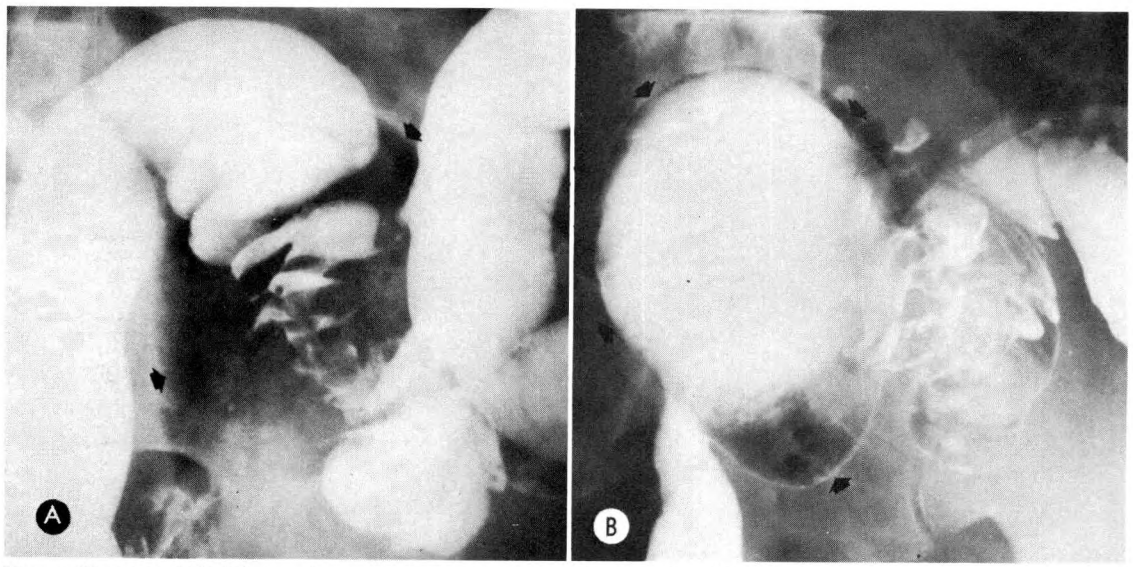


FIG. 3. Case III. (*A*) A large air-containing cyst (arrows) is noted to lie in association with the sigmoid colon in which considerable diverticulosis and diverticulitis are noted. This mass was only discovered on a routine physical examination done prior to hemorrhoidectomy. (*B*) A repeat study performed again reveals a large air-containing cyst (arrows), this time filled with barium.

presence of a giant air cyst would suggest underlying diverticular disease. The reason for its occurrence in the sigmoid is probably related to the frequency of diverticular disease, but its absence in other portions of the colon still remains unanswered.

SUMMARY

The roentgenographic features and pathogenesis of the giant air-containing cyst of the sigmoid are described as seen in 5 patients.

These cysts are readily diagnosable on the abdominal roentgenogram and barium enema examination and probably represent

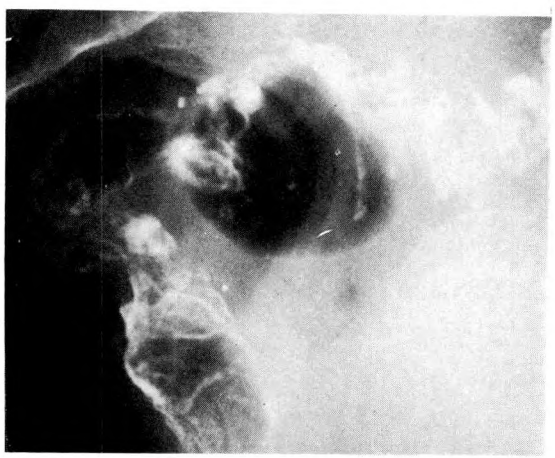


FIG. 4. Case V. Barium study in a patient with melena reveals diverticular disease within the sigmoid colon associated with a large air filled cyst.

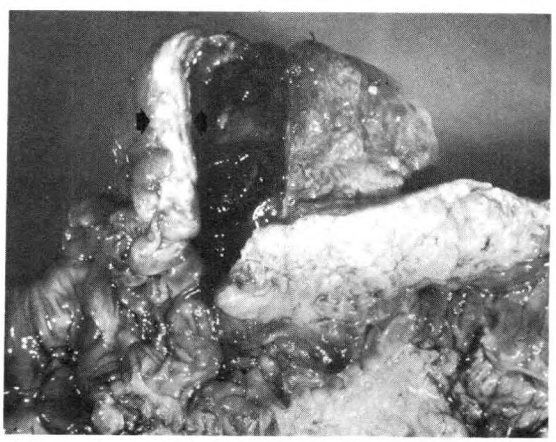


FIG. 5. Case IV. Pathologic specimen demonstrates a large cystic mass arising superiorly from the colon. Note the markedly thickened wall (arrows) and somewhat irregular inner surface.

a slowly progressing chronic diverticular abscess.

This complication, when recognized, allows one to more readily diagnose the colonic deformities visualized in the adjacent bowel.

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REFERENCES

1. Asch, T., Melikow, E., and Gump, F. Giant gas cysts of sigmoid: report of case and review of literature. *Radiology*, 1970, *96*, 409–410.
2. Bergeron, R. B., and Hanley, P. H. Giant sigmoid diverticulum. *Am. J. Surg.*, 1965, *100*, 660–662.
3. Frankenfeld, R. H., Waters, C. H., and Schepeler, T. V. Giant air cyst of abdomen: unusual manifestation of diverticulitis of sigmoid. *Gastroenterology*, 1959, *37*, 103–106.
4. Hughes, W. L., and Greene, R. C. Solitary air cyst of peritoneal cavity. *A.M.A. Arch. Surg.*, 1953, *67*, 931–936.
5. Macbeth, W. A. A. G., and Riddle, P. R. Gas filled abscess cavity as manifestation of diverticulitis of colon. *Brit. J. Radiol.*, 1964, *37*, 861–862.
6. Sagar, S. Giant solitary diverticulum of transverse colon with diverticulosis. *Brit. J. Clin. Pract.*, 1973, *27*, 145–146.
7. Swann, J. C. Giant diverticulum of sigmoid colon. *Brit. J. Radiol.*, 1971, *44*, 551–553.

LARGE DIVERTICULUM OF THE ANAL CANAL*

CASE REPORT AND REVIEW OF THE LITERATURE ON ANAL CANAL AND RECTAL DIVERTICULA

By A. F. GOVONI, M.D., C.R.C.P.(C),† *and* JAN J. SMULEWICZ, M.D.‡
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THE medical literature on diverticulosis and diverticulitis of the colon is extensive. However, in our search for reports on diverticula of the anal canal, we found only 3 cases recorded and only 29 cases of rectal diverticula reported from 1911.[3,11,14]

Regarding frequency of rectal diverticula, Turnbull[15] states that he has "seen any number of patients with diverticula below the 15 cm. level (as measured above the dentate line) . . . I must state they are not common. I have seen at least 3 patients with large diverticula of the rectum proper." Welch[18] writes, in 1958, that he has never observed a diverticulum arising from the extraperitoneal rectum, in the segment below the peritoneal floor. Mayo[9] in 1954 stated that no diverticula beneath the peritoneal reflection have been demonstrated roentgenologically.

In Tables I and II are listed the cases of anal canal and rectal diverticula reported in the literature. Only in the cases reported by Doyle,[1] Kyan and Haines[8] and Case 1 of Walstad and Sahibzada,[17] were the rectal diverticula more than 2.5 cm. in diameter. The anal canal diverticulum reported by Patil[11] was 1.0 cm. in diameter and in our case about 6 cm. in diameter. In the cases reported, including the present case, diverticula were present in the remaining segments of the large bowel, mainly the sigmoid and descending colon, in 12. A greater incidence was noted in males and the age of the patients varied between 18 and 86 years. Finally, it is worthy of mention that in Case II of Giffin,[6] in the area of one of the several diverticula, a carcinomatous lesion was considered to have developed in the diverticulum itself.

REPORT OF A CASE

H.F. (Q.H.C. 132437) A 73 year old man was admitted to the hospital with a history of

TABLE I

DIVERTICULA OF THE ANAL CANAL

No. of Cases*	Author	Sex	Age (yr.)	Shown by Barium Enema	Solitary	Size	Surgery
1	Telling and Gruner[14] 1917†						
2	Dufour *et al.*[3] 1957	F	35				V
3	Patil[11] 1968	M	65		V	1.0 cm.	V
4	Govoni and Smulewicz 1973	M	72	V	V	6.0 cm.	V

* Three cases reported by Gant[5] in 1923, observed in a boy of 5 years of age and 2 in adults of 51 and 27 years of age respectively, could have been situated in the anal canal.
† No other information was given.

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TABLE II

DIVERTICULA OF THE RECTUM

No. of Cases	Author	Sex	Age (yr.)	Shown by Barium Enema	Solitary	Multiple	Surgery
1	Giffin[6], 1911†	F	56			V	V
2	Giffin[6], 1911	M	48			V	V
3	Shoemaker[12], 1914	F	63		V		
4	Gant[5], 1923	M	27		V		
5	Gant[5], 1923	M	45		V		
6	Gant[5], 1923	F	40		V		
7	Gant[5], 1923	M	50			V	
8	Gant[5], 1923	M	30		V		
9	Gant[5], 1923‡	F	60				
10	Gant[5], 1923‡	M	26				
11	Gant[5], 1923‡	M	18				
12	Gant[5], 1923‡	M	20				
13	Spriggs and Marxer[13] 1927‡						
14	Spriggs and Marxer[13] 1927						
15	Spriggs and Marxer[13] 1927						
16	Spriggs and Marxer[13] 1927						
17	Tweddell[16], 1954	M	64		V		
18	Turnbull[15], 1959‡						
19	Turnbull[15], 1959						
20	Turnbull[15], 1959						
21	Doyle[1], 1959	M	86	V	V		
22	Weston and Schlachter[19] 1959	F	47		V		V
23	Kürten-Rothes[7] 1967	M	72	V		V	V
24	Walstad and Sahibzada[17] 1968	M	66		V		
25	Walstad and Sahibzada[17] 1968	F	78			V	
26	Walstad and Sahibzada[17] 1968	F	62			V	
27	Walstad and Sahibzada[17] 1968	M	73		V		
28	McIlwain[10], 1968‡						
29	Kyan and Haines[8], 1971	M	47	V		V	V

† The author mentions that 9 cases of rectal diverticula were reported before 1911. Of these, 2 were operated on and 7 were found at autopsy.

‡ No other information was given.

hemorrhoids, intermittent rectal bleeding and a seizure disorder. On more direct questioning, the patient admitted intermittent rectal tenesmus, irregular bowel movements, and a feeling of "something pressing" in the perineal region. On sigmoidoscopy, the presence of hemorrhoids was confirmed, and at least 1 cm. above the anal sphincter a small opening was noted in the wall of the rectal ampulla.

The patient was referred to the Department of Radiology for further evaluation of this finding. A barium enema examination was performed. The contrast medium passed freely into the rectal ampulla and was seen to enter and opacify a large diverticular pouch situated between 1.5 and 2 cm. above the anus (Fig. 1; and 2). The remaining segments of the colon were within normal limits. The roentgen diag-

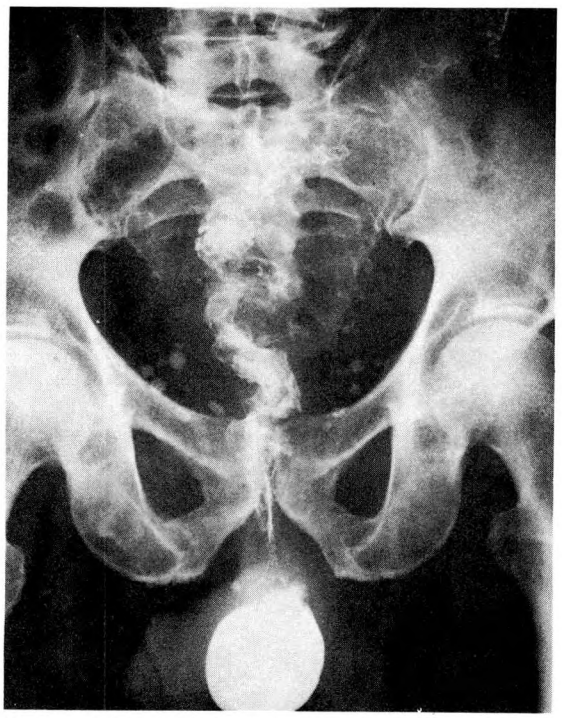


FIG. 1. The postevacuation roentgenogram shows that the anal canal diverticulum, opacified during the performance of the barium enema examination, retains the contrast medium, even after the patient applied pressure in the perineal region.

nosis of diverticulum of the anal canal was confirmed at surgery.

On microscopic examination the removed diverticular pouch was shown to be lined with rectal mucosa. The diverticulum contained fecal material and a few pieces of what appeared to be chicken bone. The postoperative course was uneventful and the patient was discharged.

DISCUSSION

In 1916 Drummond,[2] writing on diverticula of the colon, felt that there was a relationship between the site of penetration of the vasa recta and the formation of the diverticula. These weak points in the walls of the bowel would be a predisposing factor to the formation of the diverticula. More recently, Fleischner[4] pointed out that the colonic diverticulum was only an "outpouching of the mucous membrane" through a point of weakness in the circular muscle and secondary to the "effect of intraluminal pressure upon such a weak spot." The low incidence of rectal and, in particular, anal canal diverticula is most likely related to an inconstant intraluminal pressure in the rectum. Giffin[6] believed that the "longitudinal fibers of the tinea coli, by spreading out to ensheath the entire rectum, protect it from undue stress." Furthermore, strong structures support and surround the rectum and the feces exert less internal pressure in the rectal area.

Among the predisposing factors in the formation of rectal diverticula we must consider: obesity with associated fatty infiltration of the muscular layers, and secondary formation of weak spots; a sedentary occupation; probable congenital abnormalities in the walls of the rectum; an absence of the coccyx; a relaxed rectovaginal septum;[10] chronic constipation with associated changes in the tissue elements of the rectal wall and, in particular, atrophic changes in the muscular layer; and trauma by foreign bodies, as suggested by Weston and Schlachter,[19] that result in segmental weakening of the wall of the rectum. Specifically, in the formation of anal canal diverticula, Patil[11] mentions 3 important acquired local conditions which should be considered as predisposing factors in the formation of a diverticulum in this segment of the colon: "severe degree of perineal laceration, weakness of the perineal muscles, and infection of cryptic anal glands."



FIG. 2. The diverticulum is in an anterior position in its relationship with the anal canal.

In our case, because of the presence of fragments of chicken bone, one must accept trauma as the etiologic factor in the formation of the large diverticulum of the anal canal.

There are several pathologic entities to be considered in the differential diagnosis of anal canal or rectal diverticula. The presence of a rectal mass must be differentiated from a neoplastic growth. In female patients, endometriosis can be distinguished from a rectal diverticulum because of the clinical symptomatology related to the appearance of the menses. Furthermore, on physical examination, one should feel the presence of firm, tender, multiple and rather movable nodules. In carcinoma, there is a chronic and intermittent history of partial or pseudointestinal obstruction, abdominal pain, and diarrhea. Finally, in cases of fistula *in ano* there is a history of abscess formation and an associated long history pointing to the development of a peri- and para-anal abscess. The presence of rectal bleeding, as in the case of Patil,[11] could be quite confusing, unless on sigmoidoscopy one can demonstrate the site and cause of origin of the bleeding.

In all the cases of anal canal and rectal diverticula reported in the literature in which mention was made of treatment, the diverticulum was removed surgically and "healing by second intention is preferred in order to prevent recurrence."[11]

SUMMARY

A case of giant diverticulum of the anal canal, demonstrated on barium enema examination, confirmed at surgery and by microscopic examination of the specimen, is reported.

The literature is reviewed and the clinical findings and the etiopathogenesis of the diverticula of the rectum and of the anal canal are discussed.

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REFERENCES

1. Doyle, O. W. Large diverticulum of rectum. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *81*, 694–695.
2. Drummond, H. Sacculi of large intestine, with special reference to their relationship to blood vessels of bowel wall. *Brit. J. Surg.*, 1916, *4*, 407–413.
3. Dufour, R., Cabanié, H., and Sentucq, M. À propos d'un diverticule du anal canal. *Arch. mal. app. digest.*, 1957, *46*, 1178–1179.
4. Fleischner, F. G. Diverticular disease of colon, new observations and revised concepts. *Gastroenterology*, 1971, *60*, 316–324.
5. Gant, S. G. Diseases of the Rectum, Anus and Colon. W. B. Saunders Company, Philadelphia, 1923, 2, pp. 279–281, and 286.
6. Giffin, H. Z. Diverticulitis of rectum: report of two cases operated upon, one of them with carcinomatous degeneration. *Ann. Surg.*, 1911, *53*, 533–537.
7. Kürten-Rothes, R. Perforation eines extraperitonealen Rektum-divertikels bei einen Kontrasteinlauf. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1967, *106*, 155–157.
8. Kyan, M. M., and Haines, J. O. Rectal diverticula. *Radiology*, 1971, *100*, 273–284.
9. Mayo, C. W. Diverticulosis and diverticulitis of colon. *Collect. Papers Mayo Clin.*, 1954, *45*, 155–157.
10. McIlwain, A. J. Radiologic seminar LXXVI: acquired diverticulum of rectum. *J. Mississippi M. A.*, 1968, *9*, 362.
11. Patil, K. D. Bleeding from diverticulum of anal canal. *Dis. Colon & Rectum*, 1968, *11*, 292–297.
12. Shoemaker, G. E. Obstructive pelvic lesions associated with chronic diverticulitis. *Ann. Surg.*, 1914, *59*, 422–425.
13. Spriggs, E. C., and Marxer, O. A. Multiple diverticula of colon. *Lancet*, 1927, *1*, 1067.
14. Telling, W. H. M., and Gruner, O. C. Acquired diverticula, diverticulitis, and peridiverticulitis of large intestine. *Brit. J. Surg.*, 1916–1917, *4*, 468–530.
15. Turnbull, R. B. Personal communication to S. D. Weston, and I. S. Schlachter.[19]
16. Tweddell, T. N. Diverticulitis of rectum. *J. Canad. M. A.*, 1954, *70*, 569.
17. Walstad, P. M., and Sahibzada, A. R. Diverticulum of rectum. *Am. J. Surg.*, 1968, *116*, 937–939.
18. Welch, C. E. Diverticula in other segments of colon. *DM*, 1958, *34*.
19. Weston, S. D., and Schlachter, I. S. Diverticulum of rectum. *Dis. Colon & Rectum*, 1959, *2*, 458–464.

EVALUATION OF THE RETROGASTRIC SPACE*

NORMAL APPEARANCE AND VARIATION

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ONE of the most difficult areas of roentgenographic evaluation is the retrogastric space. Numerous attempts at quantitative measurement of the retrogastric space have been carried out,[2,4] but precise correlative studies of the normal contours of the posterior gastric wall relating these to the normal adjacent structure are lacking.

The stomach is a hollow viscus with pliable walls. Its shape and position in the abdomen depend upon the structures intimate to it in both the intraperitoneal and extraperitoneal space. Its shape, however, is variable depending on its position. Some stomachs extend quite inferiorly while others are high. The evaluation of the posterior wall of the stomach on the lateral roentgenograms then depends on the position of the stomach and therefore what structures are indenting it. The lateral roentgenogram must always be viewed with a frontal roentgenogram, with the patient in the same position with the tube rotated 90°.

The purpose of this communication is to demonstrate contours of the posterior gastric wall and note variations produced by change in position caused by change in organ relationships.

This study has made use of clinical roentgenograms following simultaneous opacification of the kidneys and stomach. In addition, a roentgenographic examination of a cadaver was made after the introduction of barium into the upper gastrointestinal tract. This examination consisted of matching anteroposterior and lateral views in upright, supine, right and left lateral positions. Immediately afterward, the cadaver was frozen to a solid state by placing it in a tank with dry ice for 24 hours.[6] It was then placed on a bandsaw and multiple sagittal sections through the left abdomen were made and then photographed and roentgenographed. Correlation of the structures related to the contour of the posterior gastric wall was made.

After reviewing the information available from the matching frontal and lateral roentgenograms of the intact cadaver, it became apparent that the upright views, because of the obvious air-fluid levels in both views, provided the most information about the stomach and its surrounding structures.

It is not possible to evaluate the retrogastric space on a lateral roentgenogram of the abdomen without having a corresponding frontal view taken with the subject in the same position. This allows a 3 dimensional evaluation of the abdomen by enabling the observer to determine which structures are in the same sagittal plane in the lateral view.

The importance of corresponding anteroposterior and lateral roentgenograms of the abdomen was recognized by Engle and Lysholm[1] in 1934 when they evaluated the gas filled stomach to measure the retrogastric space. Poppel *et al.*[3] in 1949 measured the midline retrogastric soft tissues and stressed the need for a matching anteroposterior view to show at which vertebral body the stomach crossed the spine. Taveras and Golden[5] demonstrated the mobility of the abdominal organs and also emphasized the need for a corresponding frontal view to allow measurement of the retrogastric space.

It is possible to identify corresponding parts of the upper gastrointestinal tract and the adjacent structures in matching frontal and lateral views by using the available clues: position of air-barium levels;

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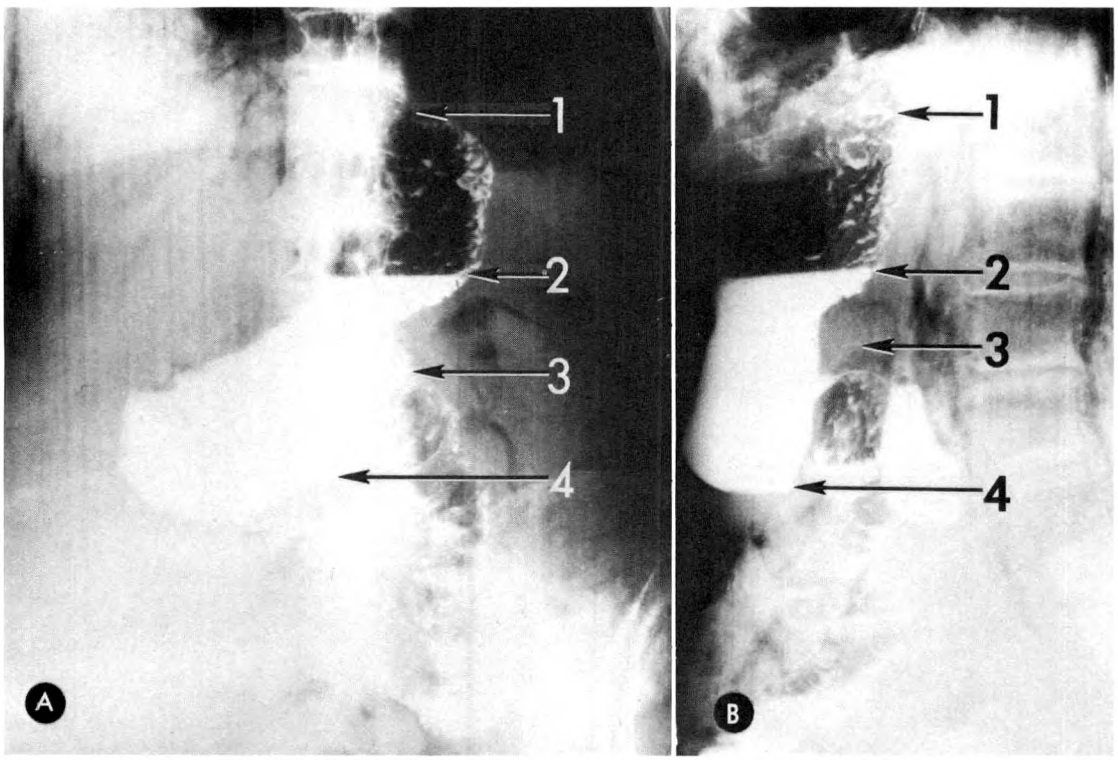


Fig. 1. Upright (*A*) anteroposterior and (*B*) lateral views of cadaver with barium in upper gastrointestinal tract. Note corresponding landmarks on the anteroposterior and lateral views: (1) cardia of stomach; (2) barium-air level in stomach; (3) duodeno-jejunal junction; (4) lowermost position of barium filled stomach.

peculiar contours; density changes or specific anatomic relationships.

Figure 1, *A* and *B*, is anteroposterior and lateral roentgenograms of the cadaver in the upright position. By identifying specific points in the frontal view it is possible to determine which structures are in the same sagittal plane in the lateral view. The air-filled cardia of the stomach can be easily identified in the anteroposterior view. Other specific points that can be identified include the gastric air-barium level, the most dependent portion of the barium filled stomach and the air filled duodeno-jejunal junction. By identifying these specific points in the lateral view a 3 dimensional image of this region can be developed.

DESCRIPTION OF THE ANATOMY

In order to produce a more complete understanding of the retrogastric space, multiple sagittal sections of a cadaver were made. Figure 2 demonstrates the planes through which sagittal sections of the left upper abdomen were made. Photographs and roentgenograms of the sections were compared. The gross sections were referred to when necessary.

Figure 3, *A* and *B*, is the photograph and the roentgenogram of the sagittal section through the medial aspect of the spleen, the most lateral portion of the stomach and the medial third of the left kidney. The comma shaped soft tissue density of the spleen is present in the upper right. A few barium coated gastric folds are visible immediately anterior to it. These gastric folds are in the most posterior portion of the stomach and are separated from the spleen by adipose tissue. Immediately beneath this most posterior portion of the stomach are the

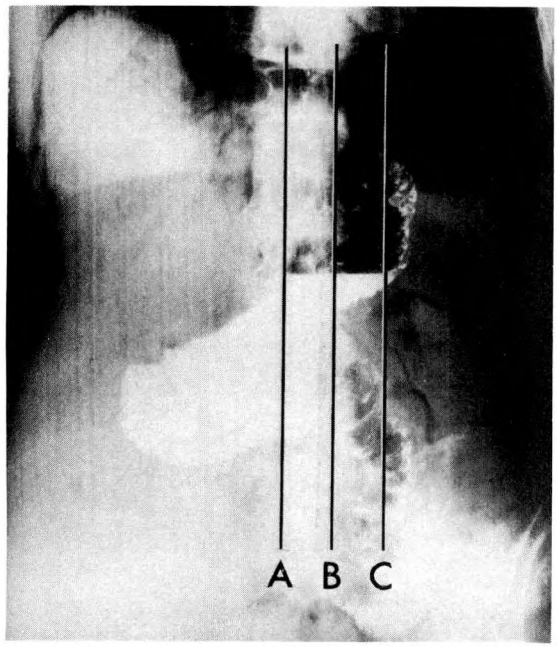


FIG. 2. Planes through which the sagittal sections of cadaver were made. These sections were then photographed and roentgenographed. (See Fig. 3, 4 and 5.)

splenic vessels. These are visible on the roentgenogram because of the calcium present within the walls as well as the contrast of the soft tissue density to the surrounding fatty tissue. These vascular structures are responsible for the contour of the posterior portion of the cardia inferiorly. Varying amounts of adipose tissue may increase the distance between the splenic vessels and the stomach. The remainder of the inferior contour of the proximal stomach is related to the distal transverse colon and the adipose tissue above it. The left kidney is positioned below the spleen. Note the great amount of perirenal fat which is responsible for a large mass effect within the abdomen.

Using this sagittal section technique it is possible to account for more medial gastric contours. Figure 4, *A* and *B*, is a section medial to the spleen, through the region of the ligament of Treitz. The margination of the posterior gastric wall by the spleen and splenic vessels is again present. In addition, below the splenic vessels, the juxtaposition

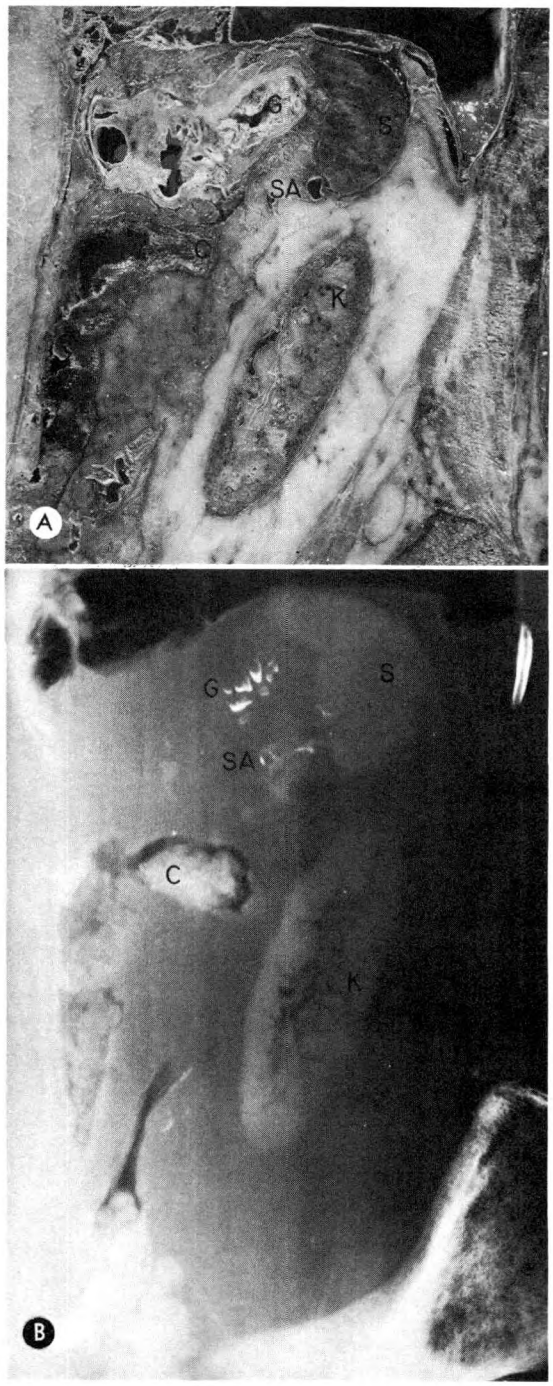


FIG. 3. (*A*) Photograph and (*B*) roentgenogram of section through medial aspect of spleen, most lateral portion of stomach and medial third of the left kidney. S-spleen; G-barium coated gastric folds; S.A.-calcification in splenic artery and its branches; C-colon; K-kidney. The photograph allows identification of the structures responsible for the contours of the stomach.

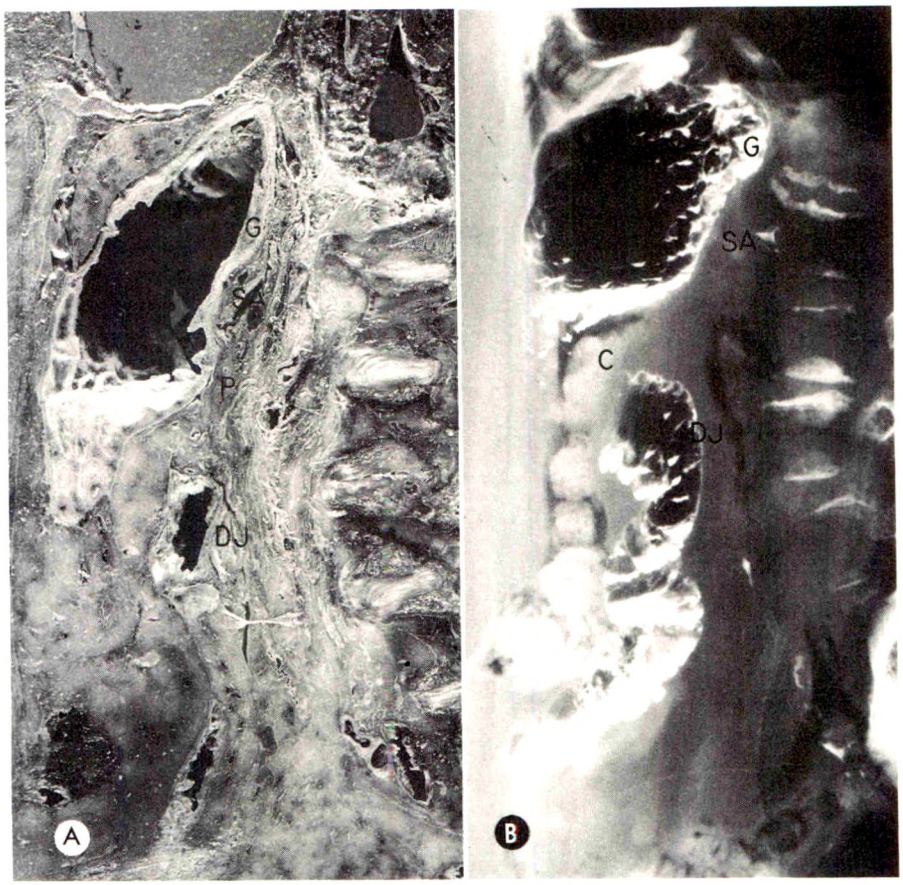


Fig. 4. (*A*) Photograph and (*B*) roentgenogram of section medial to the spleen, through the region of the duodeno-jejunal junction. S.V.-splenic vessels; G-stomach; D.J.-duodeno-jejunal junction; P-tail of pancreas. The photograph demonstrates those structures that are responsible for the contours of the stomach.

of the tail of the pancreas and the posterior margin of the stomach can be appreciated. The remainder of the posterior and inferior shape of the stomach is related to the duodenum and jejunum at the ligament of Treitz, with its surrounding connective tissue, and the distal transverse colon. Superiorly the most lateral segment of the left lobe of the liver produces an indentation upon the anterosuperior margin of the stomach.

To evaluate the retrogastric space in a lateral view it is imperative to know which structures are in the same sagittal plane. Figure 5, *A* and *B*, is another photograph and roentgenogram of a sagittal section through a cadaver in an attempt to identify those structures in the retrogastric space that are intimate to each other, and thus demonstrate the usefulness of corresponding anteroposterior and lateral roentgenograms in determining which structures are in the same sagittal plane. Figure 5, *A* and *B*, is a midline sagittal section including the mid portion of the stomach, the fourth portion of the duodenum and the left lobe of the liver. Observe in the midline the close approximation of the posterior wall of the stomach to the aorta. Calcium in the wall of the aorta and gas in the lumen of the inferior vena cava allow their identification. The soft tissue and calcium densities of the splenic vessels can again be identified and are the cause of the concave contour to the posterior portion of the stomach at the junction of its upper and middle thirds.

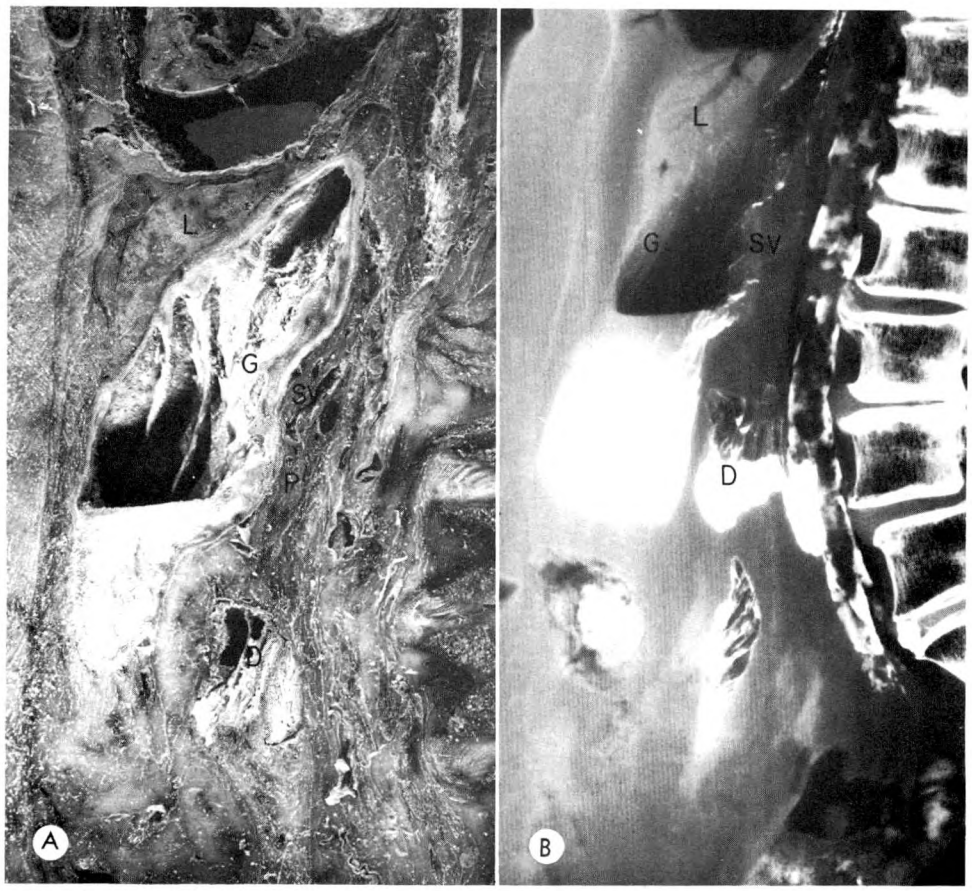


FIG. 5. (*A*) Photograph and (*B*) roentgenogram of section through the mid sagittal plane. G-stomach; L-left lobe of liver; D-fourth portion of duodenum; S.V.-splenic vessels; P-pancreas. Note that on the photograph the shape and position of the stomach are dependent upon the structures intimate to it.

The pancreas is again noted immediately inferior to the splenic vessels and posterior to the stomach. A large concavity to the posterior contour of the stomach inferiorly is produced by the fourth portion of the duodenum and the surrounding adipose tissue. The duodenum is separated from the vertebral bodies by the width of the aorta and inferior vena cava.

CLINICAL ROENTGENOGRAMS

With the experience gained from evaluation of the photographs and roentgenograms of the sagittal sections described above, normal clinical roentgenograms were examined in an attempt to evaluate the retrogastric space.

Figure 6 is a series of examinations of the abdomen following renal and upper gastrointestinal tract opacification, in left lateral (*A* and *B*), supine (*C* and *D*) and right lateral (*E* and *F*) positions. The changes in position of the stomach in these different views is evident. It then follows that the structures that lie in the retrogastric space also change as position is altered. A lateral roentgenogram of the abdomen for evaluation of retrogastric structures must be interpreted with the corresponding frontal view to permit identification of those structures that actually are behind the stomach. It is important to realize that these structures will change as the position at the time of examination changes.

Figure 6, *A* and *B*, is a frontal and lateral view of the abdomen with the subject lying

on the left side. The left kidney maintains its normal position while the right kidney has been displaced downward. The anteroposterior view allows identification of those structures that actually lie behind the stomach. Roentgenographically these are primarily the spleen and the left kidney. The splenic vessels entering the hilus of the spleen are responsible for a concave contour to the stomach along the high lesser curvature. Since in this view the stomach is actually lateral to the hilus of the spleen, it does not directly relate to the tail of the pancreas, and thus a mass in the pancreas could not produce a defect on the posterior wall of the stomach. Two prominent contours of the posterior wall of the stomach are present. When comparison is made to the frontal view it is evident that the superior of the posterior gastric contours is produced by the spleen and the inferior contour is produced by the left kidney. The opacified pyelocalyceal system of the left kidney is visualized immediately adjacent to this inferior gastric contour.

Figure 6, *C* and *D*, is a frontal and lateral examination in the supine position. The right kidney has returned to a normal position. The anteroposterior view again defines those structures in the retrogastric space. In addition to the spleen and the left kidney, the duodeno-jejunal junction is now intimate to the posterior gastric wall. Again the vessels in the hilus of the spleen are responsible for a medial contour to the proximal stomach in the frontal view. The tail of the pancreas remains medial to the stomach. The lateral projection demonstrates the distal duodenum and proximal jejunum as well as the spleen and left kidney to be responsible for the contour of the posterior wall of the stomach.

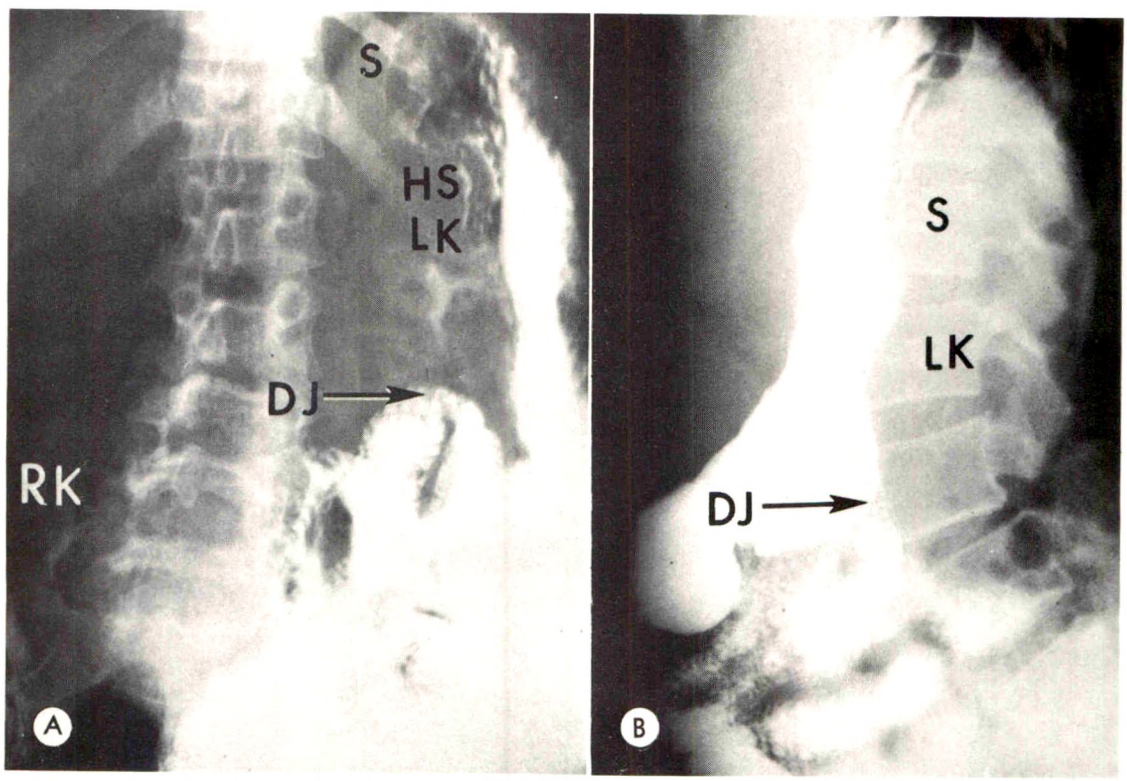


Fig. 6. Clinical roentgenograms with opacification of the upper gastrointestinal tract and kidneys. (*A*) Left decubitus and (*B*) left lateral views of the abdomen. The left kidney (LK) is normal in position. The right kidney (RK) has been displaced downward. The anteroposterior view demonstrates the structures behind the stomach. The lateral view allows evaluation of the retrogastric space. S-spleen; D.J.-duodeno-jejunal junction; H.S.-splenic hilum. A slight spondylolisthesis is present at L_3.

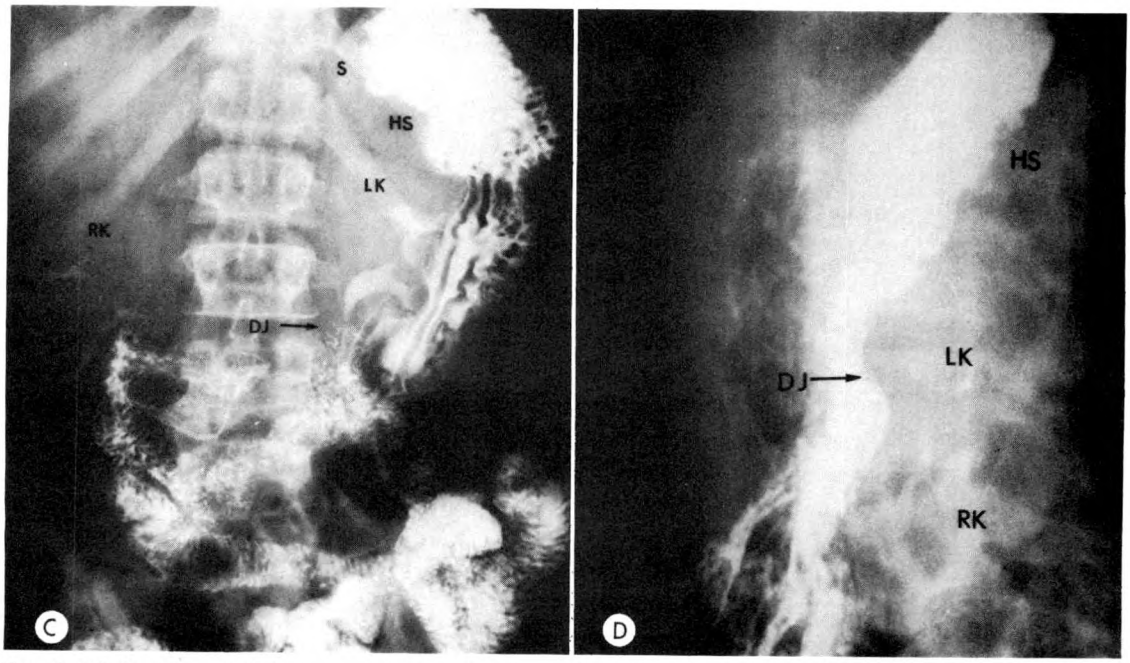


FIG. 6. (*C*) Supine and (*D*) cross table lateral views of the abdomen. The left kidney (LK) and right kidney (RK) are normal in position. The anteroposterior view again demonstrates those structures behind the stomach. This allows for evaluation of the retrogastric space in the lateral view. Note that the stomach does not overlie the tail of the pancreas and therefore a mass in this region would have no effect upon the retrogastric space. S-spleen; D.J.-duodeno-jejunal junction; H.S.-splenic hilum.

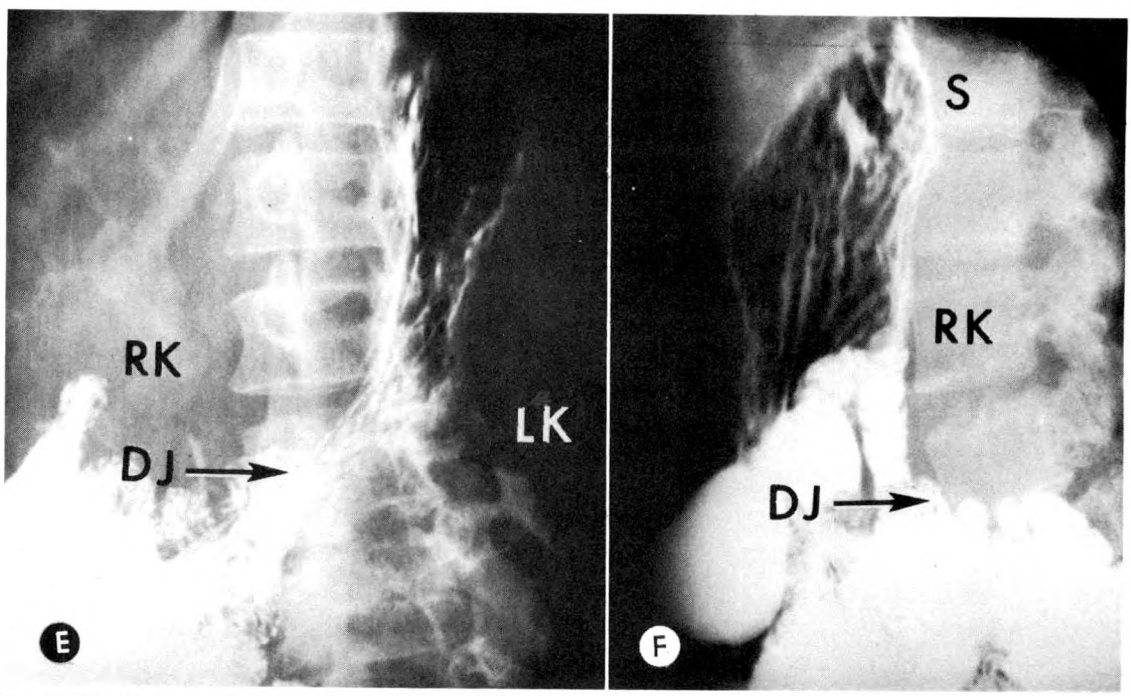


FIG. 6. (*E*) Right decubitus and (*F*) right lateral views of the abdomen. The left kidney (LK) has been displaced downward. The right kidney has been displaced slightly superiorly. The stomach relates to the left kidney for only a short segment (arrow). The majority of the posterior gastric contour is related to the spine. A slight concavity to its contour is noted in the region of the tail of the pancreas.

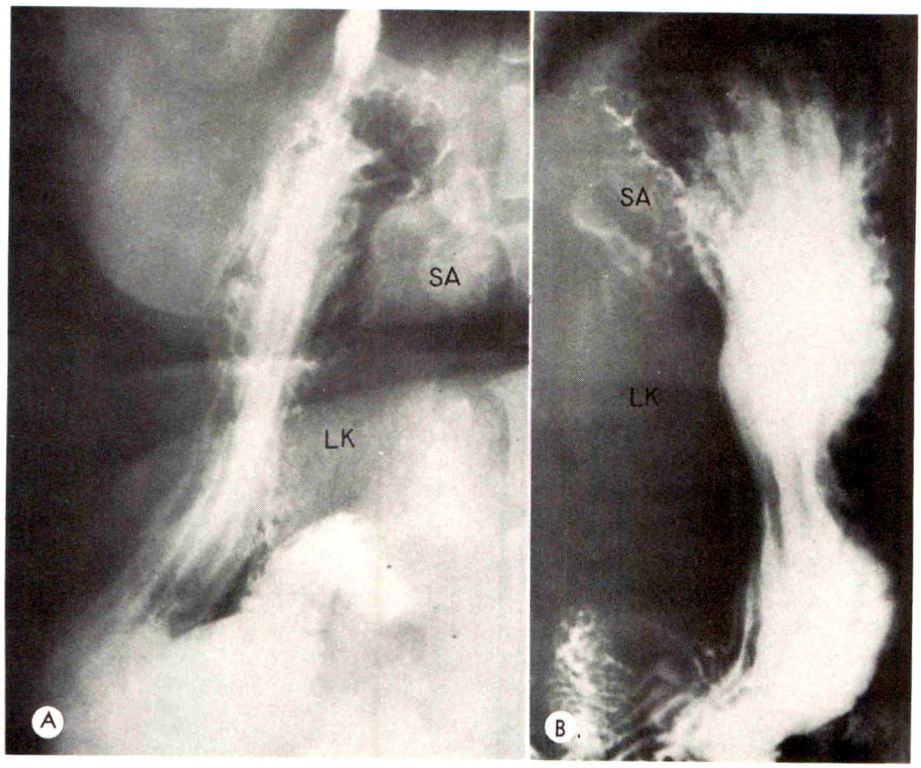


Fig. 7. Clinical roentgenograms with opacification of the upper gastrointestinal tract. A calcified tortuous splenic artery (SA) is present. It is responsible for a concavity to the gastric contour in both (*A*) the anteroposterior and (*B*) lateral views. The position of the left kidney (LK) is identified on the frontal view and can be inferred in the lateral view where an appropriate indentation upon the posterior wall of the stomach is present. (Courtesy of Fred Ziter, M.D., and Jack Westcott M.D., Hartford, Conn.)

Figure 6, *E* and *F*, is a corresponding examination in the right lateral position. The left kidney is now displaced downward. The stomach is more to the right than on the previous examinations of this series. This change in position has produced a change in the structures included in the retrogastric space. It is evident in the anteroposterior view that the stomach is overlying the medial aspect of the spleen, the tail of the pancreas, the mid lumbar spine, the superior pole of the left kidney and the duodeno-jejunal junction. It is these structures that in the lateral view will relate to the posterior wall of the stomach and be responsible for the position of the stomach in the abdomen.

Calcification of a very tortuous splenic artery (Fig. 7, *A* and *B*) provides another opportunity to examine the relationship of the splenic hilum to the stomach. The concavity to the medial aspect of the proximal stomach demonstrated in Figures 6, *A* and *B*, and 6, *C* and *D*, is again present. The close relationship of the splenic artery to this gastric contour is observed in the anteroposterior view. A concave contour to the posterior wall of the stomach is present on the lateral examination. It is related to the calcified splenic artery. This relationship was also noted in the postmortem roentgenograms. There is some separation of the calcified splenic artery and the gastric wall presumably due to interposed adipose tissue. The impression of the left kidney upon the posterior gastric wall identified in Figure 6 as well as in the postmortem roentgenograms is again demonstrated.

CONCLUSION

An attempt has been made to define the anatomic structures which may indent the

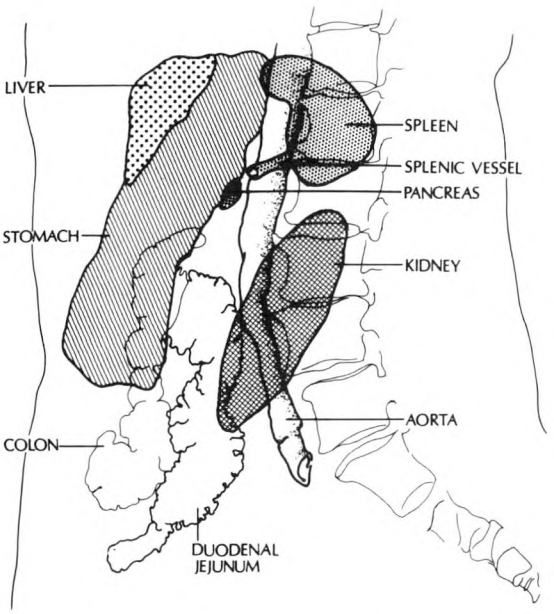


FIG. 8. A composite diagram of the structures identified in the separate sagittal sections of Figures 3, 4 and 5. Superimposition of these structures upon the lateral roentgenogram of Figure 1 makes it possible to account for the contour of the stomach and for the position of the stomach in the abdomen.

posterior surface of the stomach and to demonstrate how the contour of the posterior gastric wall may be related to these structures varying with its position from left to right in the abdomen. Frontal and lateral views of the abdomen with the subject in the same position are necessary to evaluate the position of the stomach.

Certain normal less well-known indentations do occur which should not be mistaken for pathology: notably the duodenojejunal junction and its related fat; the vessels of the hilum of the spleen; and the posterior portion of the splenic substance.

These relationships are summarized in Figure 8.

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REFERENCES

1. ENGLE, A., and LYSHOLM, E. New roentgenological method of pancreas examination and its practical results. *Acta radiol.*, 1934, *15*, 635.
2. POOLE, G. J. New roentgenographic method of measuring retrogastric and retroduodenal spaces: statistical evaluation of reliability and diagnostic utility. *Radiology*, 1970, *97*, 71–81.
3. POPPEL, M. H., SHEINMEL, A., and MEDNICK, E. Procurement and critical appraisal of width diameter of midline retrogastric soft tissues. AM. J. ROENTGENOL. & RAD. THERAPY, 1949, *61*, 56.
4. ROTH, M. Routine roentgen diagnosis of retrogastric expansive processes with special reference to extra capital parts of pancreas. *Acta radiol.* (Diag.), 1963, *4*, 277–297.
5. TAVERAS, J. M., and GOLDEN, R. Golden's Diagnostic Roentgenology. Williams & Wilkins Company, Baltimore, 1964.
6. WHALEN, J. P., EVANS, J. A., and MEYERS, M. A. Vector principle in differential diagnosis of abdominal masses. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *115*, 318–333.

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"LOWER" GASTROINTESTINAL TRACT HEMORRHAGE: NEW CONCEPTS BASED ON ARTERIOGRAPHY*

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THE patient with severe rectal hemorrhage presents a serious diagnostic and therapeutic dilemma. In contradistinction to acute upper gastrointestinal tract hemorrhage, endoscopy and the usual barium sulfate roentgenographic examinations rarely result in the precise localization of the bleeding point. Preoperative diagnostic imprecision is often compounded by difficulty in finding the bleeding site in the operating room or even ultimately in the resected specimen.

These factors have resulted in a lack of clinical understanding of lower gastrointestinal tract hemorrhage and in nonspecific surgical remedies.

Selective arteriography has proven to be extremely useful in the management of gastrointestinal tract hemorrhage.[1,13,16] In acute rectal hemorrhage no other diagnostic modality can achieve the precision and reliability of arteriography in finding the offending lesion.[4,5,8] We are herein presenting an analysis of our experience with 60 consecutive cases of severe clinical lower gastrointestinal tract hemorrhage studied over a 5 year period.

MATERIAL AND METHOD

Patients included in the series all had persistent, life threatening hemorrhage for which surgical intervention was being considered. All patients selected for arteriography were thought to be actively bleeding and transfusion of at least 2 units of blood had already been given or were being given to maintain vital signs or hematocrit. Attempts to further quantitate bleeding rates before arteriography are extremely inaccurate in rectal hemorrhage and are not useful as criteria for patient selection.

Gastric aspirates through a nasogastric tube were negative for blood in all cases. Proctoscopy had already been performed in 51 cases and was uniformly unrewarding. For 40 patients the acute episode represented their first significant hemorrhage. Seven had hemorrhaged once before and 13 had suffered multiple bleeding episodes. Eight patients had undergone previous surgery for similar attacks of acute lower gastrointestinal tract hemorrhage. The vast majority of patients had no significant previous gastrointestinal disease or symptoms other than bleeding, but 5 had a relevant history of abdominal trauma or known chronic gastrointestinal illness. None had a history of ulcerative colitis or other inflammatory disease.

Arteriography was performed on an emergency basis with constant electronic monitoring of pulse and blood pressure during the procedure. Support from the medical and surgical staff in caring for the patient was essential. Selective arteriographies were performed with a variety of different catheters introduced by the standard Seldinger technique.

Our present procedure is a result of 5 years of evolution and consists of catheterizing the femoral artery with a thin walled No. 6.7 French curved polyethylene catheter. The colon is filled with air through a previously placed rectal tube. A superior mesenteric arteriography is performed using 50 ml. of a 76 per cent solution of

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TABLE I

CAUSES OF ACUTE RECTAL HEMORRHAGE

Colonic diverticula	17
Arteriovenous malformation	4
Aortic stenosis and arteriovenous malformation of right colon	3
Duodenal ulcer	4
Carcinoma of colon	3
Meckel's diverticula	2
Jejunal diverticula	2
Arteriovenous fistula	2
Peutz-Jeghers syndrome	1
Rectal trauma	1
Ischemic colitis	1
Total Positive Studies	40
Total Negative Studies	20
Total Cases	60

sodium and methylglucamine diatrizoate. Serial filming is carried out for a period of 30 seconds and visualization of the entire distribution of the superior mesenteric artery is verified. We then proceed to a celiac artery study if the first injection is unrewarding. The subsequent inferior mesenteric study usually requires a catheter of different shape and also necessitates 2 injections to capture both the pelvic and splenic flexure portions of the artery on the usual 14 by 14 inch field. If all studies are negative, and the patient continues to actively hemorrhage, a repeat superior mesenteric arteriography is performed. When extravasation of contrast material into the bowel lumen is identified, a selective infusion of vasopressin 0.1 to 0.4 units/min. is begun. Infusion is continued for 30 minutes in the angiography suite and a follow-up study is performed to ascertain the degree of hemostasis obtained. The infusion may then be continued in the patient's room utilizing a portable constant infusion pump.

RESULTS

Of 60 patients studied in this manner, the bleeding lesion was demonstrated in 40. The relative frequency of the causes of lower gastrointestinal tract hemorrhage is summarized in Table I.

All but 1 of these 40 patients with positive arteriograms were ultimately operated on with surgery directed at removal of the specific bleeding point. In the remaining patient massive bleeding from a stress ulcer of the duodenum was permanently controlled by vasopressin infusion and no surgery was required. Six recent cases of colonic or jejunal bleeding points were also treated with vasopressin. Elective surgery was carried out in 3 after hemostasis was achieved. In the 3 others, persistent, brisk bleeding necessitated emergency surgery.

In most of these cases, the surgeon was unable to find the bleeding point at exploration, but bowel resections were performed on the basis of angiographic data. To date, there has been no known significant rebleeding in any of these patients. There were 2 operative deaths. Of the 20 negative cases, 10 patients stopped bleeding spontaneously and were not explored. In retrospect it appeared that many of these patients had stopped bleeding before arteriography was performed. The remaining 10 continued to hemorrhage and surgery was required; in 5 instances peptic ulcers of the duodenum were found and resected. In the other 5 no bleeding point could be found. Three patients underwent empiric left colectomies. Two continued to hemorrhage massively and ultimately expired in the immediate postoperative period. One had multiple colotomies and died of postoperative septicemia. The other patient had a right colectomy and has not re-bled (Table II).

CAUSES OF "LOWER" GASTROINTESTINAL TRACT HEMORRHAGE

COLONIC DIVERTICULOSIS

Colonic diverticula are by far the most common cause of rectal hemorrhage in the middle-aged or elderly adult.[12,14,15,20] The 17 patients with bleeding colonic diverticula ranged from 55 to 84 years of age and all presented with the acute onset of painless rectal hemorrhage. None of the patients had clinical signs of diverticulitis.

The angiographic appearance of bleeding

colonic diverticula is characteristic. Extravasation of contrast material begins in the early arterial phase. The opaque contrast material pools within the confines of the saccular diverticulum and frequently persists as a sharply marginated collection throughout the venous phase of the study. No arteriovenous shunting or neovascularity is seen, and the only visible abnormality is actual extravasation of contrast material. Barium enema examinations succeed only in showing the multiplicity of diverticula without recognizing the bleeding lesion (Fig. 1, *A–D*).

Diverticular bleeding may also be intermittent in nature. Therefore, a negative study can be followed in a few minutes by a positive one. This finding had led us to perform repeat injections if we suspect that active bleeding persists, and a site has not been established.

Although only single diverticula were bleeding in 15 of 17 cases, 1 case had simultaneous bleeding from 2 adjacent diverticula. In another patient with uremia and severe coagulation deficiencies, 3 bleeding diverticula were identified.

Although previous barium enema examinations interfere with arteriography and are not recommended, they do not completely obviate its usefulness. Contrast material can be identified in and around barium filled diverticula if most of the barium in the lumen has been eliminated by the strong cathartic action of intraluminal blood.

Even though most diverticula are in the sigmoid or left colon, the distribution of bleeding points in colonic diverticula is strongly weighted toward the right side. All but 1 of the bleeding points occurred in the region from the splenic flexure to the cecum (Fig. 2, *A* and *B*; 3, *A* and *B*; and 4).

TABLE II

ARTERIOGRAPHIC FINDINGS AND FOLLOW-UP RESULTS

Follow-up of Negative Arteriograms		
Stopped bleeding, no treatment		10
Surgery required		10
Duodenal ulcer	5	
Empiric left colectomy (2 deaths)	3	
Multiple colotomies (1 death)	1	
Empiric right colectomy	1	
Total Cases		20
Follow-up of Positive Arteriograms		
Preoperative control of bleeding with vasopressin		7
Surgery directed at specific lesion		39
Operative deaths		2
Re-bleeding		0
Total Cases		40

JEJUNAL DIVERTICULA

Jejunal diverticula were the source of acute rectal hemorrhage in 2 patients. These lesions had the identical angiographic appearance of colonic diverticula except that their blood supply was derived from jejunal branches of the superior mesenteric artery. Although these lesions also occur in the older age group, they are much rarer than colonic diverticula. Hemorrhage was thought to be a rare complication of jejunal diverticulosis and there were only 17 cases reported by 1958.[17]

When hemorrhage occurs it may either simulate peptic ulceration with an antecedent history of dyspepsia or present as rectal hemorrhage indistinguishable from colonic diverticular bleeding. A local jejunal resection was curative in our 2 patients.

MECKEL'S DIVERTICULUM

Young, previously healthy patients who presented with acute rectal hemorrhage were bleeding from Meckel's diverticula in 2 cases. The angiographic findings consisted of pooling of radiopaque material in the diverticulum which is supplied by a branch of the ileocolic artery. It should be recognized, however, that the bleeding point may not be within the diverticulum itself since ulceration of the ileal mucosa opposite the opening of the diverticulum is frequently the site of blood loss.

These lesions may also be detected on a scan of the abdomen following an injection of Tc^{99m} pertechnetate which collects in the

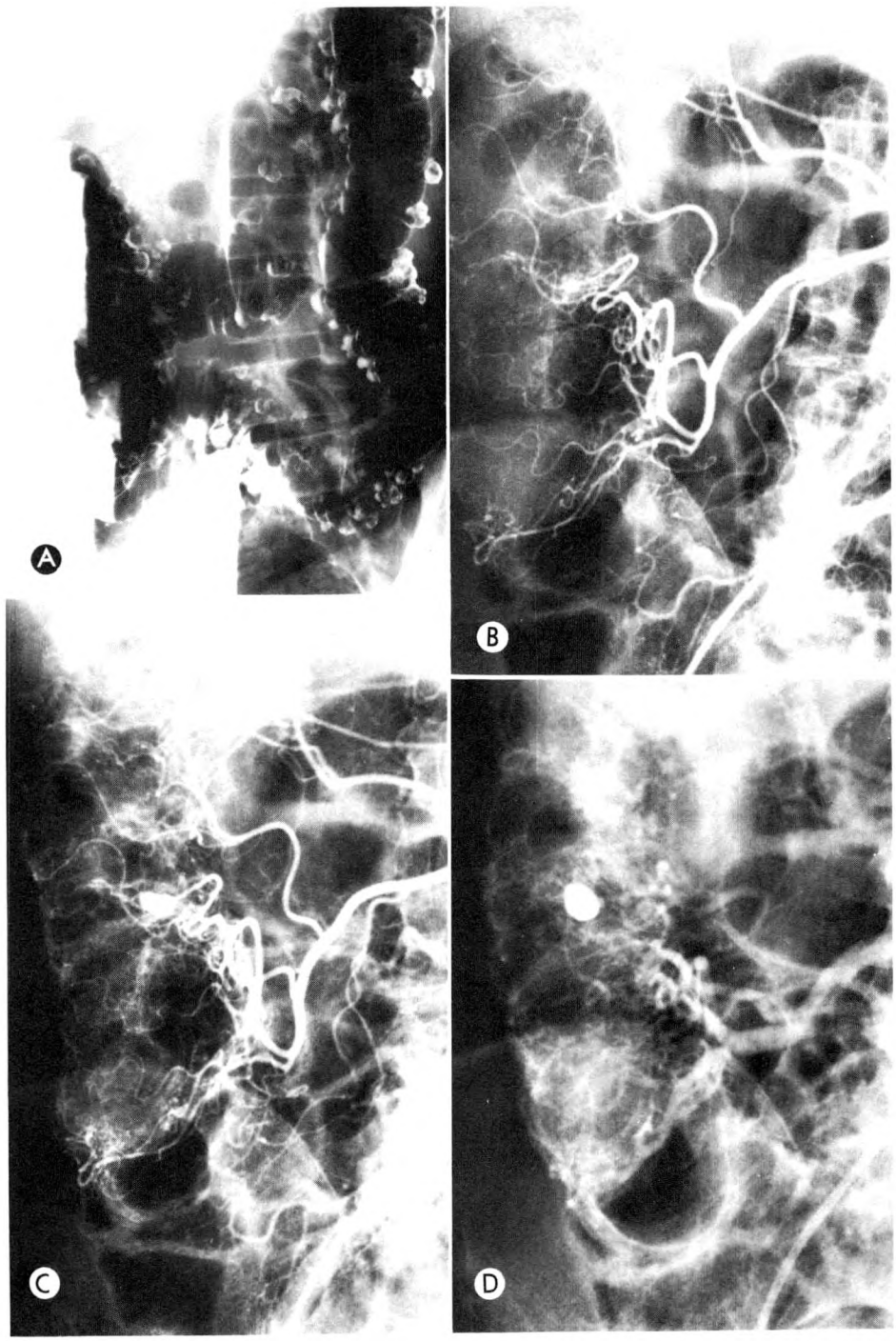


Fig. 1. (*A*) A previous barium enema roentgenogram in a 67 year old man with acute rectal hemorrhage shows only a multiplicity of colonic diverticula. (*B*) Arterial and (*C* and *D*) venous phases of a superior mesenteric arteriography show the typical appearance of a bleeding colonic diverticulum. Contrast material extravasates into the diverticulum and pools within it, revealing a persistent, sharply marginated density throughout the venous phase.

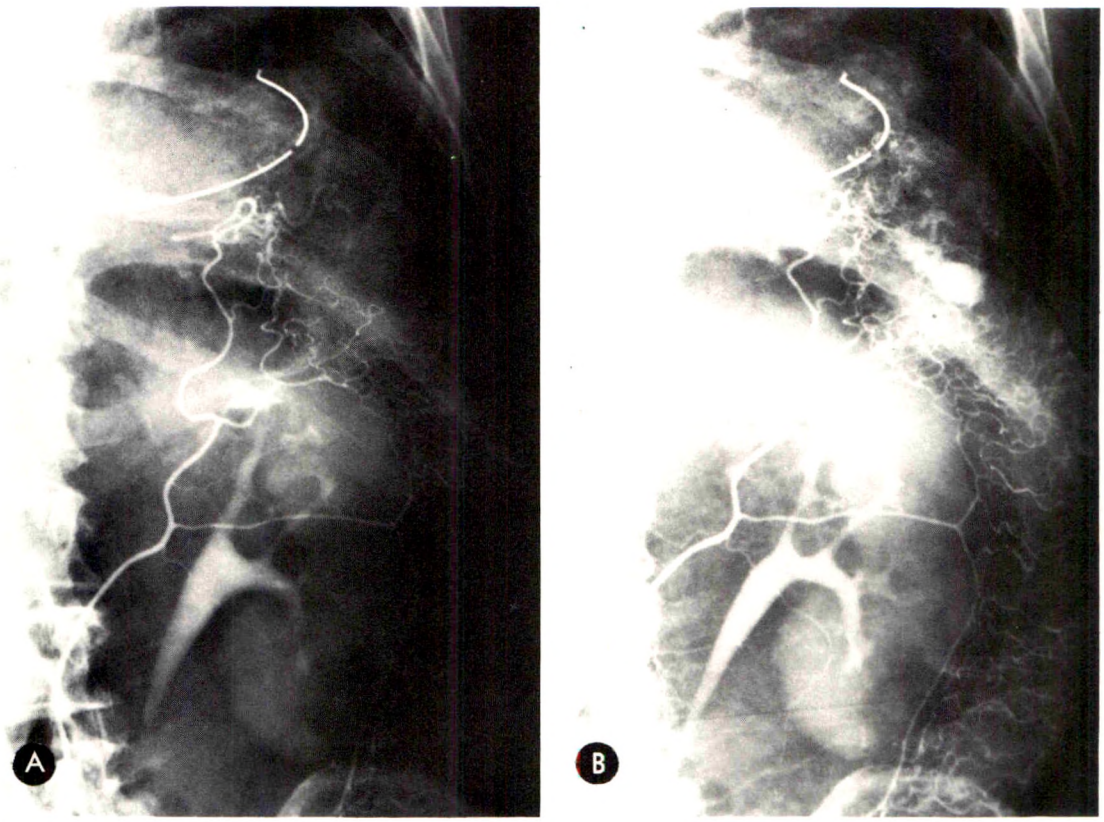


Fig. 2. (*A* and *B*) A splenic flexure diverticulum was the cause of hemorrhage in this 58 year old man and is demonstrated on these inferior mesenteric arteriograms. Separate studies of the pelvic and left upper quadrant distributions of the inferior mesenteric artery are essential to view the entire distribution of this vessel.

ectopic gastric mucosa in the diverticulum. Barium studies are generally unrewarding.

CARCINOMA OF THE COLON

Although a frequent cause of occult, chronic blood loss, carcinoma rarely presents as acute rectal hemorrhage. The diagnosis can be made by arteriography, however, and the findings are quite different from those of diverticula. A neovascular mass with vascular encasement may be seen.

In 1 case of carcinoma of the cecum, irregular extravasation into a cecal mass was identified. A barium enema examination confirmed the presence of a cecal carcinoma.

A case of metastatic carcinoma of the appendix presented as acute lower gastrointestinal tract hemorrhage when the lesion eroded into the iliac artery and produced an arterioenteric fistula. The third case of carcinoma was in the sigmoid colon and could be clearly seen as a huge neovascular mass without discrete extravasation (Fig. 5, *A* and *B*). No carcinomas were left undetected in our series. None were found on later barium studies or in the operating room.

ARTERIOVENOUS FISTULA

Acute rectal bleeding was caused by arteriovenous fistula in 2 cases. One patient was a 66 year old woman with an unsuspected postoperative iatrogenic fistula from the middle colic artery to vein. The fistula followed a closure of a transverse colostomy performed 6 weeks previously for obstructing sigmoid diverticulitis. Except for the history of recent surgery, the patient's

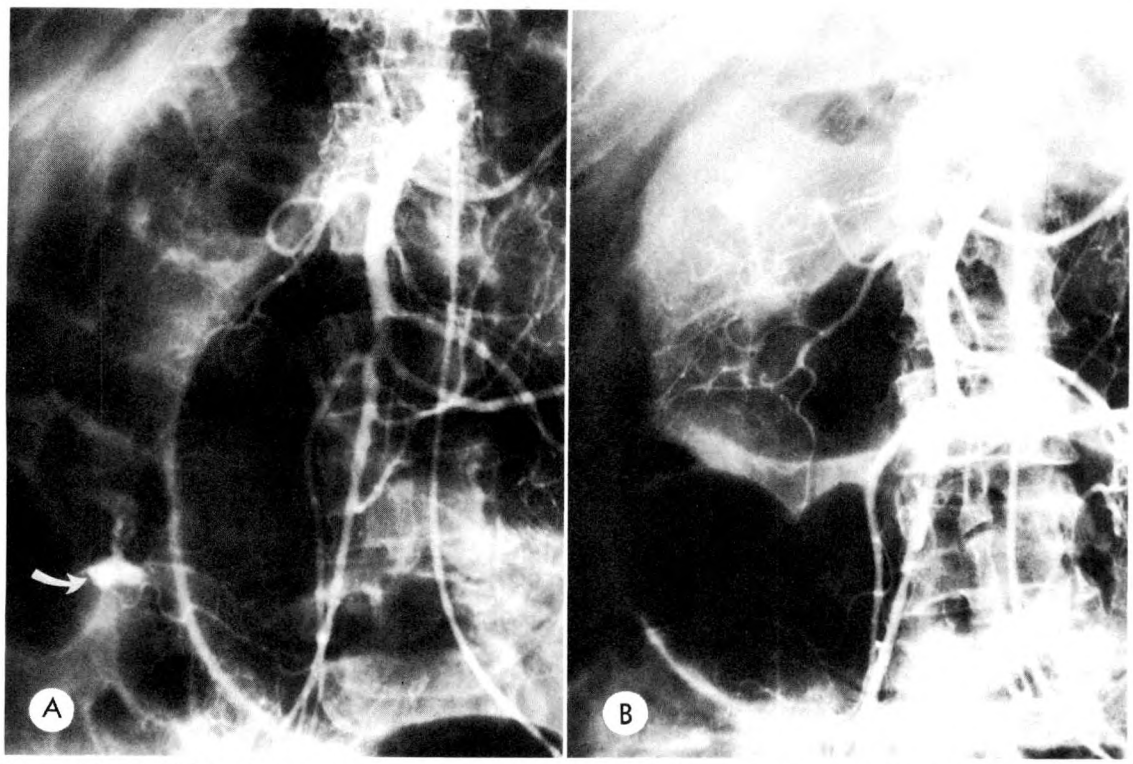


FIG. 3. (*A* and *B*) Superior mesenteric arteriograms reveal a bleeding diverticulum of the right colon (arrow). Following infusion of vasopressin into the superior mesenteric artery, hemostasis was achieved.

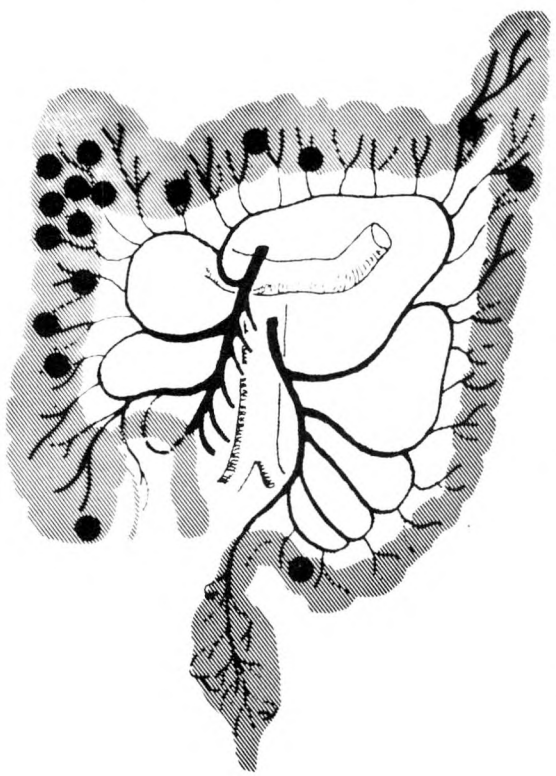


FIG. 4. The distribution of bleeding colonic diverticula is depicted in this drawing of the colon. Each black dot represents a single case of a bleeding diverticulum. The preponderance of right-sided bleeding points is evident.

symptoms were identical to those of diverticular bleeding (Fig. 6).

A year after a gunshot wound of the abdomen, rectal hemorrhage developed in a 20 year old man. A superior mesenteric artery-to-vein fistula was discovered on arteriography. A follow-up study after repair of the lesion demonstrated a persistent false aneurysm of the superior mesenteric artery (Fig. 7).

In both of these cases an actual extravasation of contrast material was not identified, but the early appearance of the portal venous system and the presence of false aneurysms confirm the diagnosis.

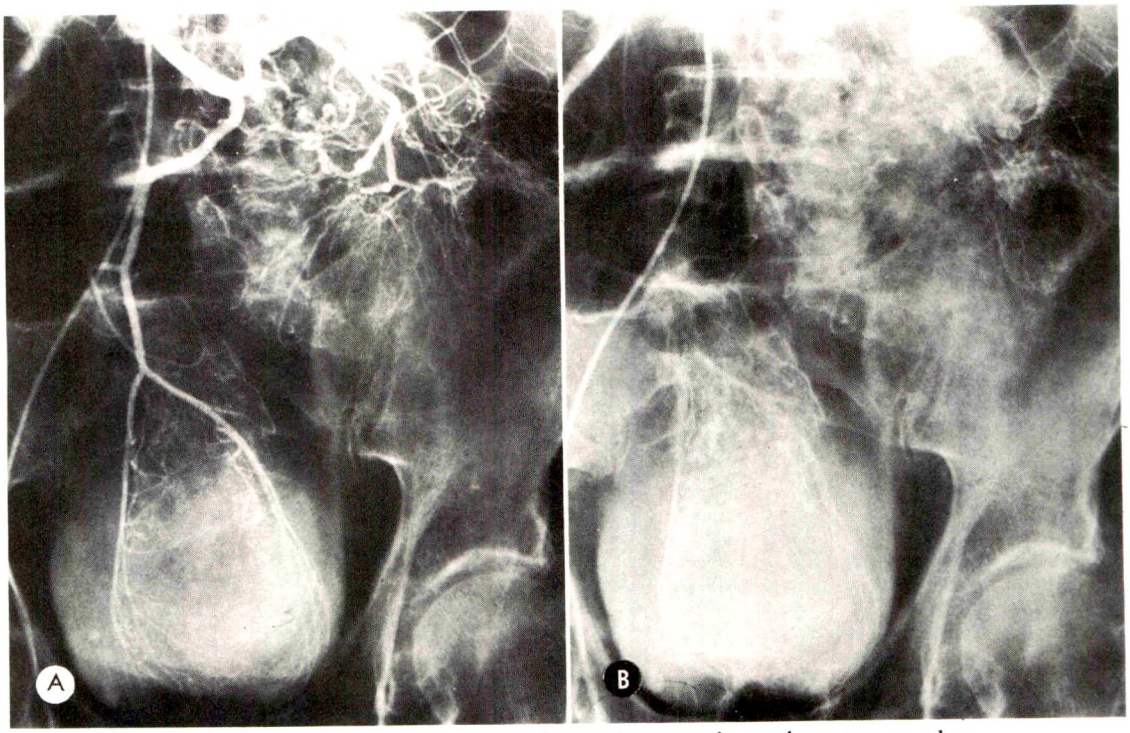


Fig. 5. (*A* and *B*) Inferior mesenteric arteriograms show a huge neovascular mass in the sigmoid colon due to a bleeding carcinoma.

ARTERIOVENOUS MALFORMATIONS

In 7 cases of rectal hemorrhage, arteriovenous malformations of the bowel were found. One was in the jejunum, 5 in the right colon, and 1 in the rectum. These lesions were in the submucosa and were all extremely difficult to identify at surgical exploration. Pathologic confirmation is also difficult unless painstaking injections of vessels followed by careful sectioning are carried out. In only 3 of the cases was actual extravasation seen on arteriography. The patients generally had a history of recurrent, moderate bleeding rather than a single episode of exsanguinating hemorrhage often seen when a diverticulum bleeds. The exception to this generality was the case of an 84 year old woman who suffered massive hemorrhage from a rectal arteriovenous malformation which was apparently spontaneous in origin.

An interesting aspect of these cases is an apparent association between arteriovenous malformation of the right colon and aortic stenosis in elderly patients. In 3 such cases small malformations of the lateral wall of the right colon were found. Right colectomies have resulted in cessation of bleeding in all patients. The malformations are difficult to identify on routine studies and magnification roentgenograms with air in the cecum may be required to make the diagnosis[10] (Fig. 8, *A* and *B*). The mechanism of formation of the arteriovenous malformations is unclear. Since the aortic stenosis is frequently isolated and congenital in origin, the vascular malformations are thought by some investigators to be congenital.[3] Others, however, suggested that these are shunts opened in response to the low perfusion pressure in the splanchnic bed.[31]

DUODENAL ULCER

Although it is widely believed that a negative gastric aspirate effectively rules out an upper tract source of acute rectal hemorrhage, this has not been our experience.

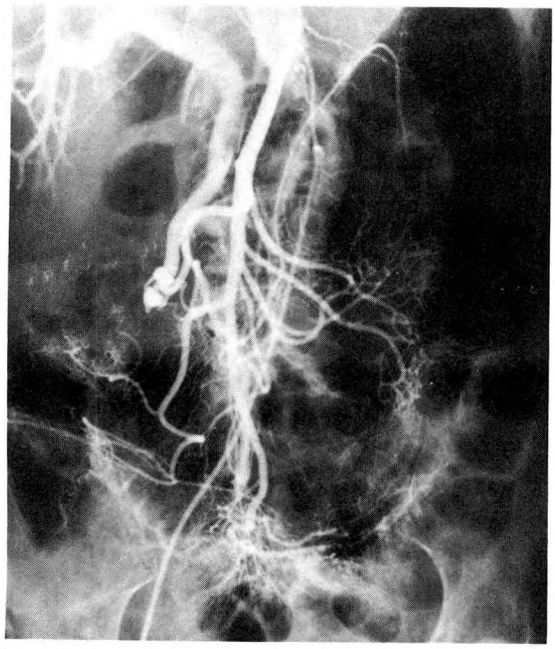


FIG. 6. An arteriovenous fistula between the middle colic artery and vein is well demonstrated on this superior mesenteric arteriogram. This iatrogenic fistula followed a transverse colostomy for obstructing diverticulitis and presented as painless rectal hemorrhage.

Massively bleeding duodenal ulcers were angiographically demonstrated in 4 patients who presented as typical lower gastrointestinal hemorrhage. All 4 had had negative gastric aspirates through nasogastric tubes and unrewarding endoscopy. These cases have prompted us to diligently study the duodenum if the bleeding site remains occult after superior and inferior mesenteric arteriographies. In 1 case, a 29 year old female with massive lower gastrointestinal hemorrhage, a bleeding stress ulcer of the duodenum was permanently controlled by selective vasopressin infusion with complete healing of the ulcer on follow-up barium enema study 10 days later (Fig. 9, *A* and *B*).

In 5 of our early negative cases, upper gastrointestinal peptic ulcers were found at surgery. In 2 of these cases celiac arteriograms were negative. The other 3 did not have celiac or gastroduodenal studies. It is interesting to note that in 9 patients out of a total 60 in the group, duodenal ulcers produced a clinical syndrome indistinguishable from lower gastrointestinal hemorrhage.

MISCELLANEOUS CAUSES OF RECTAL HEMORRHAGE

The preceding discussion is not intended to be a catalogue of all causes of rectal hemorrhage. Single cases of Peutz-Jeghers syndrome, local rectal trauma, and ischemic colitis were found in the series. Other rare causes have been reported previously and diagnosed by arteriography. Clinically known inflammatory diseases such as ulcerative colitis also can produce rectal bleeding, but these patients rarely come to arteriography.

DISCUSSION

It is apparent that angiography is clearly established as the diagnostic method of choice in acute lower gastrointestinal tract hemorrhage. Because no other diagnostic or therapeutic modality approaches the efficacy of arteriography in finding the etiology of rectal hemorrhage, its use has given us a

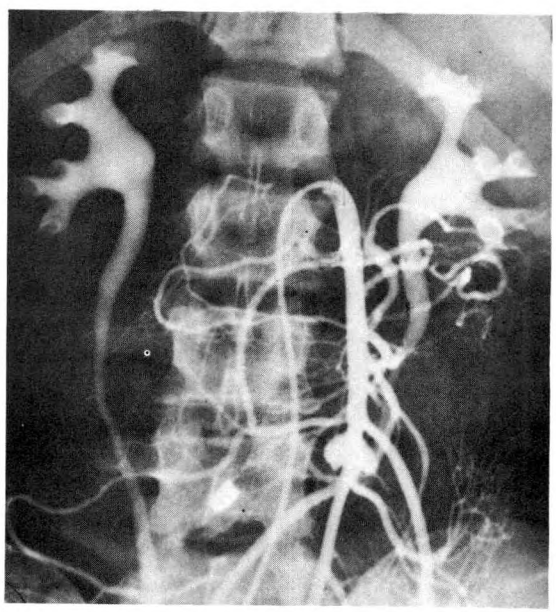


FIG. 7. A year after an abdominal gunshot wound, acute rectal hemorrhage developed secondary to an arteriovenous fistula between the superior mesenteric artery and vein. Following closure of the fistula, a false aneurysm of the superior mesenteric artery remains.

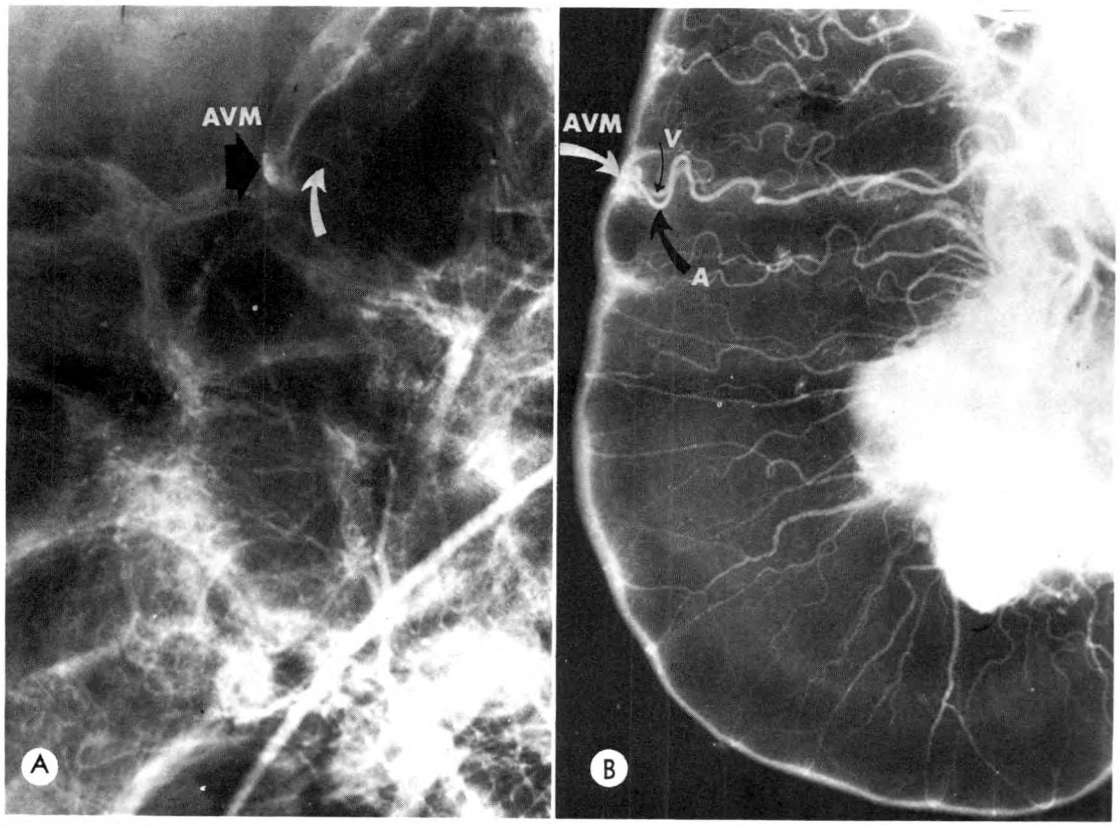


FIG. 8. (*A*) Magnification film from a superior mesenteric arteriogram in a patient with aortic stenosis and rectal hemorrhage reveals an arteriovenous malformation (AVM) of the lateral wall of the right colon (black arrow). An early draining vein (white arrow) is seen. (*B*) The lesion is confirmed on a specimen injection of the resected right colon. The enlarged feeding artery (black arrow) and vein (small black arrow) are more clearly identified.

new insight into the spectrum of pathology that can produce the clinical syndrome.

The use of selective vasoconstrictors has already contributed to the management of some of these severely ill patients.[2,23] Development of better transcatheter embolic and coagulation techniques should further accelerate this progress.

Although most bleeding lesions are in the colon, it is clear that a large number are located in the small bowel or duodenum. Eleven of 40 positive cases were located outside of the colon. The recently presented[6,18,22] surgical concept of routine total colectomy for these patients seems not only unwarranted, but can be frequently counterproductive. The preponderance of bleeding diverticula in the right colon should contraindicate the future employment of sigmoid colectomies in these patients. Because of the potentially disastrous consequences of empiric partial colectomies or multiple colotomies, a subtotal colectomy may be the procedure of choice in those few patients who require surgery without a preoperative diagnosis. Almost all lesions other than diverticula and ulcers have an angiographically recognizable pattern consisting of neovascularity or arteriovenous shunting, whether or not they are actively bleeding at the time of study. Most negative studies are probably due to intermittent or previous bleeding from 1 of these 2 causes. This conclusion is strengthened by the follow-up of our 10 negative patients who were not operated on and the 5 patients where peptic ulcers were found at surgery. Of the 10 negative, unoperated patients,

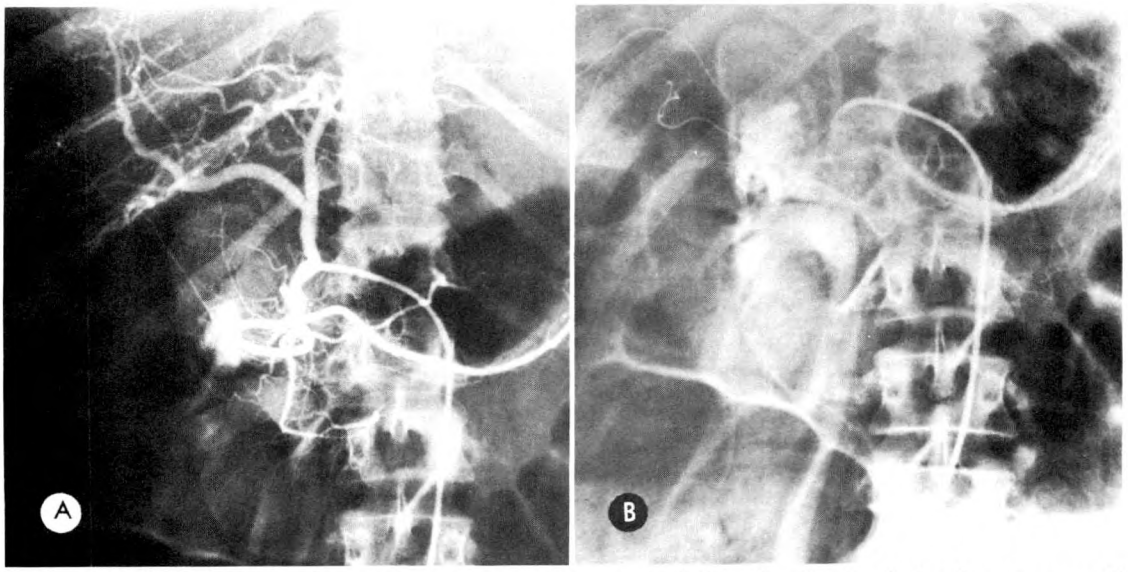


Fig. 9. (*A* and *B*) Gastroduodenal arteriograms reveal a massively bleeding duodenal ulcer in a 29 year old woman who presented with rectal hemorrhage. Infusion of vasopressin resulted in permanent hemostasis and obviated the need for exploratory surgery.

none have had known significant re-bleeding and nothing other than colonic diverticula were ever found on subsequent barium studies. Other intestinal lesions that cause hemorrhage, *i.e.*, tumors, vascular malformations, fistulas, inflammatory disease, and trauma, all have recognizable stable abnormalities of angioarchitecture which lead to the correct diagnosis without extravasation of contrast material on arteriography.

The association of aortic stenosis and arteriovenous malformation of the cecum appears to be a real one. It is known that patients with aortic stenosis frequently suffer from occult gastrointestinal blood loss,[31] and scattered case reports of right colon lesions have appeared in the literature.[3] The pathogenesis of these lesions is obscure, but may be on a congenital basis or be due to decreased perfusion pressure with subsequent opening of submucosal arteriovenous shunts. It has been productive to diligently search for these lesions in patients with aortic stenosis. Magnification techniques have been extremely helpful in making the diagnosis in 2 of our 3 cases.

A possible criticism of the use of arteriography in this clinical situation is that it accelerates the decision to operate and that some unnecessary bowel resections may be performed. It also appears that the vasodilatory effects of contrast material may accelerate the bleeding in some cases. It is true that demonstration of an active bleeding point to a surgeon often creates an irresistable urge to resect it. However, if arteriography is reserved for those patients who are already a therapeutic problem and for whom repeated transfusions are hazardous, this should not be a serious objection. The use of selective vasoconstrictors has led to more postangiography monitoring before surgery and has provided the opportunity to perform elective limited bowel resections with the patient's circulatory status stabilized. Future acceptance of transcatheter vasoconstriction as a primary mode of treatment depends only on further experience with the long term results of the procedure. Although the diatrizoates are vasodilators, their effect is very transient and the possibility of significant enhancement of hemorrhage seems unlikely.

It is our impression that the potential benefits of arteriography to these seriously

ill patients far outweigh the risks and hazards of the procedure, and that the procedure should be performed on an emergency basis before exploratory surgery is undertaken.

SUMMARY

1. Arteriography is the diagnostic method of choice in acute rectal hemorrhage and results in precise preoperative diagnosis and rational surgery. The potential for selective therapy through the catheter is also evident.

2. Colonic diverticulosis is, by far, the most common cause of acute rectal bleeding in the middle-aged or elderly adult.

3. Most bleeding colonic diverticula are located to the right of the splenic flexure.

4. Carcinoma is a rare cause of rectal hemorrhage.

5. The bleeding point is occasionally located outside of the colon.

6. A history of trauma or previous surgery should suggest the possibility of arteriovenous fistula.

7. Arteriovenous malformations of the right colon should be diligently searched for in elderly patients with aortic stenosis and recurrent hemorrhage.

8. Hemorrhage from duodenal ulcer may present as rectal hemorrhage and lead to diagnostic confusion.

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REFERENCES

1. Baum, S., Nusbaum, M., Blakemore, W. S., and Finkelstein, A. Preoperative radiographic demonstration of intra-abdominal bleeding from undetermined sites by percutaneous selective celiac and superior mesenteric arteriography. *Surgery*, 1965, *58*, 797–805.

2. Baum, S., Rösch, J., Dotter, C. T., Ring, E. J., Athanasoulis, C., Waltman, A. C., and Courey, W. R. Selective mesenteric arterial infusions in management of massive diverticular hemorrhage. *New England J. Med.*, 1973, *288*, 1269–1272.

3. Boss, E. G., and Rosenbaum, J. M. Bleeding from right side of colon associated with aortic stenosis. *Am. J. Digest. Dis.*, 1971, *16*, 269–275.

4. Casarella, W. J., Kanter, I. E., and Seaman, W. B. Right sided colonic diverticula as cause of acute rectal hemorrhage. *New England J. Med.*, 1972, *286*, 450–453.

5. Clark, R. A., and Rösch, J. Arteriography in diagnosis of large bowel bleeding. *Radiology*, 1970, *94*, 83–88.

6. Drapanas, T., Pennington, D. G., Kappelan, M., and Lindsey, E. S. Emergency sub-total colectomy: preferred approach to management of massively bleeding diverticular disease. *Ann. Surg.*, 1973, *177*, 519–526.

7. Drummond, H. Sacculi of large intestine with special reference to their relations to blood vessels of bowel wall. *Brit. J. Surg.*, 1916, *4*, 407–413.

8. Eisenberg, H., Laufer, I., and Skillman, J J. Arteriographic diagnosis and management of suspected colonic diverticular hemorrhage. *Gastroenterology*, 1973, *64*, 1091–1100.

9. Finby, N., and Begg, C. F. Massive gastrointestinal hemorrhage. *New York J. Med.*, 1971, *71*, 233–236.

10. Gieske, J. C., Kahn, P. C., and Moran, J. M. Arterial ligation for control of diverticular hemorrhage. *J.A.M.A.*, 1971, *217*, 1100–1101.

11. Healey, S. J., and Pfeffer, R. I. Exsanguinating hemorrhage from diverticulosis of ascending colon: report of case. *New England J. Med.*, 1965, *273*, 1480–1481.

12. Jones, F. A. Problems of alimentary bleeding. *Rendiconti*, 1970, *2*, 118–132.

13. Kanter, I. E., Schwartz, A. J., and Fleming, R. J. Localization of bleeding point in chronic and acute gastrointestinal hemorrhage by means of selective visceral arteriography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 386–399.

14. Kirkpatrick, J. R. Massive rectal bleeding in adult. *Dis. Colon & Rectum*, 1969, *12*, 248–252.

15. Klein, R. R., and Gallagher, D. M. Massive colonic bleeding from diverticular disease. *Am. J. Surg.*, 1969, *118*, 553–557.

16. Koehler, P. R., and Salmon, R. B. Angiographic localization of unknown acute gastrointestinal bleeding sites. *Radiology*, 1967, *89*, 244–249.

17. Lee, R. E., and Finby, N. Jejunal and ileal diverticulosis. *A.M.A. Arch. Int. Med.*, 1958, *102*, 97–102.

18. McGUIRE, H. H., and HAYNES, B. W. Massive hemorrhage from diverticulosis of colon: guidelines for therapy based on bleeding pattern observed in 50 cases. *Ann. Surg.*, 1972, *177*, 847–853.
19. MEYERS, M., VOLBERG, F., KATZEN, B., ALONSO, D., and ABBOTT, G. Angio architecture of colonic diverticula: significance in bleeding diverticulosis. *Radiology*, 1973, *108*, 249–261.
20. NOER, R. J., HAMILTON, J. E., and WILLIAMS, D. J. Rectal hemorrhage: moderate and severe. *Ann. Surg.*, 1962, *155*, 794–805.
21. NUSBAUM, M., and BAUM, S. Radiographic demonstration of unknown sites of gastrointestinal bleeding. *S. Forum*, 1963, *14*, 374–375.
22. OLSEN, W. R. Hemorrhage from diverticular disease of colon: role of emergency subtotal colectomy. *Am. J. Surg.*, 1968, *115*, 247–263.
23. RÖSCH, J., GRAY, R. K., and GROLLMAN, J. H. Selective arterial drug infusions in treatment of acute gastrointestinal bleeding: preliminary report. *Gastroenterology*, 1970, *59*, 341–349.
24. ROSENBERG, I. K., and ROSENBERG, B. F. Massive hemorrhage from diverticula of colon with demonstration of source of bleeding: case report. *Ann. Surg.*, 1964, *159*, 570–573.
25. SALVATI, E., BONG, H. H., and VARGA, C. F. Massive hemorrhage from colonic diverticula caused by arterial erosion; practical theory of its mechanism and causation: report of two cases. *Dis. Colon & Rectum*, 1967, *10*, 129–135.
26. SLACK, W. W. Diverticula of colon and their relation to muscle layers and blood vessels. *Gastroenterology*, 1960, *39*, 708–712.
27. SORGER, K., and WACKS, M. R. Exsanguinating arterial bleeding associated with diverticulating disease of colon. *A.M.A. Arch. Surg.*, 1971, *102*, 9–13.
28. STECKEL, R. J., RÖSCH, J., and ROSS, G. New developments in pharmacoangiography: (and arterial pharmacotherapy) of gastrointestinal tract. *Invest. Radiol.*, 1971, *6*, 199–211.
29. TAYLOR, F. W. Treatment of massive diverticular hemorrhage. *A.M.A. Arch. Surg.*, 1969, *98*, 505–508.
30. WEINGARTEN, M., VENET, L., and VICTOR, M. B. Direct observation of massive arterial hemorrhage from diverticulum of hepatic flexure. *Gastroenterology*, 1959, *36*, 642–644.
31. WILLIAMS, R. C. Aortic stenosis and unexplained gastrointestinal bleeding. *A.M.A. Arch. Int. Med.*, 1961, *108*, 859–863.
32. WOLLAEGER, E. E., STAUFFER, M. H., and ADSON, M. A. Massive hemorrhage from colonic diverticulitis. *Proc. Staff Meet. Mayo Clin.*, 1966, *41*, 549–559.

VOL. 121, NO. 2

VENTRAL PORTAL VEIN*

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THE purpose of this paper is to succinctly review the topic of the ventral portal vein.

In most patients, the portal vein (PV) lies dorsal or crosses behind the ascending or vertical portion of the proper hepatic artery.[7] At the porta hepatis, the right, left and middle hepatic arteries commonly have a similar relationship to the PV or to the PV's right and left branches.[7]

Ten basic types exist in the hepatic arterial blood supply.[7] In 7 of these types, which include over 90 per cent of patients, the portal vein or its right branch lies dorsal or crosses behind a right hepatic artery. In 3 variations, however, the portal vein runs ventral or in front of an aberrant right hepatic artery and in such instances the portal vein is termed a "ventral PV." In 1 of these 3 variations, an aberrant right hepatic artery (ARH) arises from the celiac axis independently of the common hepatic (Type X variant).[7] In this variation, the ARH usually courses dorsal or crosses behind the more ventrally positioned PV (Fig. 1).[7]

In other variations, (Type VII, VIIIA), an ARH arises from the superior mesenteric artery, proceeds upwards and crosses behind a more ventrally located PV.[7] These 3 variations (Type X variant, VII, VIIIA) may be of interest to both the angiographer and surgeon. The surgeon may occasionally have difficulty in constructing a portacaval shunt with a ventral PV.[4]

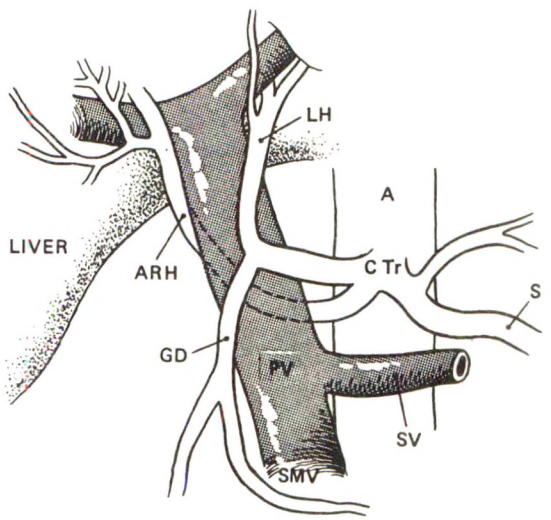


FIG. 1. Drawing showing the relationship of the ventral portal vein to the aberrant right hepatic artery in a Type X variant. ARH = aberrant right hepatic artery; LH = left hepatic artery; GD = gastroduodenal; S = splenic; CTr = celiac trunk; A = aorta; PV = portal vein. (Modified from Michels.[7])

REPORT OF CASES

CASE 1. This 59 year old housewife vomited bright red blood in January 1973.

In 1955 she had had a brief period of malaise, fatigue and jaundice. Liver biopsy in 1964 at Stanford University Hospital showed chronic active hepatitis with minimal fibrosis. Splenomegaly was documented in 1968. In 1970, esophageal varices were found on an upper gastrointestinal series.

Physical examination in January 1973 revealed a spleen tip palpable 8 cm. below the left costal margin. Multiple skin ecchymoses were present. Laboratory values included: hematocrit 41 per cent; platelets 91,000 per cu. mm.; total bilirubin 3.7 mg. per cent; alkaline phosphatase 125 (normal 30–85) units; prothrombin time 60 per cent.

Because of continued bleeding, abdominal angiography was performed using a polyurethane cobra-shaped catheter. Celiac axis angiography was performed with 60 ml. of 66 per cent meglumine diatrizoate and 10 per cent sodium diatrizoate machine injected at 10 ml. per second. An ARH arose from either a short common hepatic artery or the celiac trunk (Fig. 2*A*). The venous phase showed large varices (Fig. 2*B*). The PV in this anatomic

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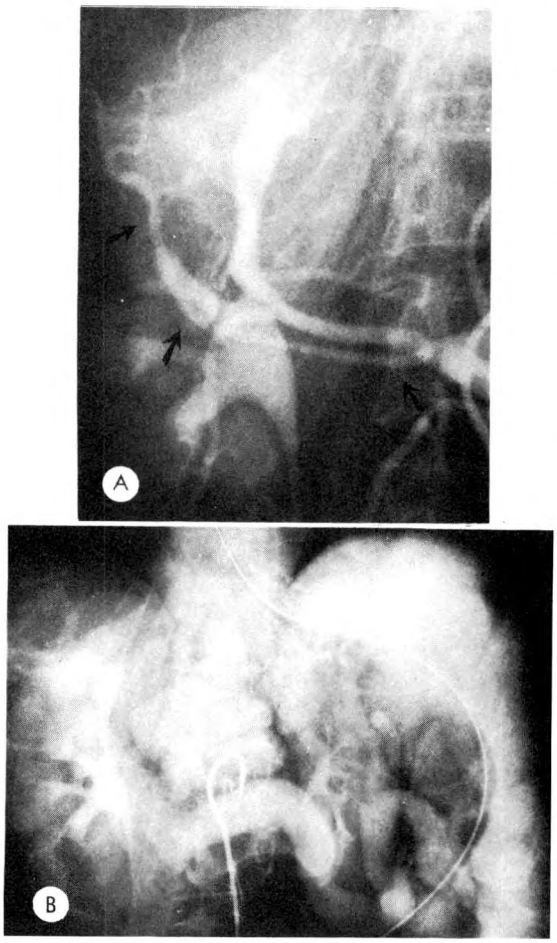


FIG. 2. Case 1. (*A*) An aberrant right hepatic artery arises from either a short common hepatic artery or the celiac trunk (arrows). (*B*) Venous phase showing large varices. The portal vein in this variant probably crosses ventral to the aberrant right hepatic artery. The surgeon performed a spleno-renal rather than portacaval shunt. Bringing a ventral portal vein posteriorly to establish a portacaval shunt may prove technically difficult.

variant probably crossed ventral to the ARH. No arterial source of bleeding was angiographically demonstrated. Superior mesenteric artery infusion with vasopressin was begun which controlled the bleeding.

Three days following angiography, the patient was taken for elective surgery and underwent splenectomy and a spleno-renal shunt from which she recovered uneventfully.

Comment. A spleno-renal shunt was performed on this patient rather than a portacaval shunt. Had a portacaval shunt been tried, the surgeon may have had difficulty attempting to bring the ventral PV posteriorly to establish the portacaval shunt.[4] According to Dr. S. Baum, operative difficulty has arisen in cases of Type X variants because of the increased distance between the ventral PV and the vena cava.[4]

Awareness of the existence of the ventral PV may occasionally be of interest to the radiologist as he attempts to relate anatomy to angiographic findings. When major arteries and major veins lie in apposition, visceral carcinomas tend to preferentially invade or compress the veins.[1,3,5] Angiographers have reported a 90 per cent or higher incidence of venous involvement in pancreatic carcinomas.[2,5] What factors occur to distinguish those patients with only arterial involvement from the majority with major venous involvement is not yet clear. In the Type X variant, major arterial involvement without concomitant venous involvement may occur.

CASE II. A 62 year old Caucasian female was referred to Stanford University Hospital for palliative radiation therapy of pancreatic carcinoma.

Her past medical history included diabetes mellitus documented in 1959 which was controlled by 39 units of NPH insulin. In July 1971 she developed pruritus and dark urine. Her serum bilirubin measured 12.2 mg. per cent—direct 9.6 mg. per cent. She underwent diagnostic laparotomy and a rock-hard mass in the superior portion of the pancreatic head was located which involved the lower end of the common duct. A choledochoduodenostomy was performed. Biopsy samples, taken in 2 places, were insufficient for definitive diagnosis. Postoperatively, her jaundice subsided.

She was first admitted to Stanford University Hospital in November 1971 for treatment of severe epigastric pain. Her total serum bilirubin was 1.6 mg. per cent—direct 0.9 mg. per cent, alkaline phosphatase 906 mU./ml. (normal <85 mU./ml.), SGOT 77 (normal <50 mU./ml.). Upper gastrointestinal examination showed a 3 cm. long irregular, nonpliable narrowing just distal to the choledochoduodenostomy. Barium refluxed into a normal appearing biliary tree.

Gastrointestinal angiography was performed through a polyurethane cobra-shaped catheter.

Celiac axis angiography was performed first. Neither the catheter nor guide wire was advanced beyond the celiac axis. Sixty milliliters of 66 per cent meglumine diatrizoate and 10 per cent sodium diatrizoate were machine injected at 10 ml. per second. Following celiac axis angiography, splenic angiography was performed with 60 ml. of 66 per cent meglumine diatrizoate and 10 per cent sodium diatrizoate injected at 6 ml. per second. A superior mesenteric angiogram in a slightly right posterior oblique position was then obtained utilizing 70 ml. of contrast medium at 6 ml. per second.

Celiac axis angiography showed an ARH originating from the celiac axis (Fig. 3*A*). Michels has termed this arterial arrangement the Type X variant.[7] In this anomaly, the PV crosses ventral to the aberrant hepatic artery

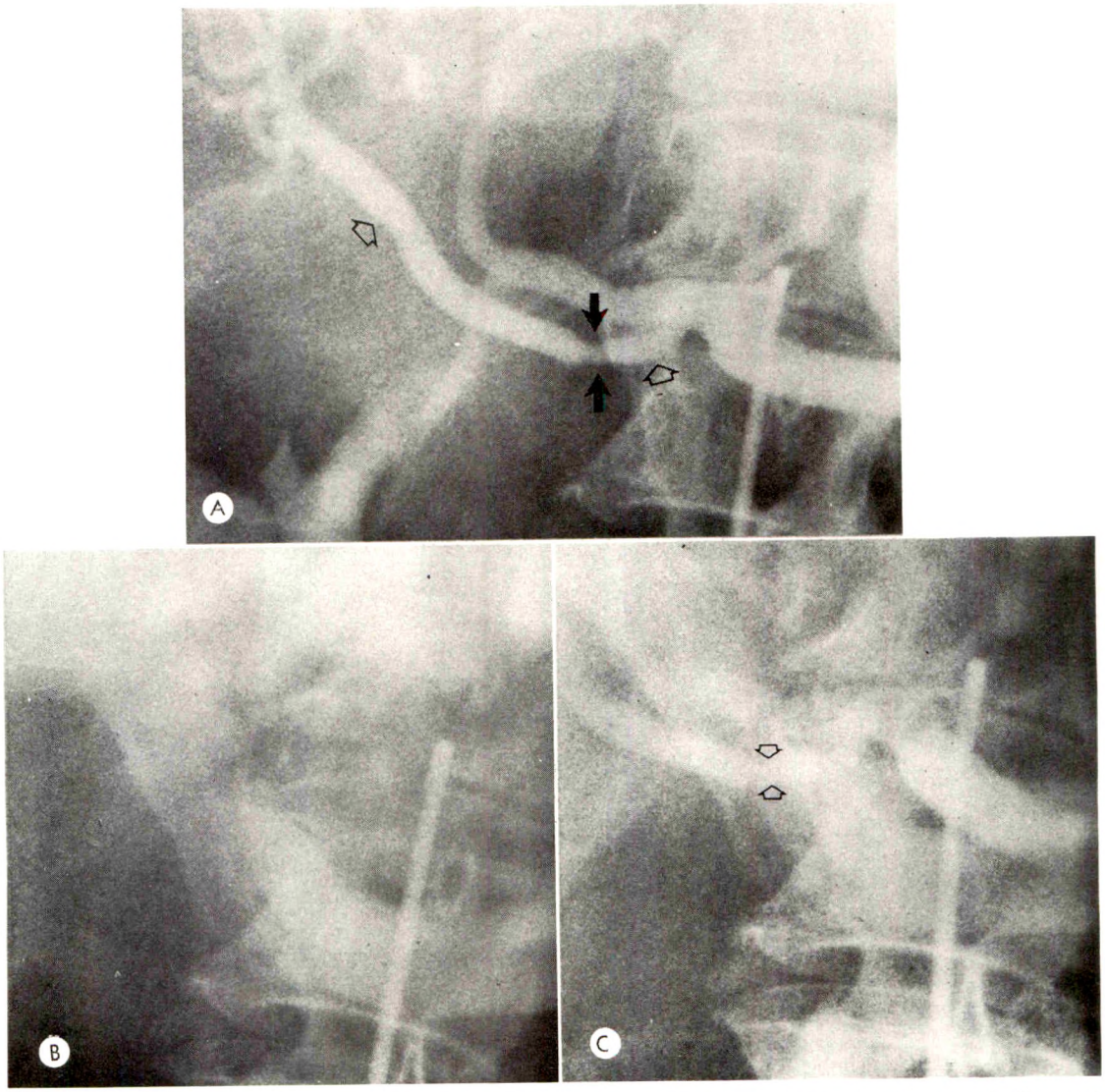


Fig. 3. Case 11. (*A*) The right hepatic artery (open arrows) arises separately from the celiac trunk. Serrated encasement of the aberrant right hepatic artery is present (black arrows). (*B*) The portal vein and splenic vein are normal. Washout from unopacified superior mesenteric venous blood creates the "filling" defect at the right lower origin of the portal vein. (*C*) Relationship of the aberrant right hepatic artery to the ventral portal vein, slightly right posterior oblique projection. The arrows indicate the encased segment of the aberrant right hepatic artery as it crosses behind the more ventrally positioned portal vein.

(Fig. 1).[7] The gastroduodenal and the middle and left hepatic arteries arose from a common hepatic artery from the celiac trunk (Fig. 3*A*). An irregular, asymmetric, narrowed area occurred several cm. from the origin of the replaced right hepatic artery. The portal vein crossed over the serrated area and was spared (Fig. 3, *B* and *C*). Splenic and superior mesenteric angiograms showed that the splenic and superior mesenteric arteries and veins were normal.

At surgery a rock-hard tumor was again located in the superior aspect of the head of the pancreas obstructing the second part of the duodenum. An anterocolic gastrojejunostomy was performed. The patient began radiation therapy for palliative relief of pain in mid December 1971 receiving 2,250 rads to the area of the pancreas over 22 days. Her condition continued to deteriorate. She died in January 1972.

Comment. At surgery, the carcinoma involved the superior aspect of the pancreatic head. Major arterial involvement was angiographically demonstrated in the aberrant right hepatic artery (Type X variant) at the point where it crossed behind the PV. The ventral position of the PV in relation to the ARH suggests an anatomic basis for the lack of PV involvement in this patient.

Pancreatic carcinomas may also involve the ventral PV.

CASE III. A 55 year old white married farmer developed fatigue and fatty food intolerance in April 1972. He described a mild epigastric pain which radiated into his back and was relieved by leaning forward. An oral cholecystogram demonstrated nonvisualization. Upper gastrointestinal series was normal. In July 1972 the patient underwent laparotomy and common bile duct exploration. A common bile duct stone was removed and cholecystectomy performed. He recovered uneventfully but still suffered epigastric pain. His first Stanford University Hospital visit was in November 1972. Routine chemical screening was normal except for an alkaline phosphatase of 250 mU./ml. (normal <85 mU./ml.). Liver scan was normal.

Gastrointestinal angiography was performed with a polyurethane cobra-shaped catheter. Celiac axis angiography was performed first. No guide wires were introduced into the celiac axis nor was the catheter advanced beyond the celiac axis. Sixty milliliters of 66 per cent meglumine diatrizoate and 10 per cent sodium diatrizoate were machine injected at 10 ml. per second. Splenic artery angiography was performed with 60 ml. of 66 per cent meglumine diatrizoate and 10 per cent sodium diatrizoate machine injected at 6 ml. per second. Superior mesenteric angiography was done in a slightly right posterior oblique projection utilizing 70 ml. contrast medium at 5 ml. per second. Celiac angiography showed an ARH arising from the region of celiac axis (Fig. 4*A*). The left hepatic and gastroduodenal arteries arose from a separate trunk from the celiac trunk. An irregular asymmetric narrowed area occurred approximately 2 cm. proximal to the origin of the gastroduodenal artery and at the origin of the ARH (Fig. 4*A*). The adjacent splenic vein and the proximal ventral PV were compressed (Fig. 4*B*). Superior mesenteric angiography showed a normal superior mesenteric artery and vein.

At laparotomy in November 1972, tumor involved the superior head of the pancreas and porta hepatis. The neoplasm extended along the common duct and involved the base of the small bowel mesentery. Pathologic examination revealed a glandular pattern with lakes of mucinous material consistent with pancreatic adenocarcinoma.

Comment. This patient had a Type X variant of the hepatic arterial supply. The common hepatic artery and the origin of the ventral PV were both involved by tumor. The neoplasm also involved the origin of the ARH but spared the ARH where it crossed behind the ventral PV. The angiographic findings in this case were typical of pancreatic carcinoma.[4]

DISCUSSION

Michels has classified the hepatic arterial blood supply into 10 basic types.[7] In a majority of patients, the vertical or ascending portion of the hepatic artery lies anterior or crosses in front of the portal vein (PV) to reach the porta hepatis.[7] In 7 of the 10 types, the PV lies behind the vertical portion of the hepatic artery or the right hepatic artery.[7] In 3 variations, however, the PV runs ventral to an aberrant right hepatic artery (ARH), and in such instances the PV is termed a "ventral PV." Ventral PVs are usually associated with ARH; however, most ARH are not associated with ventral PVs. Most ARH arise from the superior mesenteric artery and

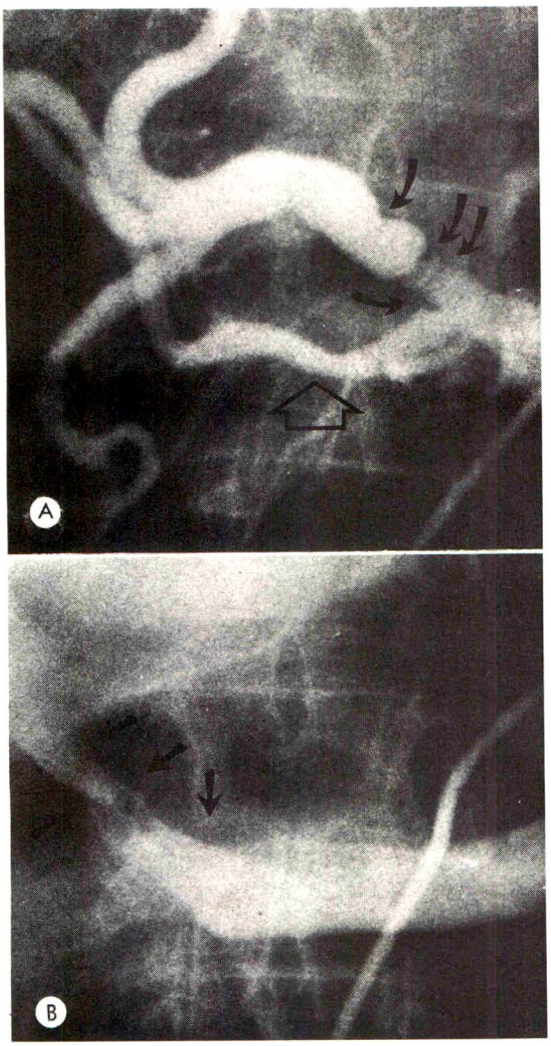


Fig. 4. Case III. (*A*) Celiac angiogram showing an aberrant right hepatic artery (open arrow) arising from the celiac axis or the very proximal common hepatic artery. Encasement of the common hepatic artery by pancreatic carcinoma has occurred (double black arrows). The origin of the aberrant right hepatic artery is also involved (single black arrow). (*B*) The venous phase of splenic angiography shows compression of the distal splenic vein and ventral portal vein (arrows).

run in front of the PV. The incidence of ventral PV may be estimated from Michels' work.[7] He reported that in 2 per cent of his dissections, a ventral PV was associated with an ARH arising from the superior mesenteric artery (Type VII, VIIIA). Michels reported an ARH arising from the celiac axis in 2 of 200 dissections (Type X variant).[7] Nebesar *et al.*[8] found 3 examples in their 236 cases. The combined incidence of the Type X variant from the series of Michels and Nebesar *et al.* is 1.2 per cent. Computation of a 95 per cent confidence limit using normal approximations yields an incidence for Type X variants of 0.2–2.2 per cent.[5]

CONCLUSION

Awareness of the existence of a ventral PV (portal vein) may help in planning shunt surgery for portal hypertension.

The surgeon may have difficulty if he attempts to bring a ventral PV posterior to establish a portacaval shunt and may wish instead to elect a spleno-renal or mesocaval approach.[4]

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REFERENCES

1. Ackerman, L. V., and del Regato, J. A. Cancer, Diagnosis, Treatment and Prognosis. C. V. Mosby Company, St. Louis, 1970.
2. Bookstein, J. Year Book of Radiology. Year Book Medical Publishers, Chicago, 1973.
3. Boyd, W. A Textbook of Pathology—Structure and Function in Disease. Lea and Febiger, Philadelphia, 1970.
4. Baum, S. Personal communication.
5. Buranasiri, S., and Baum, S. Significance of venous phase of celiac and superior mesenteric arteriography in evaluating pancreatic carcinoma. *Radiology*, 1972, *102*, 11–20.
6. Goldstein, A. Biostatistics—An Introductory Text. The MacMillan Company, New York, 1964.
7. Michels, N. A. Blood Supply and Anatomy of the Upper Abdominal Organs with a Descriptive Atlas. J. B. Lippincott Company, Philadelphia, 1955.
8. Nebesar, R. A., Kornblith, P. L., Pollard, J. J., and Michels, N. A. Celiac and Superior Mesenteric Arteries—A Correlation of Angiograms and Dissections. Little, Brown and Company, Boston, 1969.

CONTRAST ENHANCEMENT OF THE MESENTERIC AND PORTAL VEINS USING INTRA-ARTERIAL PAPAVERINE*

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VISUALIZATION of the superior mesenteric and portal veins is helpful in the diagnosis of gastric and esophageal varices, mesenteric vein-systemic shunts, and intra-abdominal neoplasm.

Celiac and superior mesenteric arteriograms in patients with cirrhosis and portal hypertension often result in poor opacification of the venous phase. A wide variety of pharmacologic techniques to enhance venous opacification have been previously reported which include the use of epinephrine,[9] tolazoline,[10] bradykinin,[1,5] histamine,[8] contrast medium,[12] glucagon,[7] and a combination of phentolamine and isoproterenol.[6,13,14]

Comparison of nonpharmacologically assisted superior mesenteric angiograms, the 10 minute postepinephrine mesenteric angiograms and superior mesenteric angiograms after papaverine infusion as devised by Bron[4] is presented to demonstrate that optimal visualization of the corresponding veins has been seen with the papaverine technique.

MATERIAL AND METHOD

In the past 3 years, a total of 139 superior mesenteric angiographies were performed on 111 patients at the Boston Veterans Administration Hospital. Fifty-five per cent had moderately or markedly enlarged spleens. Forty-two per cent had a biopsy proven diagnosis of liver cirrhosis. There were 75 nonpharmacologic angiographies, 8 postepinephrine, and 56 papaverine studies performed. Sixteen patients had both papaverine and nonpapaverine arteriographies. One patient had all 3 techniques.

Selective catheterization of the superior mesenteric artery was performed under fluoroscopic control using Becton-Dickinson radiopaque polyethylene tubing, heavy wall, 50 cm. long, I.D. .054 inches tapered over an .045 inch guidewire. An 80 cc. bolus of meglumine iothalamate* was injected by electric power injector in each of the superior mesenteric arteriographies.

NONPHARMACOLOGICALLY ASSISTED SUPERIOR MESENTERIC ANGIOGRAM

Rate of Injection. Selected by test injections under fluoroscopic control, usually 8–10 cc. per second.

Exposure Program. In the noncirrhotic patients, 12 films were exposed over a 20 second interval. In the cirrhotic patients, 14 films were exposed over a 26 second period.

10 MINUTE POSTEPINEPHRINE SUPERIOR MESENTERIC ANGIOGRAM

Epinephrine is diluted in saline to a concentration of 1 microgram per cc. Twenty micrograms are injected into the superior mesenteric artery over a period of 2 minutes. The angiogram is obtained 10 minutes after the epinephrine infusion.

Rate of Injection. Selected by test injections under fluoroscopic control 8 minutes after the end of the epinephrine infusion.

Exposure Program. The same as for the nonpharmacologic angiograms.

PAPAVERINE SUPERIOR MESENTERIC ANGIOGRAM

Forty-five milligrams papaverine hydrochloride is diluted into 25 cc. of saline. The

* Conray 60 (Mallinckrodt).

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Table I

ANALYSIS OF VISUALIZATION OF THE MESENTERIC AND PORTAL VEINS

	No. of Angiograms	0*	1	2	3
Superior mesenteric arteriogram	75	6 (8%)	21 (28%)	36 (48%)	12 (16%)
10 minute postepinephrine arteriogram	8	4 (50%)	2 (25%)	0	2 (25%)
Papaverine superior mesenteric arteriogram	56	0	4 (7%)	10 (18%)	42 (75%)
Total	139				

* 0 = No visualization of superior mesenteric vein (SMV).
1 = Faint visualization of SMV and no visualization of portal vein.
2 = Distinct visualization of SMV and portal vein but not the portal vein branches.
3 = Distinct visualization of SMV, the portal vein, and its branches.

entire amount is infused over a period of 30 seconds. The angiography is performed immediately. This is facilitated by using a 3 way stopcock.

Rate of Injection. Add 3 cc. per second to the rate selected under fluoroscopic control that would otherwise be used for a nonpharmacologic study.

Exposure Program. The same as for nonpharmacologic angiograms.

The best venous film in each angiographic run was evaluated for quality of opacification. The criteria used are listed in Table 1.

RESULTS

Because the 10 minute postepinephrine technique has been shown to be better than the nonpharmacologic celiac angiogram for demonstrating the splenic vein,[9] this technique was originally applied to the superior mesenteric artery. No improvement in venous opacification was noted when compared with the nonpharmacologic study. The effect of epinephrine on the mesenteric system was noted to be quite variable.

The infusion of papaverine in the superior mesenteric artery was consistently more reliable for the demonstration of the superior mesenteric and portal veins than either the 10 minute postepinephrine or the nonpharmacologic angiogram. There was adequate or excellent visualization of these veins (Fig. 1–6) in 93 per cent of angiograms, when using the papaverine technique, as opposed to 25 per cent and 64 per cent, respectively, with the alternative methods (Table 1).

Whereas 8–10 ml. per second injection rate could be used in nonpharmacologic superior mesenteric angiography, the use of papaverine allowed the injection of a large bolus (80 cc.) of contrast medium at a rate of 13–18 ml. per second. At no time could reflux of contrast medium into the aorta be demonstrated after using papaverine.

Sixteen patients had both papaverine and nonpapaverine superior mesenteric angiographies. Papaverine improved the venous phase in 87 per cent of cases (Table 11) (Fig. 1, *A* and *B*; 2, *A* and *B*; and 4, *A–C*).

Systemic alteration of vital signs was not

Table II

COMPARISON OF PAPAVERINE AND NONPAPAVERINE SUPERIOR MESENTERIC ARTERIOGRAPHY IN THE SAME PATIENTS

	No. of Patients
No Change	2 (13%)
Minimal Improvement (improvement by 1 category, see Table 1, of the venous visualization when using papaverine)	7 (44%)
Moderate Improvement (improvement by 2 categories)	6 (37%)
Marked Improvement (improvement by 3 categories)	1 (6%)
Total	16

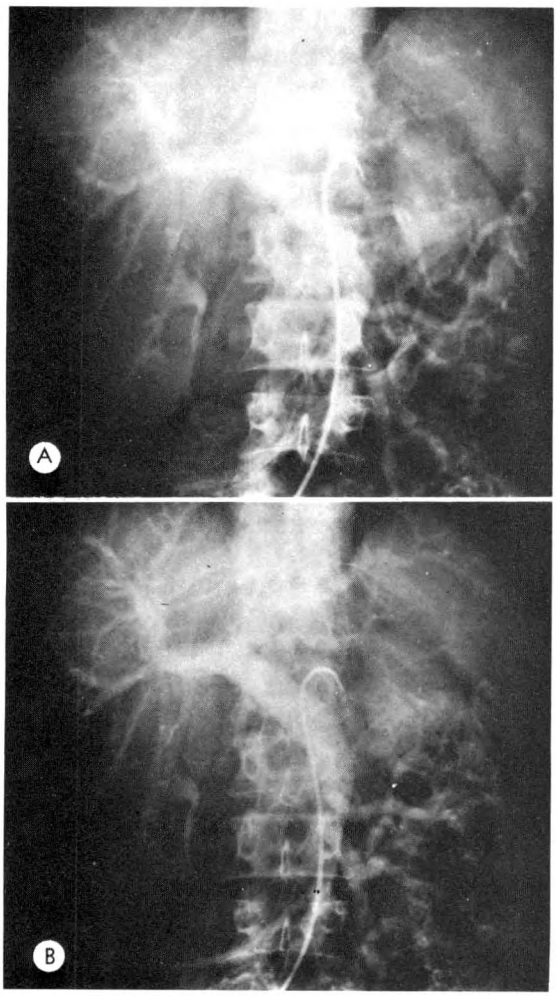


FIG. 1. (*A*) Superior mesenteric arteriogram without papaverine, and (*B*) with papaverine. *B* clearly shows better opacification and detail of the portal vein, its branches and tributaries.

clinically apparent in the patients studied with papaverine. Intra-aortic blood pressures were monitored *via* a separate catheter from the opposite femoral artery in 5 patients having these angiographies. Only transient decreases in systolic (20 mm. Hg) and diastolic (5 mm. Hg) pressures were noted, and these reverted to baseline within 60 seconds after the angiography. There was no undue discomfort experienced by any patient either during the papaverine infusion or during the angiography that followed.

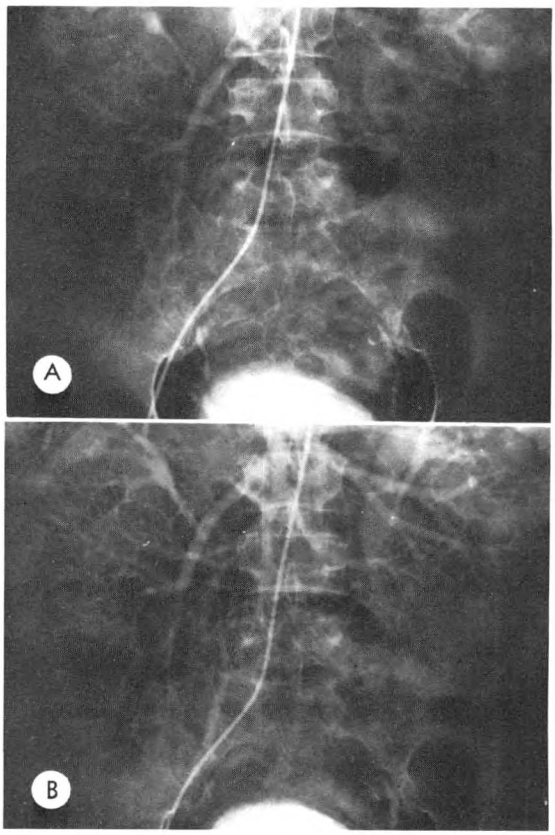


FIG. 2. (*A*) Superior mesenteric arteriogram without papaverine and (*B*) with papaverine. Note the increased detail of the mesenteric vein branches after papaverine.



FIG. 3. A 40 year old cirrhotic male had a gastric fundal mass on upper gastrointestinal series. Papaverine superior mesenteric arteriogram demonstrated huge gastroesophageal varices (arrows).

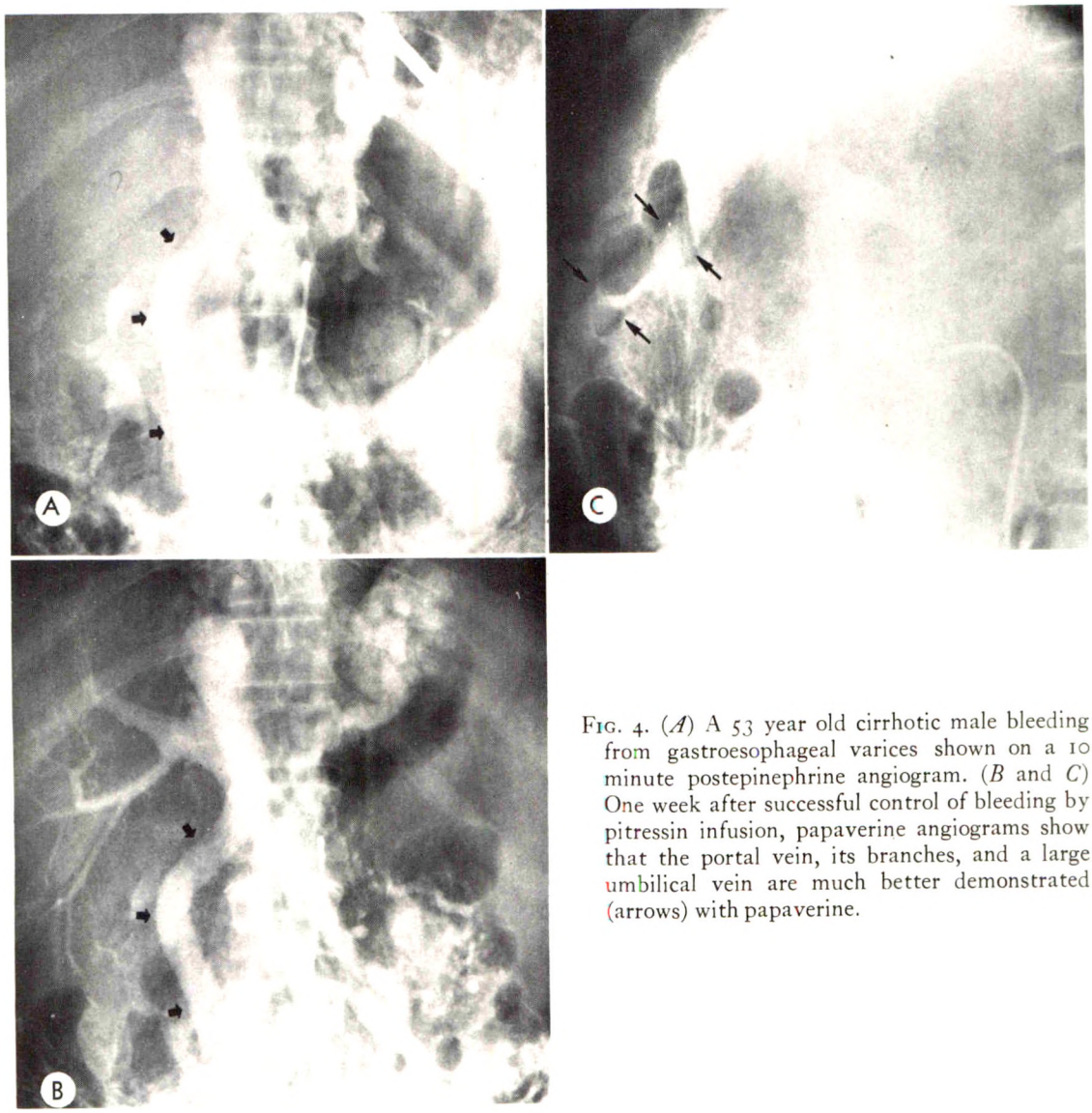


Fig. 4. (*A*) A 53 year old cirrhotic male bleeding from gastroesophageal varices shown on a 10 minute postepinephrine angiogram. (*B* and *C*) One week after successful control of bleeding by pitressin infusion, papaverine angiograms show that the portal vein, its branches, and a large umbilical vein are much better demonstrated (arrows) with papaverine.

DISCUSSION

Papaverine is a smooth muscle relaxant. It has been shown that the degradation of cyclic AMP results in vasodilatation in the mesenteric arterial bed, in a manner that is unclear. Papaverine has been reported to inhibit the action of nucleotide phosphodiesterase which normally degrades cyclic AMP.[3,11]

The injection of papaverine into the superior mesenteric artery results in the dilatation of the peripheral mesenteric arterioles. There is a resultant increase in arteriovenous flow. A bolus of contrast material can, therefore, be injected at a faster rate than if no pharmacologic agent had been used. The ability to more rapidly inject the contrast material bolus minimizes dilution. Contrast enhancement on the venous side is thus obtained.

The arterial phase of papaverine angiography often appears somewhat less sharply outlined than in the nonpharmacologic technique. This phenomenon has also been associated with the use of tolazoline[10] and bradykinin.[1] When arterial detail is essen-

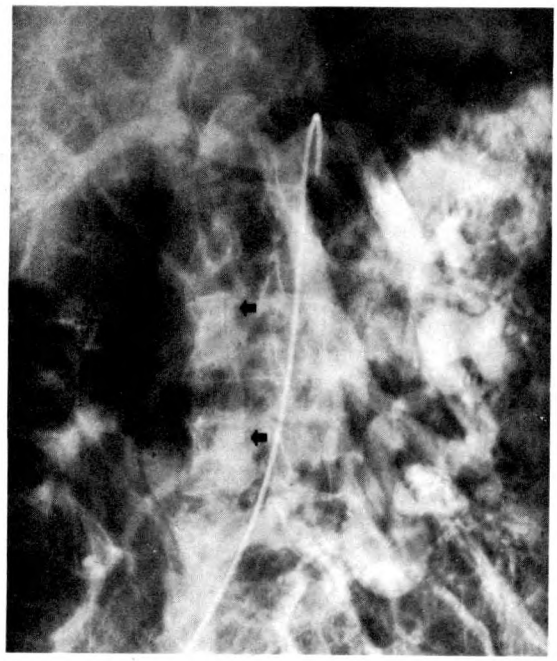


FIG. 5. A 58 year old cirrhotic male. Papaverine mesenteric angiogram demonstrates a large vein extending inferiorly from the splenic vein. Note the spontaneous splenomesenteric-caval shunt with good visualization of the inferior vena cava (arrows).

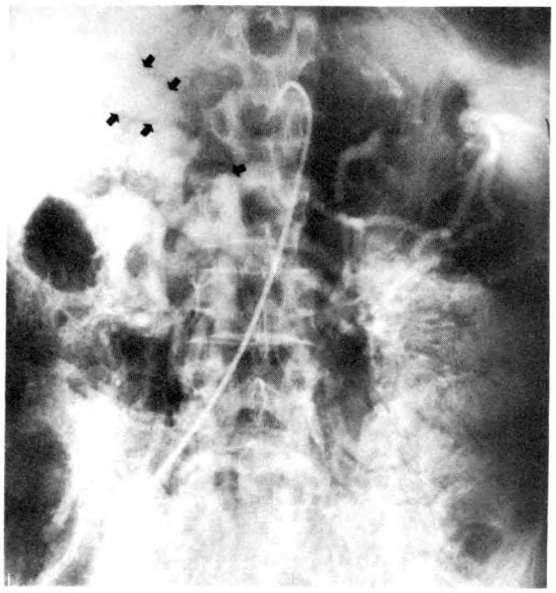


FIG. 6. A 72 year old male with carcinoma of the head of the pancreas. Papaverine angiogram shows an occlusion of the superior mesenteric vein (single arrow) and reconstitution of the portal vein which is tapered at its proximal end due to the carcinoma (paired arrows).

tial, one can omit papaverine and repeat the angiography using a smaller bolus of contrast material as well as a smaller number of films.

Epinephrine has been shown to have a lesser effect in the mesenteric vessels than in the celiac artery.[2] This accounts for the relatively inconsistent visualization of the mesenteric veins during the 10 minute post-epinephrine technique.

Chou *et al.*[5] reported in their studies, that the kinins are "the most potent vasodilators known in the superior mesenteric circulation." Unfortunately, the use of bradykinin in the superior mesenteric artery is not available for clinical application in this country.

Tachycardia, hypotension, chills, and increased painful response to contrast medium have been noted occasionally after the use of tolazoline.[8,10] Transient nausea and vomiting in dogs were noted with higher doses of glucagon.[7] Isoproterenol may be contraindicated in patients with cardiovascular disorders.[13] These limiting side effects have not been found with papaverine.

CONCLUSION

Excellent mesenteric and portal venous opacification can be difficult to accomplish, especially when there is hemodynamic or mechanical impairment.

Papaverine infused in the superior mesenteric artery was found to be safe and without significant systemic effect. A comparison study of nonpharmacologic mesenteric angiograms, the 10 minute post-epinephrine technique, and the use of papaverine clearly shows the superior quality of the latter technique in accomplishing this angiographic result.

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REFERENCES

1. Boijsen, E., and Redman, H. C. Effect of bradykinin on celiac and superior mesenteric angiography. *Invest. Radiol.*, 1966, *1*, 422–430.
2. Boijsen, E., and Redman, H. C. Effect of epinephrine on celiac and superior mesenteric angiography. *Invest. Radiol.*, 1967, *2*, 184–199.
3. Boley, S. J., Sprayregen, S., Veith, F. J., and Siegelman, S. S. Aggressive roentgenologic and surgical approach to acute mesenteric ischemia. *Surg. Ann.*, 1973, *5*, 19–41.
4. Bron, K. Personal communication.
5. Chou, C. C., Frohlich, E. D., and Texter, E. C., Jr. Comparative study of effects of bradykinin, kallidin II and eledoisin on segmental superior mesenteric resistance. *J. Physiol.*, 1965, *176*, 1–11.
6. Cioffi, C. M., Ruzicka, F. F., Jr., Carillo, F. J., and Gould, H. R. Enhanced visualization of portal system using phentolamine and isoproterenol in combination. *Radiology*, 1973, *108*, 43–49.
7. Danford, R. O., and Davidson, A. J. Use of glucagon as vasodilator in visceral angiography. *Radiology*, 1969, *93*, 173–175.
8. Kahn, P. C., and Callow, A. D. Selective vasodilatation as aid to angiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *94*, 213–220.
9. Kahn, P. C., O'Halloran, J. F., Jr., and Paul, R. E., Jr. Improved portography by delayed postepinephrine celiac and mesenteric arteriography. *Radiology*, 1969, *92*, 86–89.
10. Redman, H. C., Reuter, S. R., and Miller, W. J. Improvement of superior mesenteric and portal vein visualization with tolazoline. *Invest. Radiol.*, 1969, *4*, 24–27.
11. Shepherd, A. P., Mao, C. C., Jacobson, E. E., and Shanbour, L. L. Role of cyclic AMP in mesenteric vasodilation. To be published.
12. Siegelman, S. S., Warren, A., Veith, F. J., and Boley, S. J. Physiologic response to superior mesenteric angiography. *Radiology*, 1970, *96*, 101–105.
13. Steckel, R. J., Rösch, J., Ross, G., and Grollman, J. H., Jr. New developments in pharmacoangiography (and arterial pharmacotherapy) of gastrointestinal tract. *Invest. Radiol.*, 1971, *6*, 199–211.
14. Van Heertum, D. L., Cioffi, C. M., and Ruzicka, F. F., Jr. Use of alpha blocking and beta stimulating drugs in combination to improve opacification of portal venous system. *Radiology*, 1971, *100*, 679–682.

USE OF C-TERMINAL OCTAPEPTIDE OF CHOLECYSTOKININ IN CHOLECYSTOGRAPHY*

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CHOLECYSTOKININ is a 33 amino acid polypeptide.[5] The C-terminal octapeptide fragment (OP-CCK) reproduces all the known biologic activities of the intact molecule.[5,6]

The purpose of this study was to determine the effect on gallbladder contraction of 20 ng./kg. of OP-CCK intravenously in patients undergoing cholecystography.

METHOD

Thirty adult patients (29 male, 1 female; mean age 48.6 years) undergoing oral cholecystography for investigation of various abdominal symptoms were studied. Each patient gave informed written consent. Prior to entry into the study, each patient underwent a complete medical history and physical examination. The following laboratory tests were obtained before and 24 hours after cholecystography: hemoglobin; white blood cell count and differential; urinalysis; serum glucose; serum electrolytes; SGOT; SGPT; alkaline phosphatase; total bilirubin; serum albumin and globulin; serum calcium and phosphorus. The patients were questioned for symptoms, and the physical examination was repeated 24 hours after injection of OP-CCK.

The patients ingested 3 or 6 gm. sodium ipodate 12 hours before cholecystography. Roentgenograms were obtained with the patient in the upright, prone and prone oblique (15° elevation of the right hip and shoulder) positions. After determination that the gallbladder was well visualized, 20 ng./kg. of OP-CCK was injected intravenously over 30 seconds. Oblique roentgenograms were obtained at 5, 10 and 15 minutes after injection of OP-CCK. The films were developed immediately. Gallbladder size was estimated using the product of maximum length and maximum width perpendicular to maximum length.[7] If the gallbladder size decreased after injection of OP-CCK by less than 40 per cent and the common duct was not visualized, an additional injection of 40 ng./kg. OP-CCK was made and roentgenograms were taken 5, 10 and 15 minutes later. The patients were questioned regarding side effects at 1, 5, 10 and 15 minutes after injection.

OP-CCK* was supplied in sterile vials containing 10 μg. of OP-CCK plus 1 mg. of cysteine hydrochloride as preservative. OP-CCK was dissolved in sterile isotonic saline to a concentration of 1 μg./ml. for injection. A fresh vial was used each day. The vials were stored at −4° C. until used.

RESULTS

Figure 1 shows the mean decrease in gallbladder size 5, 10 and 15 minutes after injection of 20 ng./kg. of OP-CCK. The mean peak decrease in size, *i.e.*, the mean of the greatest decrease for each patient at any of the 3 time intervals, was 48 per cent. The range for peak decrease was 10–100 per cent. Peak decrease occurred at 5 minutes in 5 patients, at 10 minutes in 12 patients, and at 15 minutes in 13 patients.†

Four patients were given an additional injection of OP-CCK, 40 ng./kg. (Table 1). Three of these patients did not respond to the larger dose and 1 had a further 35 per cent decrease in gallbladder size.

The common bile duct was seen in 14 of the 30 patients after OP-CCK, 20 ng./kg.

* Squibb Institute for Medical Research, Princeton, New Jersey, lot number UTA-000 H/TA.

† The mean decreases in size for the 3 time periods were not significantly different ($p > 0.4$).

* From the Research and Medical Services, Gastroenterology Section, Veterans Administration Wadsworth Hospital Center, Los Angeles, California; and the Department of Medicine, UCLA School of Medicine, Los Angeles, California.

(Fig. 2, *A–D*). Forty ng./kg. did not produce additional common duct visualization.

The symptoms occurring after OP-CCK are listed in Table II; they were reported by 25 of the 30 patients. All symptoms were mild, easily tolerated, and disappeared within 3 minutes after injection.

No abnormalities were found by physical examination 24 hours after OP-CCK. There were no significant changes in any of the laboratory tests after OP-CCK.

DISCUSSION

This study provides evidence that OP-CCK, 20 ng./kg., injected intravenously over 30 seconds is safe and effective as a gallbladder contracting agent in man. While side effects were reported by 25 of 30 patients, these were mild, transient and easily tolerated. The patients were urged to report any sensation which they experienced after injection of OP-CCK and were specifically questioned as to the presence or absence of a list of symptoms 1 and 5 minutes after injection. This technique produces a higher incidence of side effects than if only spontaneously reported side effects are recorded. Other reports of the effects of OP-CCK in man have noted similar side effects.[2,4,7] The side effects are probably related to small intestinal contraction and other alterations of gastrointestinal motility.

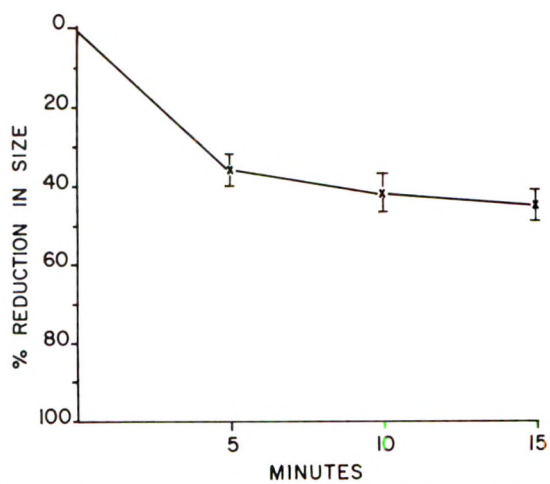


FIG. 1. Mean decrease in gallbladder size at 5, 10 and 15 minutes after intravenous injection of 20 ng./kg. octapeptide of cholecystokinin in 30 patients. Vertical bars indicate ±SEM.

TABLE I

EFFECT OF AN ADDITIONAL DOSE OF 40 NG./KG. OCTAPEPTIDE OF CHOLECYSTOKININ IN 4 SUBJECTS WITH LESS THAN 40 PER CENT DECREASE IN GALLBLADDER SIZE FOLLOWING 20 NG./KG.: PEAK RESPONSE IS PER CENT DECREASE FROM PRE-INJECTION (20 NG./KG.) SIZE

Subject	Peak Response	
	20 ng./kg.	40 ng./kg.
	Per cent	Per cent
1	28	28
2	33	68
3	10	12
4	22	20

Our patients had a greater mean response to 20 ng./kg. OP-CCK than Hedner and Lunderquist's patients after 33 ng./kg.[2] In a previous study from our laboratory, 20 ng./kg. produced a greater response in normal men than 40 ng./kg.[7] On the basis of these data, it is possible that 33 ng./kg. may be a supramaximal dose. Alternatively, differences between subjects or in the experimental conditions may have produced the differences in response between the present study and that of Hedner and Lunderquist.[2]

The diagnostic value of gallbladder contraction during cholecystography is uncertain. Possible advantages include: visualization of gallstones not seen without contraction; visualization of bile ducts and choledocholithiasis; reproduction of the spontaneous pain of biliary dyskinesia,[8] and correlation of the pain with roentgenographic features, and possible prediction of future cholelithiasis.[1]

The common bile duct was visualized in 47 per cent of our patients after OP-CCK. This figure is comparable to that obtainable with a standard fat meal. Since gallbladder contraction occurs at a more rapid rate after OP-CCK than after a fat meal, use of OP-CCK can shorten the time and proba-

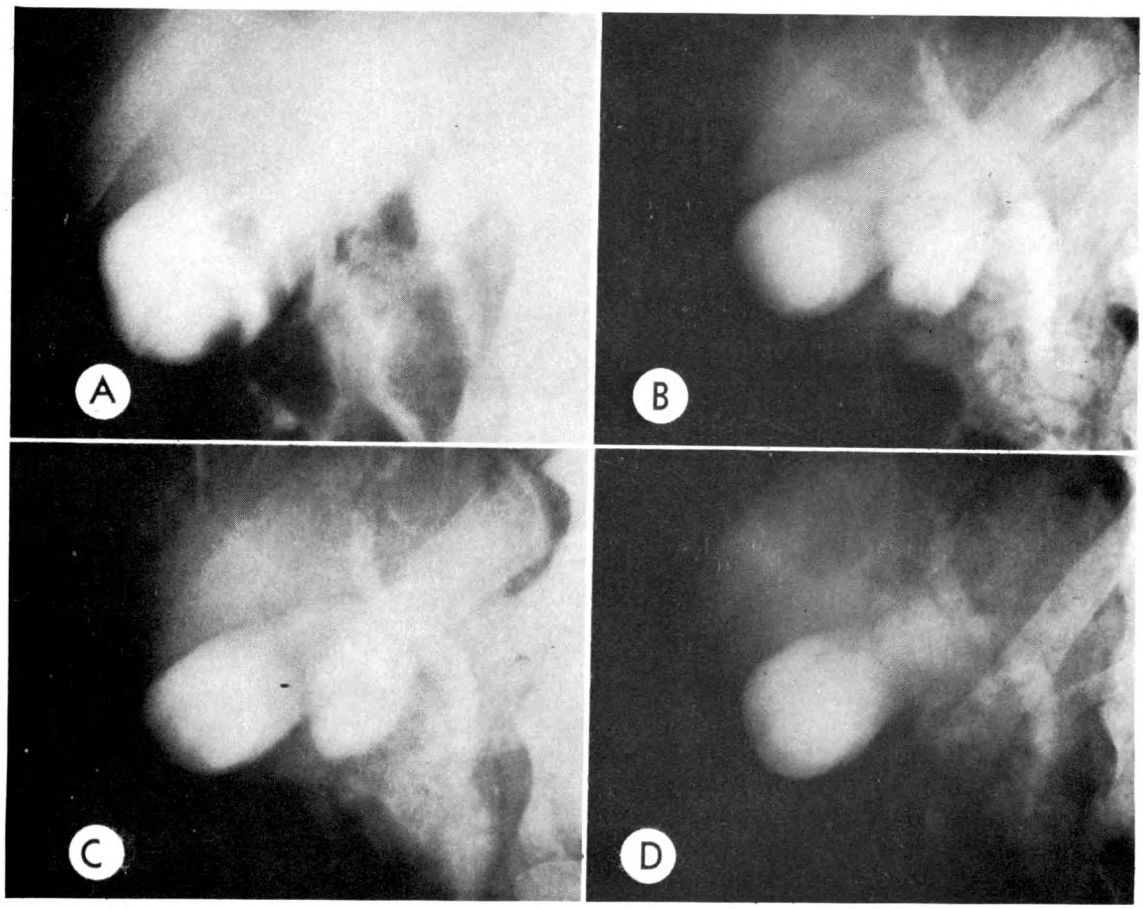


FIG. 2. Gallbladder (*A*) before and (*B*) 5, (*C*) 10, and (*D*) 15 minutes after intravenous injection of 20 ng./kg. octapeptide of cholecystokinin. The common bile duct, common hepatic duct, and left and right hepatic ducts are visualized after octapeptide of cholecystokinin.

bly decrease the number of film exposures and amount of radiation required to perform oral cholecystography.

SUMMARY

The effect of an intravenous injection of 20 ng./kg. of the C-terminal octapeptide of cholecystokinin (OP-CCK) on gallbladder contraction was investigated in 30 adult patients undergoing oral cholecystography.

The mean peak decrease in gallbladder size was 48 per cent. Less than 40 per cent decrease in gallbladder size occurred in 4 patients. An additional injection of 40 ng./kg. of OP-CCK in these 4 patients produced further contraction in only 1 patient.

The common bile duct was visualized in 47 per cent of patients after 20 ng./kg. OP-CCK. Common bile duct visualization was not increased by a second injection of 40 ng./kg.

The side effects were mild and transient. No evidence of toxicity was found on physical examination or laboratory evaluation.

This study provides evidence that OP-

TABLE II

SYMPTOMS FOLLOWING 20 NG./KG. OCTAPEPTIDE OF CHOLECYSTOKININ

Symptoms	Number of Subjects
None	5
Nausea	15
Abdominal cramps	9
Abdominal pressure or other discomfort	9

CCK, 20 ng./kg., is safe and effective for use as a gallbladder contracting agent in man.

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REFERENCES

1. Backlund, V. Kolecystokinin vid röntgenundersökningar. *Läkartidningen*, 1967, *64*, 2473–2476.
2. Hedner, P., and Lunderquist, A. Use of C-terminal octapeptide of cholecystokinin for gallbladder evacuation in cholecystography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 320–326.
3. Jorpes, J. E., and Mutt, V. Secretin, pancreozymin and cholecystokinin: their preparation and properties. *Gastroenterology*, 1959, *36*, 377–385.
4. Levant, J. A., Kun, T. L., Sturdevant, R. A., and Isenberg, J. I. Effect of graded doses of C-terminal octapeptide of cholecystokinin of intestinal transit in man (abst.). *Gastroenterology*, 1973, *64*, 759.
5. Ondetti, M. A., Rubin, B., Engel, S. L., Pluscec, J., and Sheehan, J. T. Cholecystokinin-pancreozymin: recent developments. *Am. J. Digest. Dis.*, 1970, *15*, 156–159.
6. Rubin, B., Engel, S. L., Drungis, A. M., Dzelzkalns, M., Grigas, E. O., Waugh, M.H., and Yiacas, E. Cholecystokinin-like activities in guinea pigs and dogs of C-terminal octapeptide (SQ 19 844) of cholecystokinin. *J. Pharmacol. Sc.*, 1969, *58*, 955–959.
7. Sturdevant, R. A. L., Stern, D. H., Resin, H., and Isenberg, J. I. Effect of graded doses of octapeptide of cholecystokinin of gallbladder size in man. *Gastroenterology*, 1973, *64*, 452–456.
8. Valbert, L. S., Jabarri, M., Kerr, J. W., Curtis, A. C., Ramehand, S., and Prentice, R. S. A. Biliary pain in young women in absence of gallstones. *Gastroenterology*, 1971, *60*, 1020–1026.

INTRAHEPATIC ARTERIAL PORTAL COMMUNICATIONS: ANGIOGRAPHIC STUDY*

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TEL-HASHOMER, AND TEL-AVIV, ISRAEL

INTRAHEPATIC arterial portal shunts have been well documented angiographically in liver cirrhosis,[4,10,11] but have only been occasionally reported in other pathologic conditions.[2,4,5,7–11]

The purpose of this paper is to describe the angiographic features of intrahepatic arterial portal communications as observed in different pathologic conditions.

Normally, the terminal hepatic arterial branches and portal vein radicles enter into the same sinusoidal area within the liver acinus, resulting in an appropriate interconnection between these 2 systems. The outflow for both hepatic arterial and portal venous blood is *via* a central hepatic vein.[6] Occlusion of the normal outflow of blood from the liver or intrahepatic arteriovenous shunts alter the normal flow pattern. These altered hemodynamics can be demonstrated by selective hepatic arteriography.

A. OBSTRUCTION OF THE NORMAL VENOUS OUTFLOW

When the hepatic veins are occluded, as in the Budd-Chiari syndrome, the major route of egress of arterial blood from the liver is *via* the hepatic sinusoids into the portal system and by reversed hepatofugal flow to the systemic venous system through gastric, esophageal and mesenteric collaterals.[2,8]

In liver cirrhosis with severe portal hypertension, the primary site of vascular obstruction is at the distal end of the sinusoid or in the proximal venule. This postsinusoidal block causes reduction in the size of the venous bed of the portal vein,[10] and when advanced hypertension is present, portal flow is often reversed. The increased hepatic arterial flow that may coexist is drained in part by retrograde portal flow. In the presence of these altered hemodynamics various vascular intrahepatic shunts exist, in particular intrahepatic arterial portal shunts and hepatic portal venous communications.[4,10,11]

The different angiographic patterns observed in the Budd-Chiari syndrome and in liver cirrhosis with severe portal hypertension make it possible to differentiate between these 2 forms of venous outflow obstruction.[1,2]

In the Budd-Chiari syndrome selective hepatic angiography shows stretching of the hepatic artery branches.[1] In the late capillary phase opacification of the portal vein branches occurs (Fig. 1, *A* and *B*).

It should be noted, however, that in 20 per cent of cases with hepatic vein thrombosis a concomitant thrombosis of the portal vein is present.[8] In these cases hepatofugal flow into the portal vein may not occur, but the stretching of the hepatic arterial branches will still be evident.

In contrast to this, selective hepatic angiography in liver cirrhosis reveals intrahepatic arterial branches of decreased, normal or increased caliber; however, they generally appear distorted and present a characteristic spiral course. During the capillary phase the liver may appear mottled. Retrograde visualization of the portal vein radicles may occasionally be observed with advanced portal hypertension.[10,11]

B. SYSTEMIC-PORTAL ARTERIOVENOUS COMMUNICATION

1. ARTERIOVENOUS FISTULA

Filling of the portal vein or one of its branches from the hepatic artery may also

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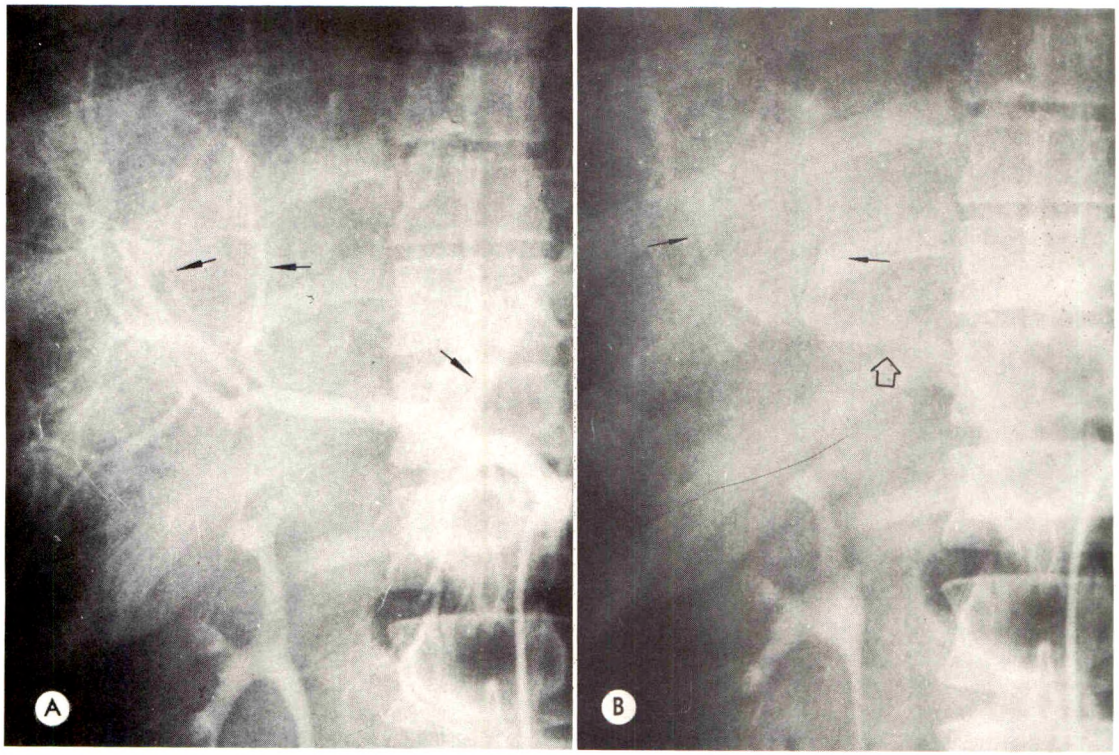


FIG. 1. A 24 year old male with primary veno-occlusive disease of the hepatic veins proven at autopsy.

Selective hepatic arteriography. (*A*) Arterial phase shows stretching of the hepatic artery branches (arrows). (*B*) Parenchymatous phase reveals retrograde filling of the portal radicles (arrows) and portal vein (open arrow).

occur in the presence of intrahepatic systemic portal arteriovenous fistulae. These fistulae may be congenital, traumatic, arteriosclerotic or idiopathic in nature.[3,5] Angiography may reveal opacification of the portal vein or one of its branches during the arterial phase of the examination. If the filming rate is 4 per second or more, the site of communication of the arteriovenous fistula may be easily demonstrated (Fig. 2).

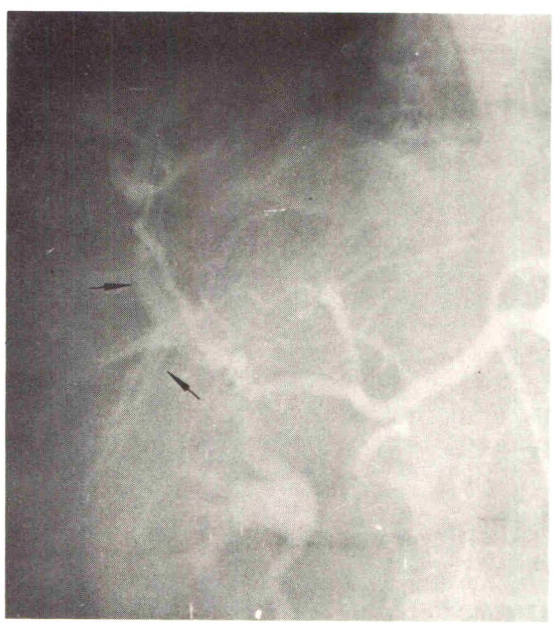


FIG. 2. Traumatic arteriovenous fistula of the liver in a 52 year old female.

Selective hepatic arteriography reveals, in the early arterial phase, retrograde filling of the portal branches (arrows).

2. TUMORS OF THE LIVER

The intrahepatic portal radicles together with the portal vein are occasionally filled in a retrograde manner in some cases of hepatoma.[4,9] These neoplasms are supplied by branches of the hepatic artery and can be recognized by the presence of tumor vessels. Neovascularity is a most characteristic feature in the angiographic appearance of hepatoma.[9] Tumor vessels are large, tortuous, irregular and form bizarre vascular networks (Fig. 3, *A* and *B*). Arterio-

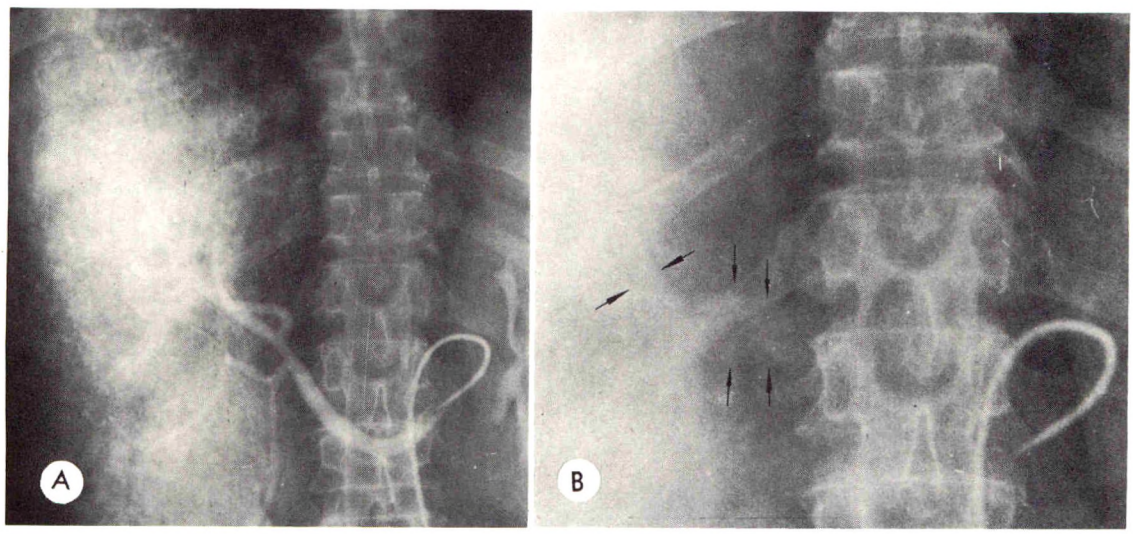


Fig. 3. Primary hepatoma of the liver.

Selective hepatic arteriography. (*A*) Arterial phase. (*B*) In the late parenchymatous phase retrograde filling of the portal vein is clearly demonstrated (arrows).

venous shunts may also be present, with filling of irregular vascular lakes and early visualization of portal radicles which may exhibit irregular luminal defects secondary to tumor thrombosis (Fig. 4). Occasionally these tumors are complicated by portal vein thrombosis. Visualization of the portal vein in these cases is unexpected.

Highly vascular metastatic neoplasms of the liver such as leiomyosarcoma or hemangiosarcoma may also show on the selective hepatic angiogram opacification of the portal radicles with hepatofugal flow, because of arteriovenous communications within the tumor (Fig. 5). Visualization of portal radicles on selective hepatic angiog-

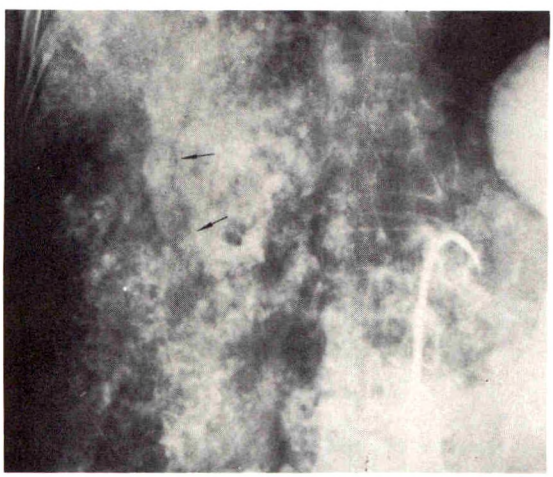


Fig. 4. Celiac arteriography in a case of a highly vascular hepatoma demonstrates retrograde filling of large branches of the portal vein (arrows).

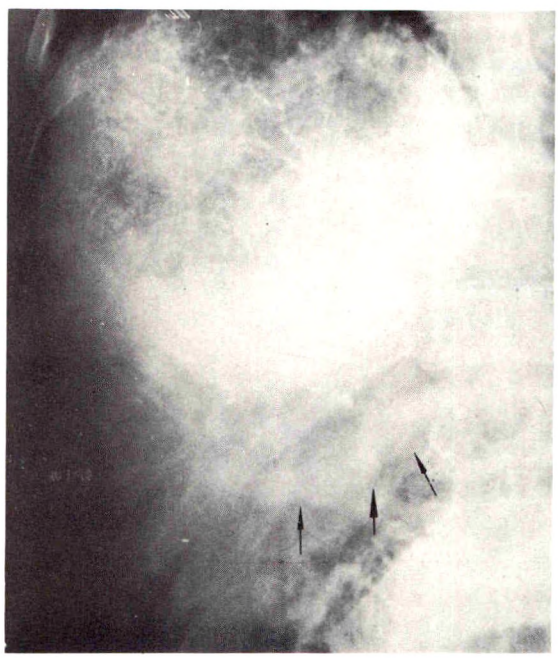


Fig. 5. A huge solitary metastasis of the right lobe of the liver in a patient with leiomyosarcoma of the small bowel proved at operation.

Arteriography reveals in the late arterial phase early retrograde filling of the portal vein which is displaced caudally (arrows).

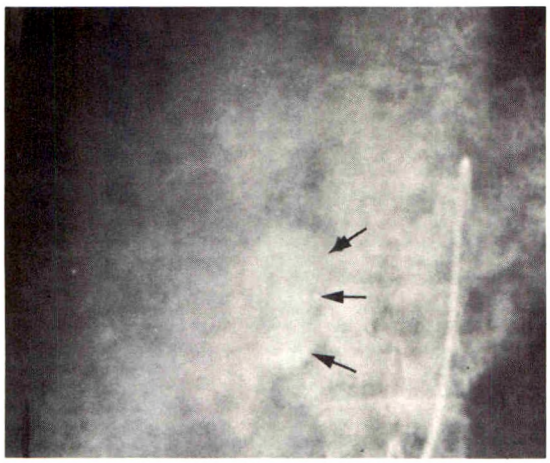


Fig. 6. A 52 year old male with diffuse benign hemangioma of the liver which has been proven by open biopsy.

Celiac arteriography, in the late arterial phase, shows retrograde filling of the portal vein (arrows).

raphy in the presence of a highly vascularized tumor in the liver indicates invasion of portal radicles by the tumor with tumorous arteriovenous shunts.

Retrograde filling of the portal radicles may occur rarely in large hemangiomas of the liver. These benign tumors consist of wide, irregular, tortuous sinusoids and vascular spaces or lakes. They are supplied by normal or only slightly enlarged arteries and may displace adjacent hepatic vessels.[7] The egress of arterial blood from the liver is usually *via* the hepatic veins, but in some of the cases retrograde filling of the portal radicles may be observed (Fig. 6). The reversed hepatofugal flow may occur *via* intrahepatic shunts between hepatic and portal venules or by reversal of the sinusoidal circulation.[7]

Postsinusoidal elevation of venous pressure or direct communication between intrahepatic arterial and portal radicles may cause hepatofugal flow into the portal system and retrograde portal visualization on selective hepatic angiography. This occurs in the Budd-Chiari syndrome, in some cases of cirrhosis with severe portal hypertension, in intrahepatic arteriovenous fistulae, as well as in some cases of hepatoma, in hypervascular metastatic tumors and in large hemangiomas of the liver.

SUMMARY

Several examples of intrahepatic arterial portal communications are demonstrated.

The only way to differentiate between the various etiologies of this angiographic phenomenon is by the analysis of the different patterns of the intrahepatic arterial branches, which are usually characteristic for each pathologic process.

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REFERENCES

1. Deutsch, V., Rosenthal, T., Adar, R., and Mozes, M. Budd-Chiari syndrome: study of angiographic findings and remarks on etiology. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 430–439.
2. Deutsch, V., Adar, R., Bogokowsky, H., Itzchak, Y., and Mozes, M. Diversion of hepatic arterial flow into portal vein in Budd-Chiari syndrome. To be published.
3. Donovan, A. J., Reynolds, T. B., Mikkelsen, W. P., and Peters, R. L. Systemic portal arteriovenous fistulas: pathologic and hemodynamic observations in two patients. *Surgery*, 1968, *66*, 474–482.
4. Farrell, R., Steinman, R., and Green, W. H. Arteriovenous shunting in regenerating liver simulating hepatoma. *Radiology*, 1972, *102*, 279–280.
5. Foley, W. J., Turcotte, J. G., Hoskins, P. A., Brant, R. L., and Ause, R. G. Intrahepatic A-V fistula between hepatic artery and portal vein. *Am. J. Surg.*, 1971, *174*, 849–855.
6. Marks, C. Developmental basis of portal venous system. *Am. J. Surg.*, 1969, *117*, 671–681.
7. Pantoja, E. Angiography in liver hemangioma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 874–879.
8. Pollard, J. J., and Nebesar, R. A. Altered hemodynamics in Budd-Chiari syndrome demonstrated by selective hepatic and selective splenic angiography. *Radiology*, 1967, *89*, 236–243.
9. Reuter, S. R., Redman, H. C., and Siders, D. B. Spectrum of angiographic findings in hepatoma. *Radiology*, 1970, *94*, 89–94.
10. Viamonte, M. W., Martinez, L. O., Parks, R. E., Warren, W. D., and Fomon, J. J. Liver shunts. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 773–775.
11. Viamonte, L. O. Angiographic investigation in portal hypertension. *Surg., Gynec. & Obst.*, 1970, *130*, 37–53.

PYOGENIC LIVER ABSCESS

ANGIOGRAPHIC DIAGNOSIS AND TREATMENT BY CLOSED ASPIRATION

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THE high mortality seen with pyogenic hepatic abscesses is in part due to a delay in diagnosis.[1,2] Earlier diagnosis and more precise localization may be accomplished by radioisotopic liver scanning and hepatic arteriography. The only successful method of therapy has been external surgical drainage with antibiotics as a valuable adjunct.[2,3]

Four cases of pyogenic hepatic abscess studied by angiography are described. Two of these cases were initially managed by fluoroscopically guided percutaneous transcatheter drainage as an alternative to surgical intervention.

REPORT OF CASES

CASE I. F.W., a 48 year old Negro female who had an ovarian neoplasm removed 6 years earlier, developed generalized weakness, chills, and fever. Anemia and a purulent right pleural effusion were discovered. In spite of the drainage of a subhepatic purulent collection, the patient's chills and fever continued.

A liver scan with Tc^{99m} sulfur colloid revealed a "cold" area in the right lobe (Fig. 1*A*). An upper gastrointestinal examination disclosed an ulcerated submucosal mass in the descending limb of the duodenum.

Celiac and superior mesenteric arteriography demonstrated curvilinear displacement of the superior portions of the anterior and posterior segmental branches of the right hepatic artery by an avascular mass (Fig. 1*B*). A well defined hypervascular lobulated neoplasm was opacified to the right of the gastroduodenal artery coinciding with the findings on the upper gastrointestinal examination.

Exploratory laparotomy confirmed the presence of a hepatic abscess which was drained, and a perforated leiomyoma of a second portion of the duodenum was resected. *Enterobacter* and anaerobic *Streptococci* were cultured from the abscess and she was treated by chloramphenicol and penicillin. She did well following surgery with complete resolution of symptoms.

CASE II. M.D., a 62 year old white female, was admitted because of abdominal pain, chills, and fever. Approximately 6 weeks prior to this admission, the patient was hospitalized for perforation of a sigmoid diverticulum which was treated by a transverse colostomy and antibiotics. She had a 3 year history of scleroderma and a sympathectomy for the treatment of Raynaud's phenomenon of the hand.

A liver scan showed a large "cold" area in the right lobe of the liver. Celiac arteriography revealed marked displacement of the branches of the right hepatic artery outlining a large lobular avascular mass with thin hypervascular margins considered to be a multilocular abscess 14 cm. in maximum dimension (Fig. 2, *A* and *B*).

Exploratory laparotomy confirmed the presence of an abscess in the right lobe of the liver which was drained. A gram-negative rod was grown from her blood stream and identified as a *Proteus morganii*. No specific organism was grown from the abscess. The patient was given gentamycin and responded well to this treatment.

CASE III. J.A., a 19 year old white male, was admitted in March 1970 with persistent leukocytosis 4 weeks after an upper respiratory infection. A bone marrow examination revealed

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acute myelogenous leukemia and the patient was referred to M. D. Anderson Hospital.

Physical examination disclosed lymphadenopathy and hepatosplenomegaly. He improved on chemotherapy COPP (Cytoxan, Oncovid, Prednisone, and Procarbazine). Pancytopenia resulted, accompanied by a gram sepsis and bilateral pleural effusion which was treated by antibiotics, carbenicillin, keflin, and kanamycin. Acute right upper quadrant abdominal pain precipitated an exploratory laparotomy on May 1, 1970. Acute gangrenous cholecystitis and a pericholecystic abscess were found. The abscess was drained and cholecystostomy was performed. The patient remained febrile and his condition continued to deteriorate.

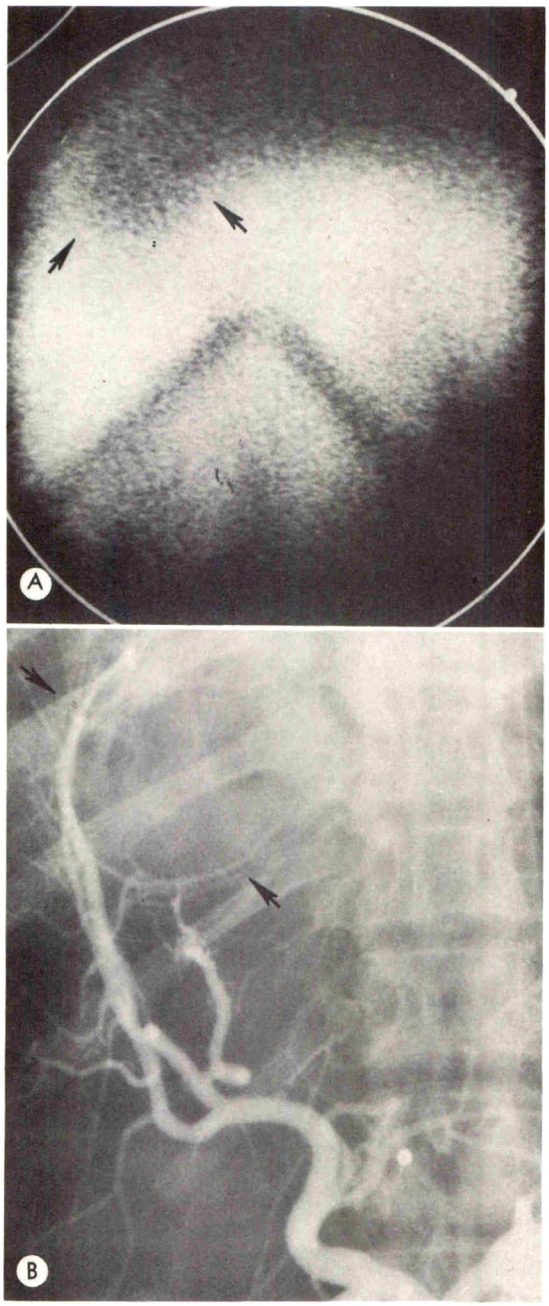


FIG. 1. Case 1. (*A*) Tc^{99m} sulfur colloid liver scan demonstrates cold area in right lobe. (*B*) Celiac and superior mesenteric arteriogram demonstrates curvilinear displacement of the superior portions of the anterior and posterior segmental branches of the right hepatic artery by an avascular mass.

A Tc^{99m} sulfur colloid liver scan delineated a "cold" area in the right lobe of the liver. Celiac arteriography on May 10, 1970, demonstrated curvilinear displacement of the superior branches of the anterior and posterior segments of the right hepatic artery (Fig. 3*A*). A radiolucent defect surrounded by a relatively thick zone of hypervascularity was visualized (Fig. 3*B*). These findings along with his clinical picture led to the consideration of a hepatic abscess.

In view of the patient's poor general status, it was felt that he would not tolerate surgical intervention. Utilizing the liver scan and angiogram for localization, percutaneous drainage under fluoroscopic guidance was undertaken. After preparing the skin with betadine and local anesthesia, a small incision was made in the right anterior axillary line at the midpoint of the lateral aspect of the liver shadow. The patient was asked to suspend respiration in midcycle or in the position of rest and the abscess was punctured with a 6 inch polyethylene sheathed 18 gauge needle. Approximately 20 cc. of purulent material was removed for culture and air and water based contrast material were injected to outline the cavity. A .035 inch guidewire was inserted through the polyethylene sheath which was then withdrawn and a radiopaque polyethylene catheter .055 inch ID, .075 inch OD with 2 side holes placed near the tip was introduced. The entire procedure was repeated with another catheter at a slightly different angle, so that the 2 catheters would allow adequate irrigation with normal saline solution and drainage (Fig. 3*C*). The cultures revealed *Bacteroides*. The first catheter came out after 3 days with no obvious bleeding. The second catheter remained in the cavity for 10 days and came out uneventfully. He responded to this regimen and the chemotherapy was resumed. On June 24, 1970, an elective cholecystectomy was performed.

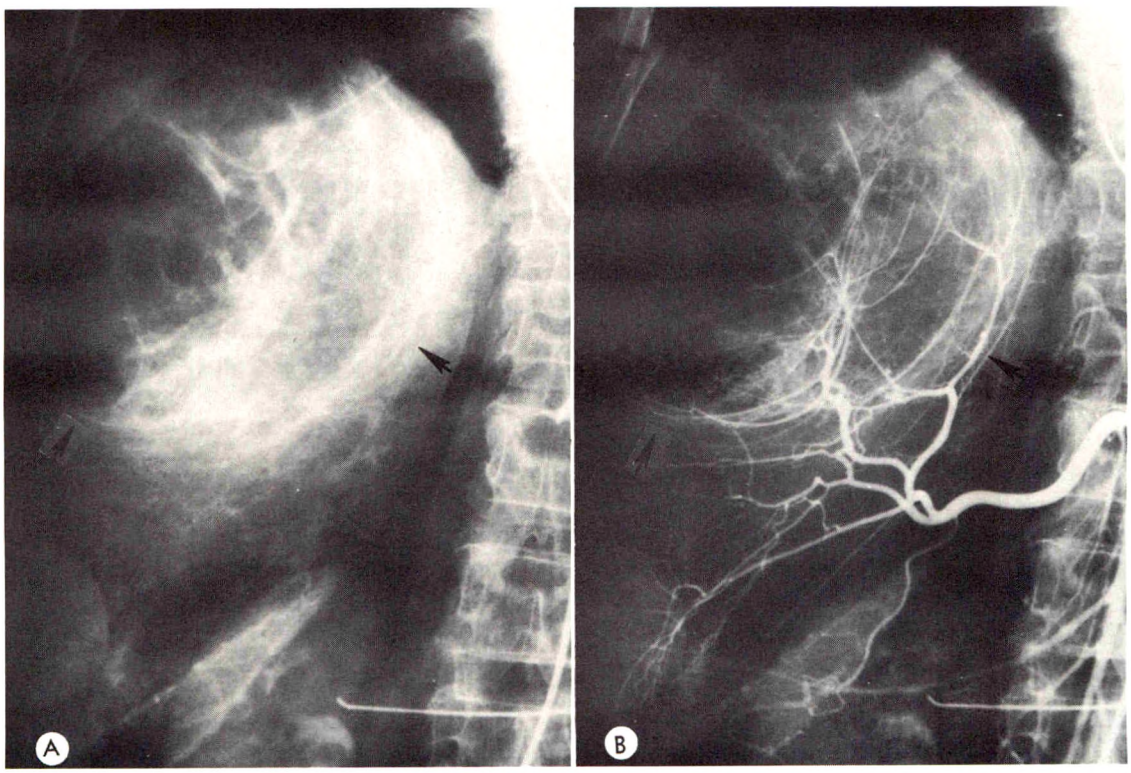


FIG. 2. Case II. (*A* and *B*) Celiac arteriograms demonstrate marked displacement of the branches of the right hepatic artery outlining a large lobular avascular mass with thin hypervascular margins.

CASE IV. D.B., a 53 year old white male, had for 2 weeks a persistent elevation of temperature to 103°F. in spite of antibiotic treatment. Bone marrow examination was interpreted as being suspicious of acute leukemia and he was referred to M. D. Anderson Hospital.

On admission he complained of night sweats, chills, and fever. Laboratory studies revealed a leukocytosis of 13,000 per mm.^{3}; hemoglobin 11.6 gm. per cent. Re-examination of his bone marrow was considered consistent with dysplastic granulocytic hyperplasia with no definite evidence of acute leukemia. *Bacteroides* was consistently found in blood cultures. Chest roentgenogram revealed a right pleural effusion with an associated inflammatory reaction at the right base.

A liver scan demonstrated a large "cold" area in the posterolateral aspect of the right lobe of the liver. Celiac angiography performed on May 1, 1972, confirmed the presence of a mass in the right lobe localized by splaying of the superior portions of the anterior and posterior segmental branches of the right hepatic artery. No tumor vascularity was noted. In the hepatogram phase a relatively thin zone of hypervascularity surrounded the avascular mass (Fig. 4, *A* and *B*).

As in the preceding case, percutaneous needle aspiration was performed under fluoroscopic guidance. A milky yellow exudate with a foul odor was withdrawn and cultured. Water soluble contrast material was injected into the cavity outlining the irregular contour and septation within the cavity. Catheters were inserted for irrigation with antibiotics. A smaller abscess and a fistulous tract communicated with the larger cavity (Fig. 4*C*). *Bacteroides* was grown from the abscess cavity contents. Follow-up injections of the cavity with contrast medium through the catheter demonstrated disappearance of both the smaller abscess cavity and fistulous tract. In spite of this the patient continued a febrile course. In search for a primary focus of infection, an intravenous cholangiogram revealed nonvisualization of the gallbladder and a normal biliary tree. The catheters remained in place until May 17, 1972, when a cholecystectomy, abscess drainage, and biopsy of the wall of the residual cavity were performed. Chronic

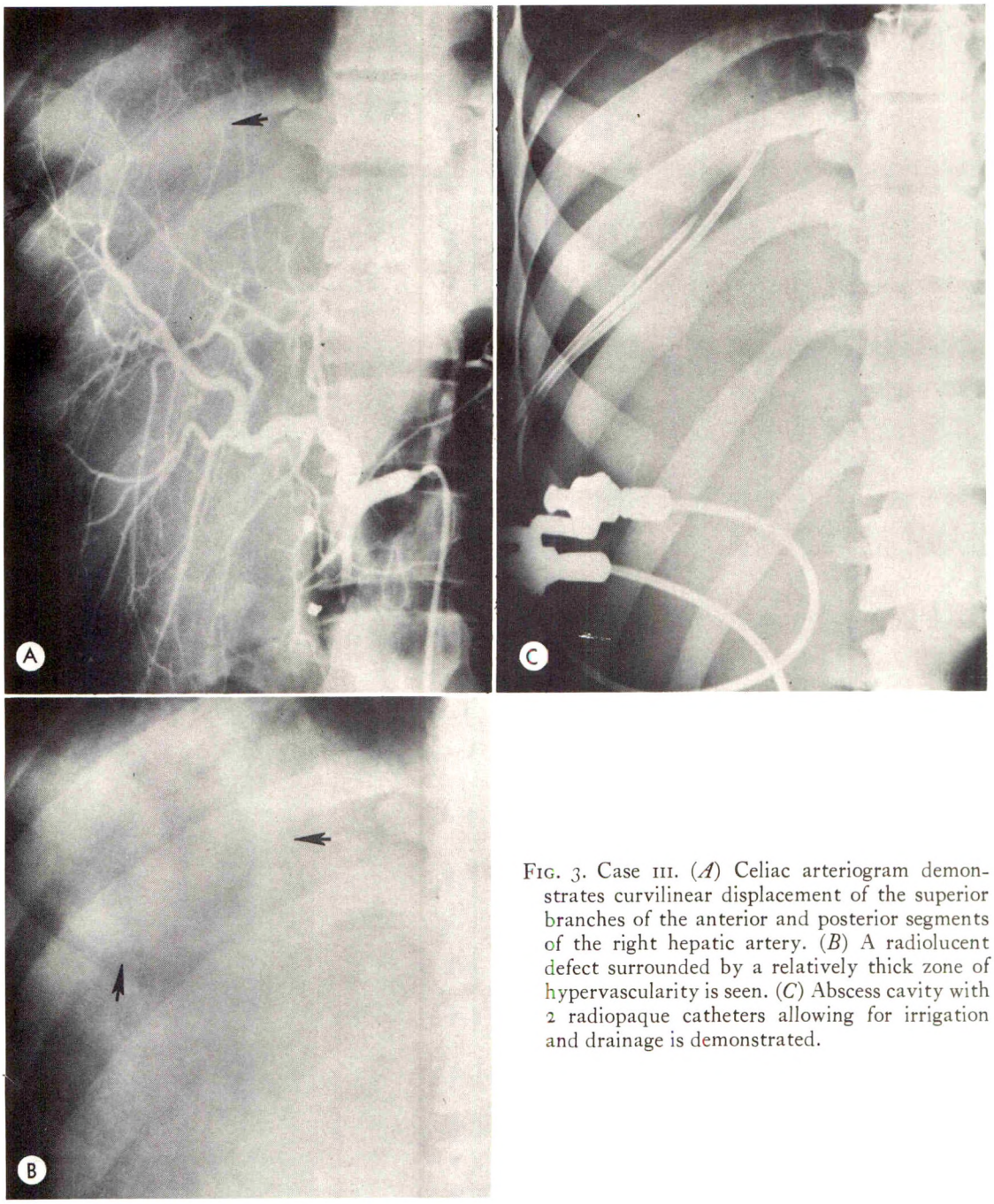


Fig. 3. Case III. (*A*) Celiac arteriogram demonstrates curvilinear displacement of the superior branches of the anterior and posterior segments of the right hepatic artery. (*B*) A radiolucent defect surrounded by a relatively thick zone of hypervascularity is seen. (*C*) Abscess cavity with 2 radiopaque catheters allowing for irrigation and drainage is demonstrated.

cholecystitis with cholelithiasis was found and the presence of pyogenic abscess was histologically confirmed. The patient did well until May 30, 1972, when he developed fever and tenderness about his incision. Repeat laparotomy was negative. The patient improved and was discharged.

DISCUSSION

Liver abscess has been a clinical entity of life threatening importance. It has been reported in approximately 1 per cent of postmortem examinations.[8]

Hepatic abscesses may be pyogenic,

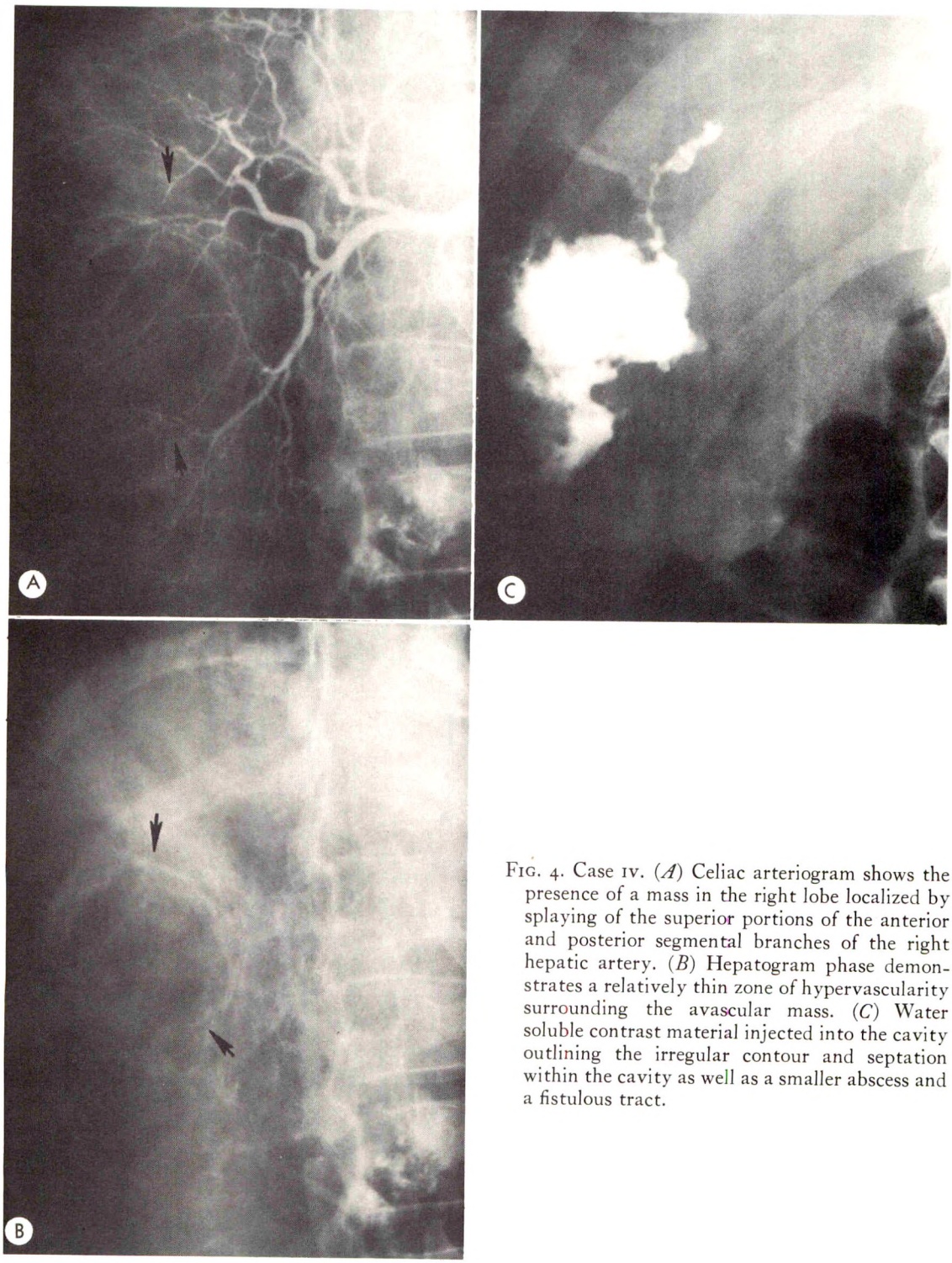


FIG. 4. Case IV. (*A*) Celiac arteriogram shows the presence of a mass in the right lobe localized by splaying of the superior portions of the anterior and posterior segmental branches of the right hepatic artery. (*B*) Hepatogram phase demonstrates a relatively thin zone of hypervascularity surrounding the avascular mass. (*C*) Water soluble contrast material injected into the cavity outlining the irregular contour and septation within the cavity as well as a smaller abscess and a fistulous tract.

amebic, or echinococcal in origin. Pyogenic abscesses are primarily caused by anaerobic bacteria[4,5] with the *Bacteroidacea*[17] group the most frequent. These organisms are in the normal intestinal flora, increasing in the distal half of the small intestine and becoming the major organism in the stool. The difficulty in culturing *Bacteroides*, because of their slow growth, is in part responsible for negative cultures with the mistaken diagnosis of amebic etiology. Systemic sepsis, portal spread, ascending biliary tract infections and trauma are some of the mechanisms suggested in the production of pyogenic hepatic abscesses.[4,5,15]

A persistent leukocytosis is seen[4,5] and at times even a leukemoid reaction, as was found in Case IV. Liver function studies are usually normal. Roentgenographic examination of the chest often shows elevation of the right hemidiaphragm and a right pleural effusion as a reaction to the hepatic abscess which is most frequently located in the superior aspect of the right lobe, as is seen in the cases described. Displacement of adjacent organs, in particular the right kidney and gastrointestinal tract, the hepatic flexure of the colon or the duodenum is usually secondary to hepatomegaly.

Radioisotopic liver scan and hepatic arteriography are complementary procedures in the diagnosis, localization, and the determination of the size and number of hepatic lesions. Areas of diminished uptake less than 2 cm. in diameter are beyond the competence of the radioactive isotopic liver scan. Masses larger than 3 cm. are readily detected.[14] This is of especial importance in the evaluation of the left lobe, which is difficult by arteriography. The specific etiology of a "cold" area on the liver scan cannot be determined.[4,5,15] Therefore, an abscess presents as a localized rounded area of diminished uptake indistinguishable from any mass lesion, a cyst, primary or secondary neoplasm, etc.

Arteriography offers additional information. The vascular anatomy aids in more exact localization. Abscesses are most common in the dome of the liver, distorting the superior portions of both the anterior and posterior segments of the right hepatic artery. Hypervascular lesions less than 2 cm. in diameter are better appreciated by arteriography than by liver scan. Avascular processes under 2 cm. in diameter are difficult to define by either scanning or arteriography. Above 2 cm., avascular lesions may be visualized by distortion and displacement of the arteries as well as the localized areas of relative radiolucency during the hepatogram phase.[14] The left lobe of the liver, in particular the left lateral segment, is difficult to evaluate by angiography. Separation of the superior and inferior branches of the left lateral hepatic artery may be due to a pathologic process or compensatory hypertrophy. Superimposition of the left gastric and splenic branches adds to the confusion. Superselective hepatic arteriography as well as oblique views may assist in the evaluation of the left lobe.

Arteriography is of considerable assistance in establishing the specific etiology. The 4 cases studied reveal a gamut of changes in hepatic abscesses, probably related to the organization of the wall of the abscess. There is usually displacement and distortion of the arteries. A halo of increased vascularity around an avascular area may occur, possibly representing compressed normal parenchyma or reactive hyperemia about the abscess cavity.[19] Abnormal vascularity is not usually seen.[20] Rarely, arteriovenous shunting with opacification of the hepatic veins has been reported in hepatic abscesses.[19]

Although the arteriogram supplies more descriptive information, differentiation from other entities may be difficult. A hepatic cyst usually has a sharp transition zone between the avascular cyst and the normal tissue, at times presenting as a thin dense line of compressed tissue. Primary or secondary neoplasms of the liver may be confused with an abscess.[21] Hemorrhage into a benign or malignant neoplasm may present as an avascular mass with a hyper-

vascular margin. Metastatic malignancies, in particular leiomyosarcoma and colon carcinoma, may have a vascularized wall of variable thickness, surrounding a central area of avascularity which at times represents necrosis. Angiographic findings in metastatic disease usually mirror the vascularity of the primary lesion.[1] In Case I the mass in the liver was avascular in contrast to the hypervascular duodenal process. This suggested that the liver lesion was an abscess rather than metastasis.

Other etiologic agents angiographically indistinguishable from pyogenic abscess are hematoma and abscess produced by ameba or echinococcus.

The untreated pyogenic abscess has a reported mortality of 100 per cent.[2,3,10] Successful therapy is only accomplished by surgical drainage of the abscess, usually combined with systemic antibiotics. Percutaneous aspiration, because of the possibility of peritoneal and pleural contamination,[11,17] has been reserved by some for the gravely ill patient in whom the operative procedure itself might be accompanied by a high risk.[15,16,18] However, percutaneous closed aspiration under fluoroscopic guidance has been employed as the primary or at times the initial mode of treatment for both amebic or pyogenic abscesses.[6,9,13] Crane *et al.*[6] utilized this approach in the management of 200 patients with amebic abscesses in the liver. McFadzean *et al.*[13] reported a success rate of 100 per cent in the treatment of solitary pyogenic abscesses of the liver by closed aspiration and antibiotics. Additional advantages of this approach are: the ease of establishing a specific diagnosis; determining the causative organism; and following the response to therapy by injection of water based contrast material or air into the cavity.[7]

This procedure was followed in the management of Cases III and IV. Localization of the pyogenic abscess was accomplished by the scan. Hepatic arteriography confirmed the localization and specifically established the diagnosis of a multilocular abscess in Case IV. Surgical drainage was considered too great a risk in these patients. In Case III percutaneous closed aspiration successfully managed the hepatic abscess. This allowed an opportunity to better prepare the patient for the subsequent elective cholecystectomy. The multilocular abscess in Case IV which was considered less likely to respond to this approach than a solitary abscess, markedly improved following percutaneous aspiration, but the persistent temperature elevation was either due to the residual abscess or the patient's chronic cholecystitis.

Exploratory laparotomy, cholecystectomy, and drainage of the abscess were necessary for adequate treatment.

SUMMARY

Four cases of pyogenic liver abscess were diagnosed and localized by angiography and liver scan.

Closed drainage by percutaneous aspiration under fluoroscopic guidance was employed for treatment in 2 cases.

The use of closed aspiration along with appropriate antibiotics can decrease morbidity and mortality as well as adequately manage the patient with pyogenic liver abscess.

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REFERENCES

1. Alfidi, R. J., Rastogi, H., Buonocore, E., and Brown, C. H. Hepatic arteriography. *Radiology*, 1968, *90*, 1136–1142.
2. Altemeier, W. A. Recent trends in management of hepatic abscesses. *Delaware M. J.*, 1971, *43*, 327–336.
3. Altemeier, W. A., Schowengerdt, C. G., and Whitely, D. H. Abscesses of liver: surgical considerations. *A.M.A. Arch. Surg.*, 1970, *101*, 258–266.
4. Block, M. A. Principles in management of liver abscesses. *Am. J. Surg.*, 1968, *115*, 587–588.
5. Block, M. A. Suspect liver abscess and you may save life. *Consultant*, April, 1972, 141–142.
6. Crane, P. S., Lee, Y. T., and Seel, D. J. Experience in treatment of two hundred pa-

tients with amebic abscess of liver in Korea. *Am. J. Surg.*, 1972, *123*, 332–337.
7. Fariss, B. L. Amebic liver abscess. *Am. J. Gastroenterol.*, 1965, *44*, 559–564.
8. Foster, S. C., Schneider, B., and Seaman, W. B. Gas-containing pyogenic intrahepatic abscesses. *Radiology*, 1970, *94*, 613–618.
9. Gaisford, W. D., and Mark, J. B. D. Surgical management of hepatic abscess. *Am. J. Surg.*, 1969, *118*, 317–326.
10. Grant, R. N., Morgan, L. R., and Cohen, A. Hepatic abscesses. *Am. J. Surg.*, 1969, *118*, 15–20.
11. Joseph, W. L., Kahn, A. M., and Longmire, W. P. Pyogenic liver abscess. *Am. J. Surg.*, 1968, *115*, 63–68.
12. Lee, J. F., and Block, G. E. Changing clinical pattern of hepatic abscesses. *A.M.A. Arch. Surg.*, 1972, *104*, 465–470.
13. McFadzean, A. J. S., Chang, K. S., and Wong, C. C. Solitary pyogenic abscess of liver treated by closed aspiration and antibiotics. *Brit. J. Surg.*, 1953, *41*, 141–152.
14. McLaughlin, M. J., and Gilday, D. L. Angiography and colloid scanning of benign mass lesions of liver. *Clin. Radiol.*, 1972, *23*, 377–391.
15. Ogden, W. W., Hunter, P. R., and Rives, J. D. Liver abscess. *Postgrad. Med.*, 1961, *30*, 11–19.
16. Ostemiller, W., and Carter, R. Hepatic abscess. *A.M.A. Arch. Surg.*, 1967, *94*, 353–356.
17. Paterson, H. Open aspiration for solitary liver abscess. *Am. J. Surg.*, 1970, *119*, 326–329.
18. Price, J. E., Joseph, W. H., and Mulder, D. G. Diagnosis and treatment of intrahepatic abscess. *Am. Surgeon*, 1967, *33*, 820–825.
19. Reuter, S. R., and Redman, H. C. Inflammatory disease. In: Gastrointestinal Angiography. W. B. Saunders Company, Philadelphia, 1972.
20. Rossi, P., and Ruyicha, F. Differentiation of intrahepatic and extra-hepatic masses by arteriography. *Radiology*, 1969, *93*, 771–780.
21. Viranuvatti, V., Kalayasiri, C., Cherani, O., and Plenguanit, U. Selective celiac angiography in carcinoma of liver and amebic liver abscess. *Geriatrics*, 1972, *27*, 176–180.

GALLBLADDER ECTOPIA*

A REVIEW OF ANOMALIES OF POSITION

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and STANLEY R. MANDEL, M.D., F.A.C.S.‡
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ANOMALOUS position of the gallbladder is a relatively uncommon finding. Reports of gallbladder ectopia are infrequent in the surgical and radiologic literature. Robert Gross[3] in 1936 reviewed extensively the congenital anomalies of the gallbladder, including in his survey a detailed discussion of ectopia, and of the anomalous positions previously reported. This subject was again reviewed by Caster and Flannery[2] in 1956.

Of anomalies of position of the gallbladder, the most frequently encountered is that of Levo position, in which the gallbladder is located beneath the left lobe of the liver. Intrahepatic location, almost universally subcapsular along the anterior inferior border of the right lobe of the liver, is the second most frequent anomaly of location. A single case of a gallbladder located within the anterior abdominal wall has been reported, as has a case of the gallbladder being found within the falciform ligament. Several examples of a transversely positioned gallbladder beneath the right lobe have been also discussed (Fig. 1, *A–D*).

Retroplacement of the gallbladder, on the other hand, is relatively infrequently encountered when compared with the other anomalies of position, and presents special clinical, surgical and roentgenographic problems. Gross[3] cites 5 examples of a retroplaced gallbladder, one of which was surgically proven to be retroperitoneal in location. No other case reports of retroplaced gallbladders[1] have appeared in the English language literature since the classic survey by Gross.

We report a case of retroplaced normal gallbladder which, we believe, is probably retroperitoneal in location, and, if so, would be the first such case diagnosed preoperatively.

REPORT OF A CASE

A 44 year old white female entered North Carolina Memorial Hospital with a chief complaint of chest and right upper quadrant pain. The chest pain was consistent with angina. The right upper quadrant pain had its onset 3 weeks prior to admission, was not related to food, exercise or position. Medications included nitroglycerin for the chest pain, and Elavil for depression. Physical examination revealed an obese, depressed female, but in no acute distress. Examination of all systems was essentially normal except for minimal right upper quadrant pain without rebound or guarding. Slight hepatomegaly was noted.

Evaluation of the patient's chest pain was consistent with angina and mild congestive failure. The symptoms of right upper quadrant abdominal pain were extensively evaluated, when a routine liver battery revealed mildly abnormal hepatic functions. A liver scan employing 2 millicuries of technetium sulfur colloid revealed a focus of decreased activity in the posterior portion of the right lobe of the liver which was initially interpreted as a space occupying lesion (Fig. 2, *A–D*). This finding prompted an extensive work-up for primary or secondary hepatic tumor including an upper gastrointestinal series, oral cholecystography, intravenous pyelography, barium enema examination, celiac and superior mesenteric angiography, and, eventually, an intravenous cholangiography. The above extensive investigation failed to reveal evidence for primary or secondary tumor. Nevertheless, surgical consultation was requested to evaluate the patient for possible exploratory celiotomy with a preoperative diagnosis of primary malignancy of the right lobe of the liver.

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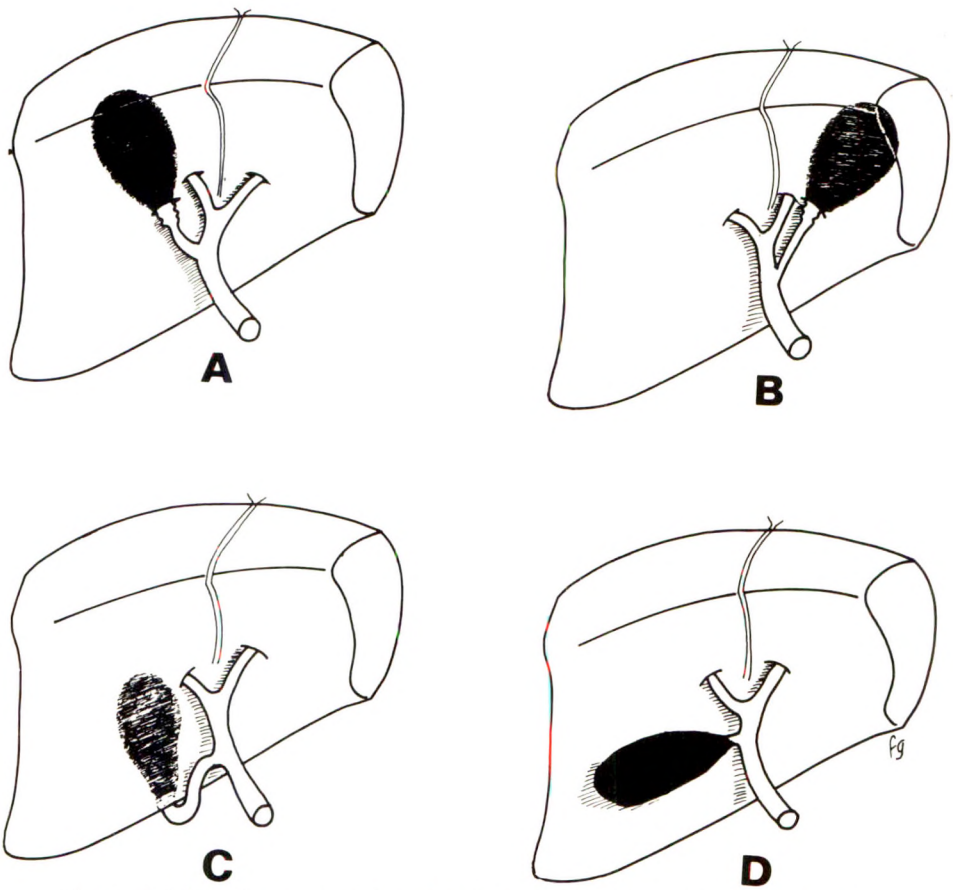


Fig. 1. (*A*) Intrahepatic location, right lobe. (*B*) Intrahepatic location, left lobe. (*C*) Retroplaced gallbladder. (*D*) Transverse position.

Prior to surgery, however, consultation between the surgical and radiologic staff, and a thoughtful review of the radiologic and laboratory findings prompted the performance of an intravenous cholangiography with posteroanterior and lateral tomograms, which revealed a retroplaced, probably retroperitoneal gallbladder, undoubtedly the cause for the focal defect seen on the posterior view of the liver scan. This having been discovered, the planned celiotomy was cancelled and the patient discharged to be followed in the out-patient department.

DISCUSSION

The embryogenesis of the biliary system occurs in the 3 mm. embryo with a thickening of endoderm at the ventral junction of the foregut and yoke sac. This thickening forms a sacculation, which divides into a craniad, and caudad portion. The craniad portion further divides into right and left lobules, grows ventrad into the septum transversum, eventually differentiating into the right and left hepatic lobes and hepatic bile ducts. The caudad portion becomes saccular, developing into a gallbladder and a common duct system. Initially the rudimentary gallbladder is attached to the ventral surface of the duodenal precursor; however, with differential growth, rotation to the right occurs, by the 7 mm. stage, which brings the attachment to the dorsal aspect of the duodenum. The gallbladder precursor then comes to lie in the free edge of the ventral mesentery. With further differential growth, the gallbladder migrates to its usual position at the anterior inferior margin of the right lobe of the liver. With

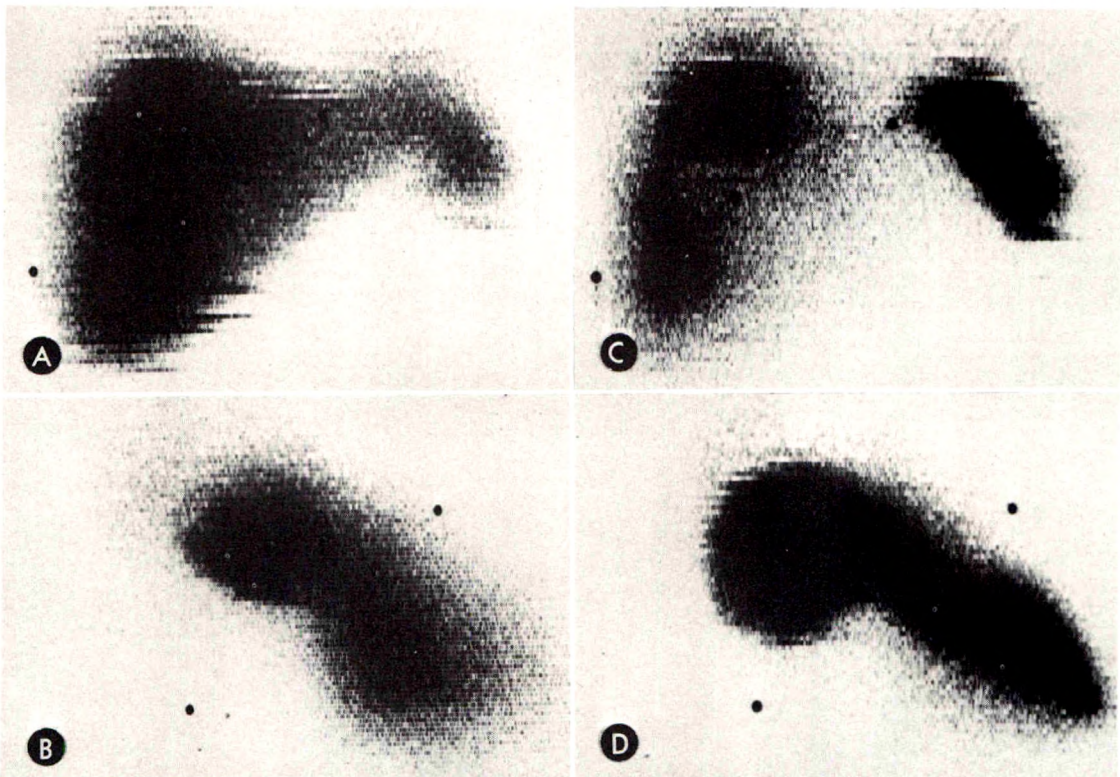


FIG. 2. (*A*) Anterior view of liver scan is normal. (*B*) The right lateral scan shows a defect posteriorly. (*C*) The posterior view shows a focal defect. (*D*) The left lateral view is normal.

anomalous migration of the gallbladder precursor, the definitive gallbladder may come to lie in any number of abnormal locations. It may be secondarily postulated, however, that an anomalously placed gallbladder may be representative of a persistent secondary gallbladder diverticulum arising from the precursor, with atrophy of the original. Most unusually, the gallbladder precursor may come to lie in the retroperitoneal space.[4]

Retroposition of the gallbladder has clinical significance in that acute cholecystitis may present as right costovertebral angle tenderness, rather than the more usual right upper quadrant manifestation. From a surgical standpoint, it is important to recognize any anomaly of position prior to the incision, so that the approach be geographically appropriate. Retroplacement of the gallbladder presents special problems in that resection is difficult due to the deep position of the gallbladder and, if retroperitoneal, may present a special problem of dissection.

It is important roentgenographically to recognize the retroplaced gallbladder, for reasons stated above, and in the case reported here, to recognize the possibility that a retroplaced, or indeed any anomalously positioned gallbladder may present as an intrahepatic mass lesion on liver scan. A retrospective review of the rather extensive roentgenographic work-up to which our patient was subjected revealed a number of features concerning the retroplaced gallbladder. The liver scan should not have been confusing, if notation had been taken of the fact that the apparent mass was visualized only in the posterior view, the anterior and lateral views being normal (Fig. 2, *A–D*). The oral cholecystogram, in retrospect, was specific in that with rotation of the patient to the right the gallbladder silhouette moved to the left, and with rotation to the left, the gallbladder

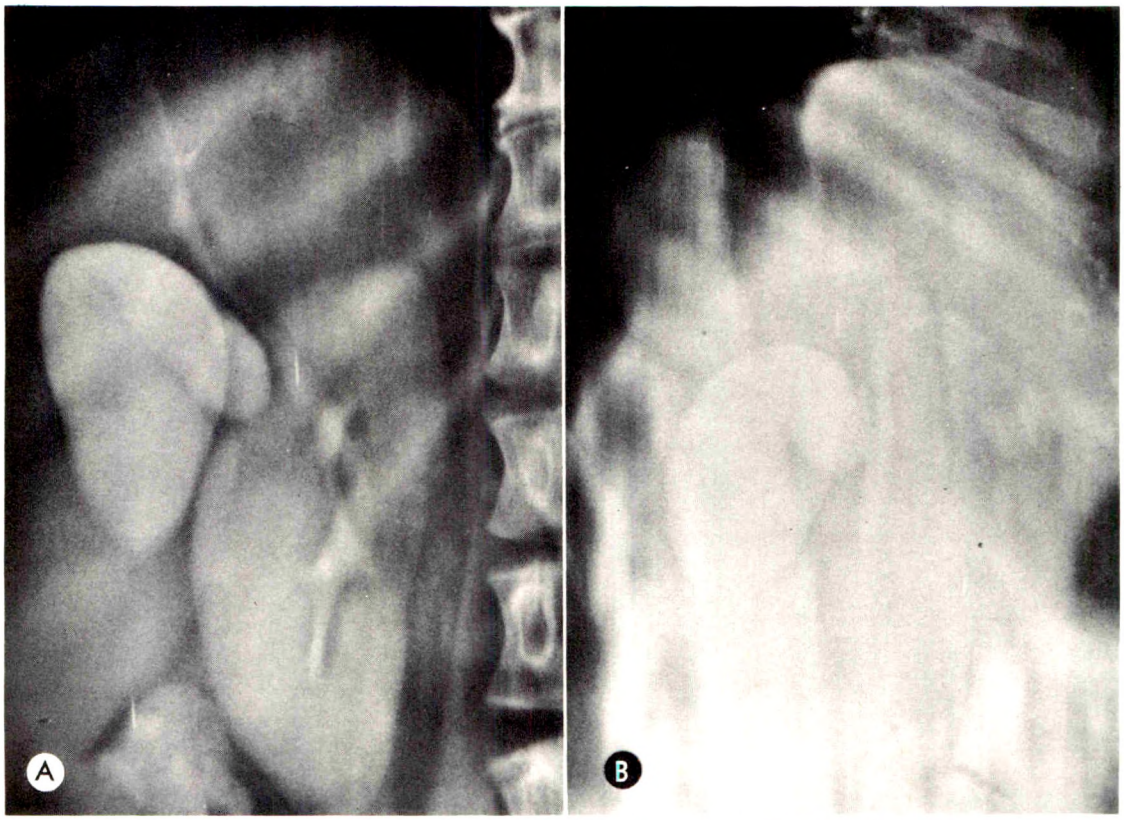


Fig. 3. (*A*) Anterior tomogram of intravenous cholangiography. The position of gallbladder corresponds to the defect on the liver scan. (*B*) Lateral tomogram demonstrates posterior position of the gallbladder.

shadow projected to the right. This is in contradistinction to the usual case with anterior placed gallbladder. With intravenous pyelography, fortuitously concomitant with the oral cholecystography, it would be appreciated that on the oblique films, the gallbladder moved with the kidney. Angiography undertaken in this patient in quest of the artifactual mass is generally not necessary to establish the diagnosis, but was informative in that the cystic artery exhibited a somewhat anomalous course, arising rather more distally on the right hepatic artery than is usual. The definitive diagnostic procedure was an intravenous cholangiography which confirmed the retroplaced and probably retroperitoneal location of the gallbladder, and disclosed an otherwise normal ductal anatomy (Fig. 3, *A* and *B*). Although not carried out in our work-up, a rose-bengal scan would, of course, reveal a concentration of nuclide in the cold region demonstrated by the technetium sulfur colloid scan.

The differential diagnosis of the above described retroplaced gallbladder should include an intrahepatic gallbladder. Although certainly not an impossibility, a retroplaced intrahepatic gallbladder would be rare. As already described, the anatomic feature can be mistaken for an intrahepatic mass lesion, cyst, or extension of cyst or tumor from the right kidney. Adequate demonstration of the abnormally positioned gallbladder at cholangiography, as well as intravenous pyelography can exclude the above mentioned lesions.

CONCLUSION

Recognition of the possibility of occurrence of anomalous position of the gallbladder is essential in the appropriate interpre-

tation of the hepatic scan.

Being cognizant of gallbladder ectopia may prevent an unnecessary extension of the patient's radiographic and clinical work-up.

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REFERENCES

1. BURKE, J. Anomaly in position of gallbladder. *J.A.M.A.*, 1961, *177*, 508–509.
2. CASTER, M. G., and FLANNERY, M. G. Congenital abnormalities of gallbladder: one hundred and one cases. *Internat. Abstr. Surg.*, 1956, *103*, 439–457.
3. GROSS, R. Congenital anomalies of gallbladder. *Arch. Surg.*, 1936, *32*, 131–162.
4. PATTEN, B. M. Human Embryology. McGraw-Hill Book Company, Inc., New York, 1968, pp. 389–391.

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MACRO-REGENERATING NODULE IN THE CIRRHOTIC LIVER*

RADIOLOGIC FEATURES AND DIFFERENTIAL DIAGNOSIS

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HEPATOMA is a well recognized complication of cirrhosis and is usually the first diagnosis considered when a patient with this disease presents with an enlarged nodular liver. However, the cirrhotic liver also demonstrates a remarkable capacity for regeneration. This feature is a vital part of the pathologic changes that accompany cirrhosis. Most often the regenerating nodules produced are small and uniform, but occasionally form macro-nodules that are easily mistaken for hepatoma (Fig. 1). Statistically, however, giant regenerating nodules occur more frequently than hepatoma.[6] In fact, during the past 2 years we have encountered 7 cases of macro-nodular regeneration in cirrhotic livers and 1 with carcinoma. Moreover, our observations have led us to believe that the radiographic diagnosis is quite specific and that the regenerating nodules can be differentiated from other mass lesions affecting the liver. Although arteriography and hepatic scintigraphy have been used extensively in the diagnosis of liver masses, little attention regarding the differential features of nodular regeneration has appeared in the radiologic literature.[2,5,7,10,11]

Therefore, this paper will: (1) describe our findings observed in these 7 patients during hepatic arteriography and the hepatic scintiscan; (2) correlate these observations; and (3) differentiate this lesion from others with which it may be confused. By this it is hoped that recognition of the entity will be easily accomplished and unnecessary surgery prevented.



FIG. 1. *Macro-nodular regeneration in cirrhosis.* Gross specimen. Note large irregular masses in an enlarged liver. On the left there is a prominent nodule (arrows) in a somewhat atrophic lobe.

CASE MATERIAL

Our 7 patients consisted of 2 males and 5 females ranging from 35 to 67 years of age. Six patients presented with a history of moderate to severe alcoholism accompanied by weight loss and anorexia. An enlarged nodular liver was encountered in 5 of these patients. In the seventh patient an enlarged nodular liver was discovered during a general examination for a Stage III carcinoma of the cervix. After some insistence a history of alcoholism was uncovered. Two of the 6 patients with a past history of alcoholism entered primarily with bleeding esophageal varices. Hepatic scannings were performed on 6 of the patients utilizing radioactive colloidal gold or technetium 99m colloidal sulphide. Hepatic arteriography was done on all patients. The isotopic scans were all initially interpreted as space-occupying lesions suggesting hepatoma or metastasis. In 1 patient, the arteriogram was misinterpreted and in 2 patients the diagnosis of nodular regeneration was not

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accepted. All 3 of these patients underwent surgery for a possible hepatoma. In the remaining patients the diagnosis was confirmed by biopsy or during surgery for a shunt procedure or as in the seventh patient for a Stage III carcinoma of the cervix.

HEPATIC SCINTISCANNING

The major isotopic compounds presently utilized to visualize the liver depend primarily upon their deposition and accumulation within the reticuloendothelial system, notably the Kupffer cells. In this category, colloidal gold, microaggregated human serum albumin labeled with I^{131}, or technetium 99m colloidal sulphide have been used. Technetium 99m was used in the majority of our cases and is probably the compound of choice because of its short half life and high resolution capability.

The scans in our patients demonstrated a mixture of alternating patterns of activity and filling defects (Fig. 2*A;* and 6*A*). Most often the latter occurred in areas of pure cirrhosis and is related to either fibrotic distortion of the liver, blockage of the phagocytic ability of the Kupffer cells, or to poor sinusoidal flow. As a result, multiple areas of variable size and location with decreased to no activity are produced. These are frequently falsely diagnosed as space-occupying masses within an enlarged liver. The amount of Kupffer cells in the regenerating macro-nodules is also somewhat variable. This ranges from little to normal in amount. In general, they are quite prevalent throughout this tissue and, since none of the above factors are present, the scintillation scan over the macro-nodules in the majority of cases is completely normal. In 1 patient, however, poor activity was encountered.

ROENTGENOGRAPHIC FEATURES

Most initial roentgenographic studies prior to arteriography are nondiagnostic and demonstrate only an enlarged liver causing displacement of adjacent viscera. Arteriographic examination of the liver was performed *via* either the celiac axis or the superior mesenteric artery. The features encountered are consistent and diagnostic and in general demonstrate 2 coexistent angiographic patterns. These reflect basically the underlying pathologic processes of cirrhosis in association with the enlarged regenerating mass or masses.

1. *Cirrhosis.* The vascular pattern at any one time depends on the degree of prevailing cirrhosis. In most cases the findings are typical for a relatively advanced stage in which the hepatic parenchyma has already undergone marked reduction in size. Consequently, the vessels are foreshortened, grouped together, and demonstrate considerable tortuosity as well as a corkscrew appearance (Fig. 2*B*; and 3). If moderate involvement is present, variable degrees of vascular alteration as described above will be observed. The ensuing hepatogram is irregular, demonstrating loss of homogeneity characterized by zones of radiolucency.

2. *Regenerating nodule.* The angiographic pattern in the regenerating nodule is often confused with space-occupying lesions. In fact some authors have described them as avascular masses causing displacement of adjacent hepatic artery branches.[5,7] Although few vessels are present within the enlarged nodule, our observations indicate that the major artery supplying the nodule traverses through the relative center of the mass, with little, if any, deviation, displacement or bowing of the vessel *per se* (Fig. 2*B*; 5; 6*B*; 7*A*; 8*A*). The vessel is stretched and elongated and contains relatively few branches. These branches arise symmetrically, but by number are relatively sparse and by size relatively narrow. Consequently, an impression of increased space suggesting the presence of space-occupying masses is created.

The entire vascular appearance in the nodule is probably the end result of a normal hepatic vessel and its branches that have been stretched and elongated commensurate to the growth of the regenerating nodule within which it is centrally incorporated. The contribution of the hepatic artery to the efferent blood supply of the

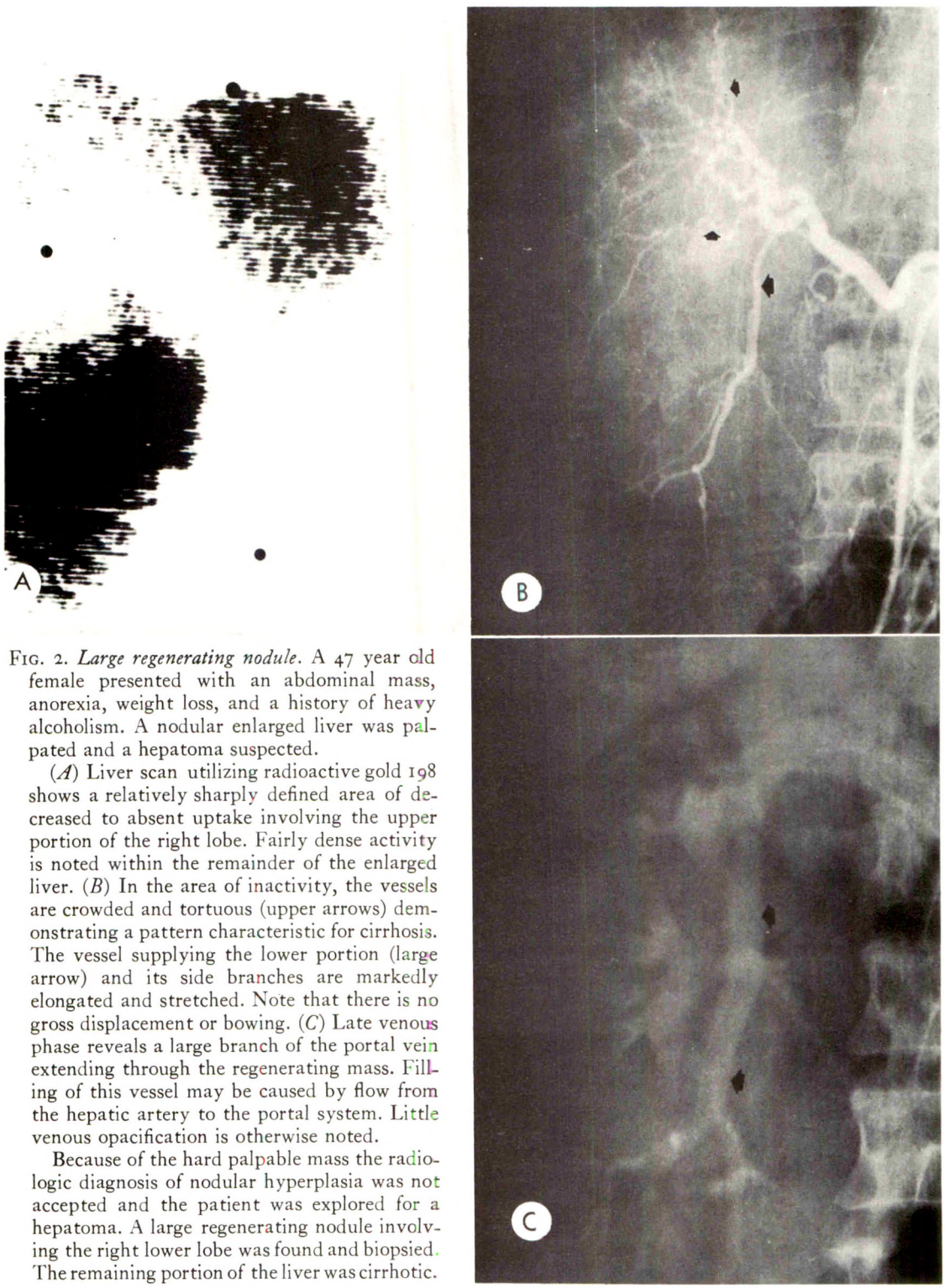


Fig. 2. *Large regenerating nodule.* A 47 year old female presented with an abdominal mass, anorexia, weight loss, and a history of heavy alcoholism. A nodular enlarged liver was palpated and a hepatoma suspected.

(*A*) Liver scan utilizing radioactive gold 198 shows a relatively sharply defined area of decreased to absent uptake involving the upper portion of the right lobe. Fairly dense activity is noted within the remainder of the enlarged liver. (*B*) In the area of inactivity, the vessels are crowded and tortuous (upper arrows) demonstrating a pattern characteristic for cirrhosis. The vessel supplying the lower portion (large arrow) and its side branches are markedly elongated and stretched. Note that there is no gross displacement or bowing. (*C*) Late venous phase reveals a large branch of the portal vein extending through the regenerating mass. Filling of this vessel may be caused by flow from the hepatic artery to the portal system. Little venous opacification is otherwise noted.

Because of the hard palpable mass the radiologic diagnosis of nodular hyperplasia was not accepted and the patient was explored for a hepatoma. A large regenerating nodule involving the right lower lobe was found and biopsied. The remaining portion of the liver was cirrhotic.

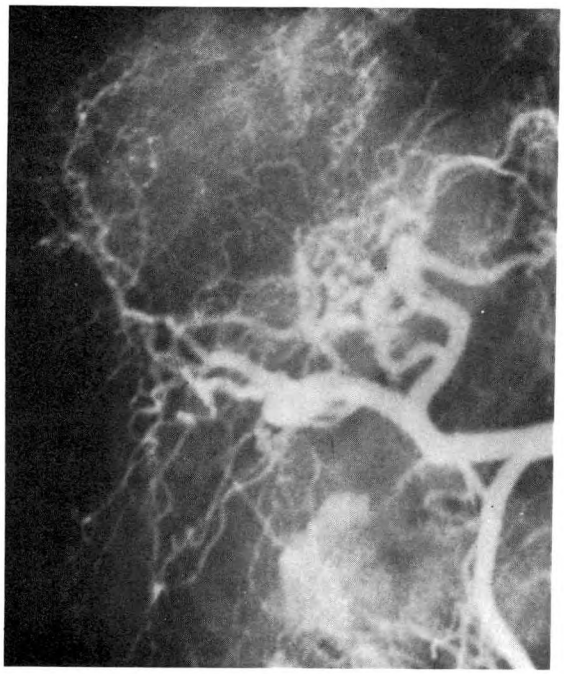


FIG. 3. *Cirrhosis.*
The vessels in this stage of cirrhosis are squeezed, and coiled, producing a corkscrew appearance.

regenerating nodules is greater than to the normal liver parenchyma. Consequently, regenerating nodules growing to massive proportions require an even greater blood flow. Despite the increased demand, minimal if any new branches are developed and the changes involve only those vessels originally present.

The vessels in the smaller nodules appear relatively normal. The angiogram in these lesions more closely resembles normal liver tissue and therefore may be difficult to interpret (Fig. 4). However, the vessel is still located within the center of the mass, and an over-all sparcity of vessels within the mass is encountered. The failure to produce new vessels in any fashion regardless of the size of the regenerating nodule is characteristic for this lesion.

The hepatographic phase for the most part is quite homogeneous and resembles the normal hepatogram (Fig. 7*B*; and 8*B*). This occurs, although the flow of blood through the nodule is probably directed from the hepatic artery to the portal vein and is to some degree increased. In many cases, the venous phase demonstrates a stretched and elongated prominent branch of the portal vein (Fig. 2*C*). As anticipated, no visualization of the hepatic veins was

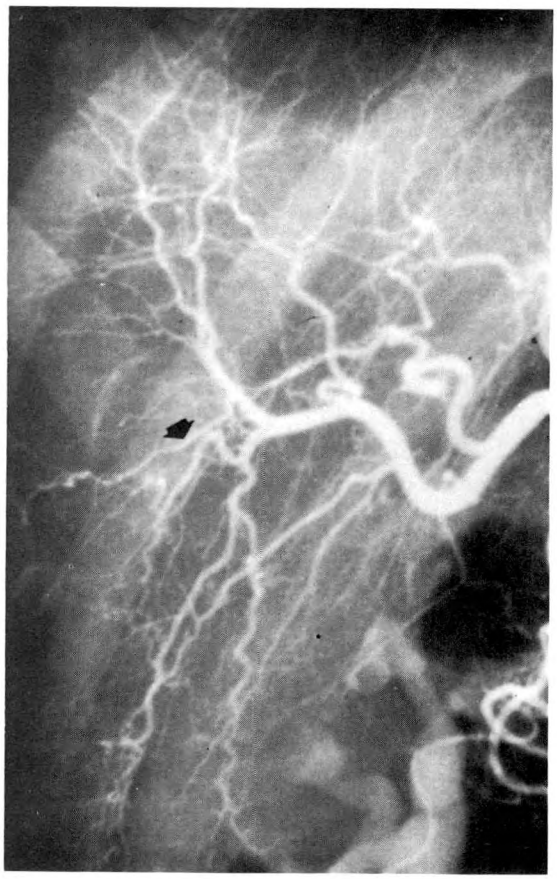


FIG. 4. *Small regenerating nodule.* A 45 year old female with a questionable history of moderate alcoholism and ill-defined symptoms of malaise. Liver function studies were normal and no palpable mass was detected. A liver scan, however, revealed a sharply defined area of inactivity in the right lateral portion of the liver and metastatic disease was suggested.

Arteriogram demonstrates a somewhat elongated hepatic artery vessel with few branches (arrow) supplying the area of inactivity. The vessel is centrally located and there is no evidence of corkscrewing or displacement. The combined studies suggested a small regenerating nodule in a cirrhotic liver.

Unfortunately the above diagnosis was not accepted and the patient underwent surgery. A 3 cm. mass was found and excised. Histologic examination revealed a regenerating nodule.

encountered in these studies. These vessels are frequently occluded early in the disease.

DISCUSSION

The regenerative powers of the liver are remarkable.[4,13] In fact, liver cells continually undergo maturation, aging, dying and replacement. The process nevertheless occurs slowly and is not too easily detected. However, when large masses of liver tissue are destroyed by disease or intoxication the process is greatly accelerated. Mitotic figures become visualized within a few days following the catastrophe. Bile duct epithelium as well as Kupffer cells participate in this mitotic activity.

Cirrhosis as defined by Boyd[3] is a "progressive chronic destruction, diffuse in extent, accompanied by fibrosis, retrogressive changes in the parenchymal cells, and proliferation of the remaining cells in the direction of regeneration." As a result, the final picture of cirrhosis is that of an atrophic firm organ with a nodular surface. In general, the nodules are uniform in size measuring 2–3 mm. in diameter, but occasionally large nodules 2 to 16 cm. in diameter have been encountered.

Microscopically, the changes depend upon the amount and location of connective tissue proliferation, size of regenerative nodules and degree of necrosis. In general, the cells surrounding the central veins, or more specifically those located within the periphery of the acinus, undergo necrosis and are replaced by connective tissue. This tissue now surrounds the surviving cells located around the afferent vessels that basically form the center of the acinus. These cells are rich in enzymes and contain the capacity for cytogenesis and regeneration. If the vascular supply remains active, regeneration and large nodules may be produced. The size of the nodule is usually not dependent on the type of disease, but more often on the stage of involvement. The concept of the liver acinus as the basic structure of the liver more easily explains the reason for the central vein being excluded

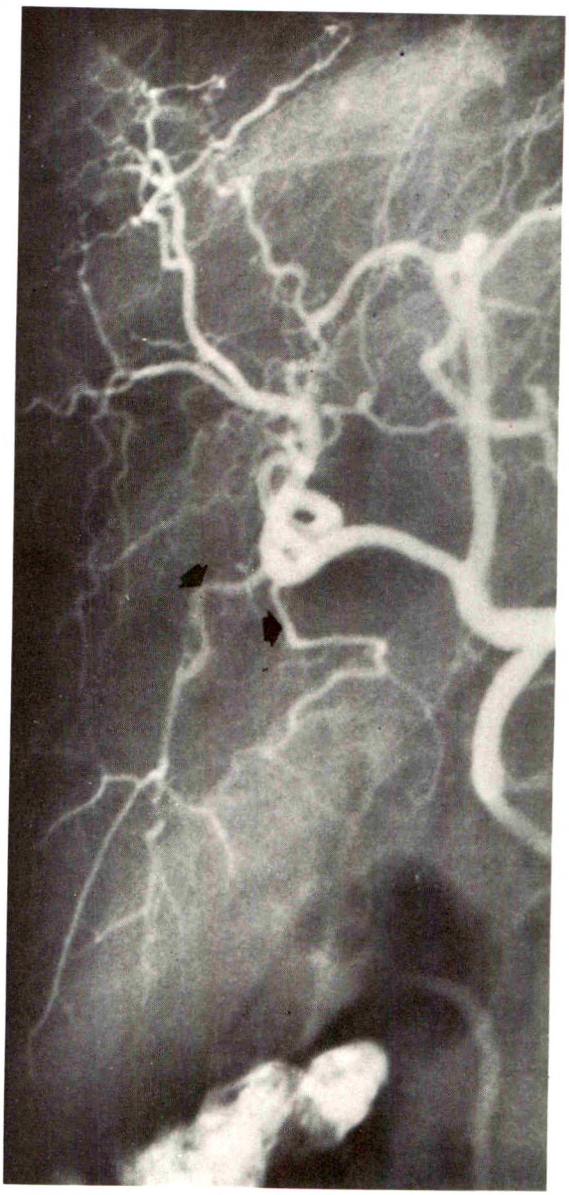


Fig. 5. *Regenerating nodular mass.* A 45 year old veteran with past history of alcoholism entered the hospital because of weakness and malaise. A large liver was palpated. No scanning was performed on this patient.

Arteriogram of the liver reveals a large regenerating mass involving the right lobe as demonstrated by a large single stretched vessel extending downward and through the mass (upper arrow). The vessel is stretched with few branches. The cystic artery (lower arrow) is also well seen. The vessels through the remaining portion of the liver demonstrate the pattern of cirrhosis.

Biopsy confirmed the diagnosis.

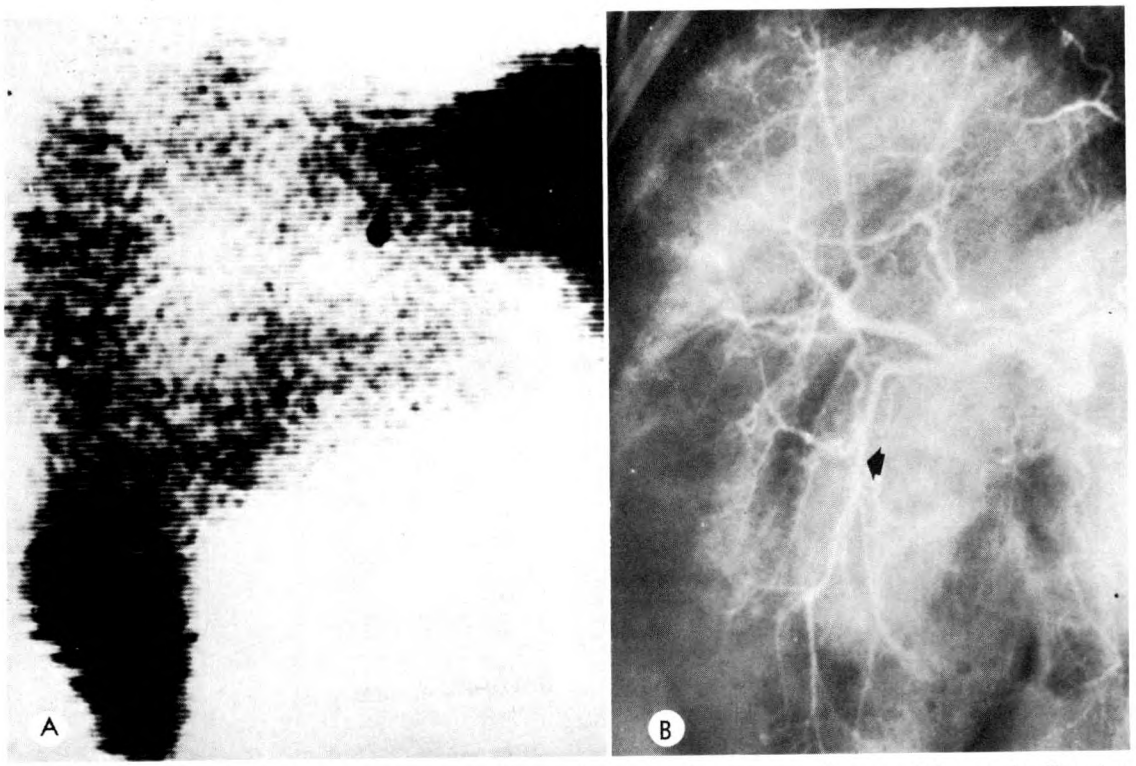


FIG. 6. *Regenerating nodular mass.* A 52 year old female, with a Stage III carcinoma of the cervix. Routine examination revealed a large nodular liver. Despite considerable questioning only a minimal history of alcoholism could be elicited.

(*A*) Liver scan with technetium 99m reveals multiple areas of poor to no activity in the mid-portion of the right lobe in contrast to the surrounding liver in which normal activity is demonstrated. Because of this finding the presence of metastases was considered. (*B*) Arteriogram reveals a diffuse corkscrew pattern within the area of inactivity noted on the isotope scan. Although one vessel is bowed, the pattern is typical for cirrhosis. In the lower portion, a single elongated and stretched vessel is present (arrow). The branches of this vessel are similarly stretched and attenuated.

Cirrhosis with nodular regeneration was diagnosed. The patient underwent surgical exploration because of the carcinoma of the cervix. The liver was noted to be cirrhotic containing a large regenerating mass in the right lobe. The diagnosis was further confirmed by multiple biopsies throughout the liver.

from the developing pseudo-lobule.[13] On this basis the pseudo-lobule may be considered a distorted but true representation of the normal lobulation of the liver. The macro-nodule is therefore an even greater exaggeration. (The reader is referred to the work of Rappaport for a more detailed anatomic explanation of the liver acinus.[13])

According to Florey[4] a perfect balance is maintained between the parenchymal cells and the stroma in uncomplicated regeneration, and lobules are added at the growing nodal points like leaves on a twig. Growth occurs by an increase in size of the individual lobules rather than by an increase in their number. Nevertheless, any injurious agent, according to its mode of action, duration, and concentration may give rise to different end results. For example, in rats and mice by varying the dosage, long continued intoxication with carbon tetrachloride may lead at one time to multiple nodular hyperplasia with cirrhosis, and another time to nodular hyperplasia with hepatoma.[4] In addition, diets deficient in choline have been known to produce hepatomas in some animals.[13] Although difficult to correlate with the human liver, it is conceivable that some form or forms of hepatic injury causing tissue change also occur in

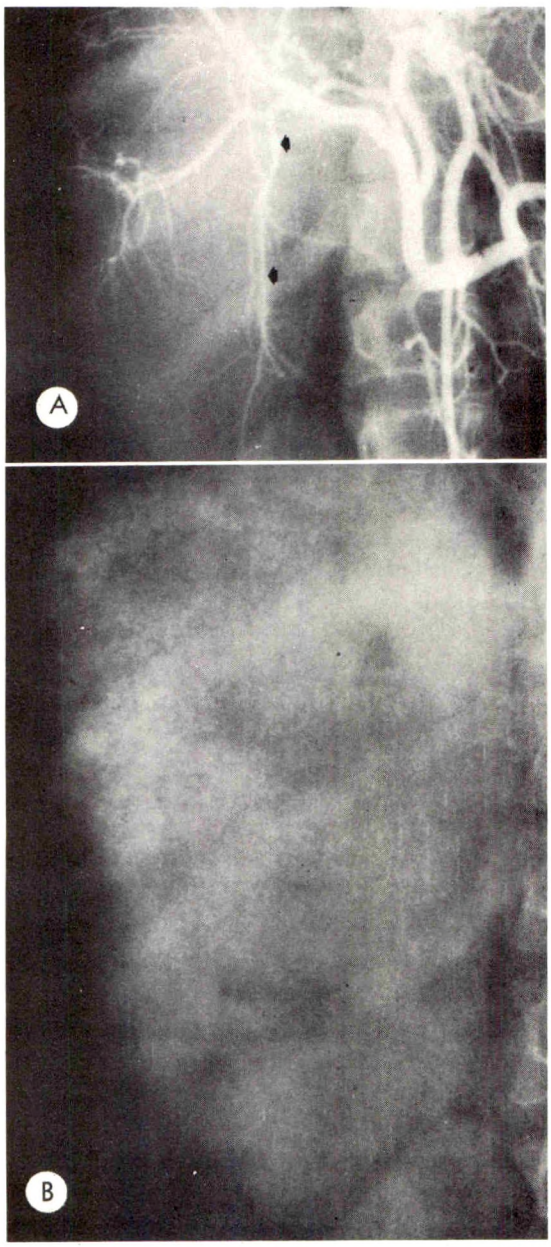


Fig. 7. *Regenerating nodular mass.* A 35 year old female with a past history of heavy alcoholism presented with a history of severe anorexia, ascites and weight loss. On physical examination a palpable nodular liver was noted. The liver scan using radioactive colloid gold demonstrated good activity in the right lobe and little activity in the left lobe.

(*A*) Arteriogram reveals an almost avascular right lobe. The vessels present are elongated and stretched. One vessel is almost devoid of branches (arrow). There is, however, no displacement, bowing or neovascularity. The vessels in the left lobe are crowded and a corkscrew pattern is present.

(*B*) Hepatographic stage reveals a fairly diffuse homogeneous pattern throughout the right lobe of the liver.

Although the arteriographic studies could be confused for space-occupying masses, *e.g.*, multicentric carcinoma or metastasis, the diagnosis of cirrhosis with macro-regeneration was made. This patient died and on autopsy the findings were confirmed.

the cirrhotic liver. This predisposes to the development of a large macro-regenerating nodule at one time and perhaps hepatoma at another time. Although some experimental evidence suggests that regenerating nodules may undergo transition to hepatoma, it is believed that macro-nodules occur in those patients undergoing vigorous treatment with a protein rich diet following removal of their alcohol.[13] Fortunately, the large regenerating nodule occurs more commonly than hepatoma. In one series of 12 cirrhotic patients with large nodular livers, regenerating nodules were found in 9, and hepatoma in the remaining 3.[7]

The diagnosis of regenerating nodule in the cirrhotic liver is easily established when the liver scan and the arteriogram are properly correlated. In general, the liver scans are all abnormal showing variations in size, shape and distribution of activity. In the shrunken cirrhotic areas, activity is markedly reduced and may falsely suggest mass to the interpreter previously prejudiced by this diagnosis. By contrast, the regenerating nodules demonstrate almost always a normal uptake. In 1 of our patients, however, these features were reversed. According to McLoughlin and Gilday,[7] the "only lesion which may appear as an avascular mass on the angiogram and not be represented by an appropriate area of reduced activity in the scan is nodular hyperplasia." This statement is not completely accurate since the regenerating nodule is not truly avascular. It does, however, emphasize the fact that anatomic superimposition of the arteriogram and the nuclear scan is important

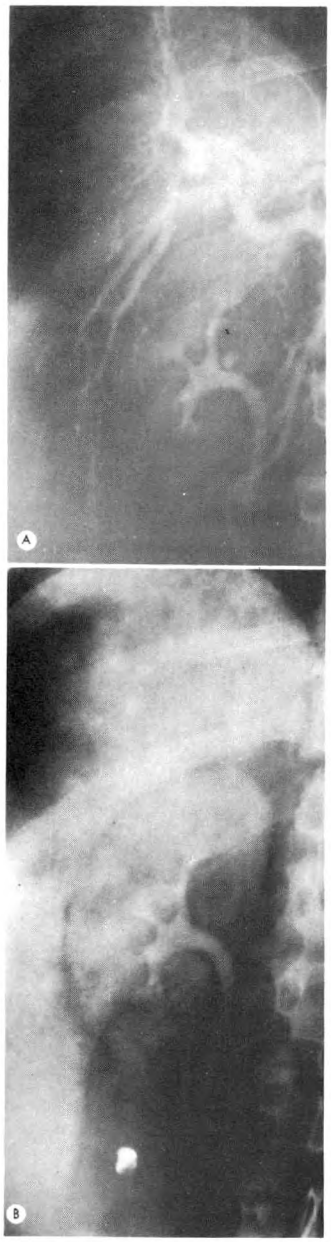


FIG. 8. *Regenerating nodular mass.* A 50 year old chronic alcoholic was admitted with bleeding esophageal varices. A palpable nodular liver was felt on physical examination. Liver scanning performed with technetium 99m colloid revealed decreased activity over the right upper lobe.

(*A*) Arteriogram reveals an advanced pattern of cirrhosis in the upper portion of the liver. The remaining liver is slightly avascular. The vessels present are markedly enlarged, elongated, and stretched. (*B*) The hepatogram reveals blotchy areas of lucencies in the region of cirrhosis, whereas the remaining liver is homogeneously opacified.

The diagnosis of regenerating nodules in a cirrhotic liver was made. This patient had a bleeding tendency and major surgery was not desired. Because of our diagnosis a small excisional biopsy was made to confirm the diagnosis.

in establishing the correct diagnosis. Interpretation on the scan alone is unreliable.

The arteriographic pattern in general represents the combination of both a decrease and increase in liver volume. In the early stages of cirrhosis when fatty degeneration is occurring, the liver also becomes quite enlarged. During this stage, the hepatic artery branches throughout the entire liver are stretched and in this respect resemble the changes encountered in the regenerating nodule (Fig. 9). However, they are diffuse in nature and change their appearance when the liver decreases in size.[11] Since the arteries are not intrinsically involved in the degenerative and scarring process, they retain their original length regardless of the amount of destruction and loss of hepatic tissue. As a result, they eventually coil upon themselves and produce the corkscrew appearance.[11]

The roentgenographic pattern of a cirrhotic liver with regenerating macro-nodules is easily recognized once one is aware of the entity and its associated findings. Nevertheless it still must be differentiated from other lesions causing liver enlargement with or without underlying cirrhosis. In this category, alcoholic hepatitis and hepatocellular carcinoma are 2 primary considerations because of their association with cirrhosis, whereas focal nodular hyperplasia, adenomas, hemangiomas, metastasis, etc., may occur in the normal liver.

Acute alcoholic hepatitis is a term utilized by Sherlock to describe severe hepatic decompensation that occurs following a heavy drinking episode, vomiting, diarrhea, infection or prolonged anorexia in a patient with known alcoholic cirrhosis.[12] Pathologically, the liver undergoes rapid fatty degeneration as well as focal necrosis in which highly refractile dense cytoplasmic material

(alcoholic hyaline of Mallory) is found. The liver becomes acutely enlarged and the angiographic pattern resembles, to a vast degree, the changes seen in nodular hyperplasia. The hepatic arteries are dilated and stretched. However, smudging and pooling of the opaque medium within the necrotic tissue occurs at the level of all the side branches as well as the peripheral ramifications of the main vessels.[12] In addition, the hepatogram is nonhomogeneous and demonstrates a mottled picture consisting of alternating areas of increased density and lucency.

Nodular regeneration must also be distinguished from carcinoma since 75 per cent of all hepatocellular carcinomas arise within the cirrhotic liver. The arteriographic features are, however, quite different (Fig. 10). The pattern characteristic for hepatocellular carcinoma consists basically of arterial displacement, arterial encasement, and—of utmost importance—the formation of a neovascularity that demonstrates bizarre, widened and tortuous vessels.[11] During the capillary phase staining occurs in the vascular tumors. This feature is encountered only in a certain percentage of lesions, whereas the avascular lesions are relatively lucent. Pooling of opaque material in the necrotic neoplastic tissue is also visualized. Although not basically related to vascularity, the nuclear scan in all tumors reveals a sharply demarcated area of decreased or absent activity. The regenerating nodule, by contrast, may show a completely normal uptake.

Metastatic disease, particularly if avas-

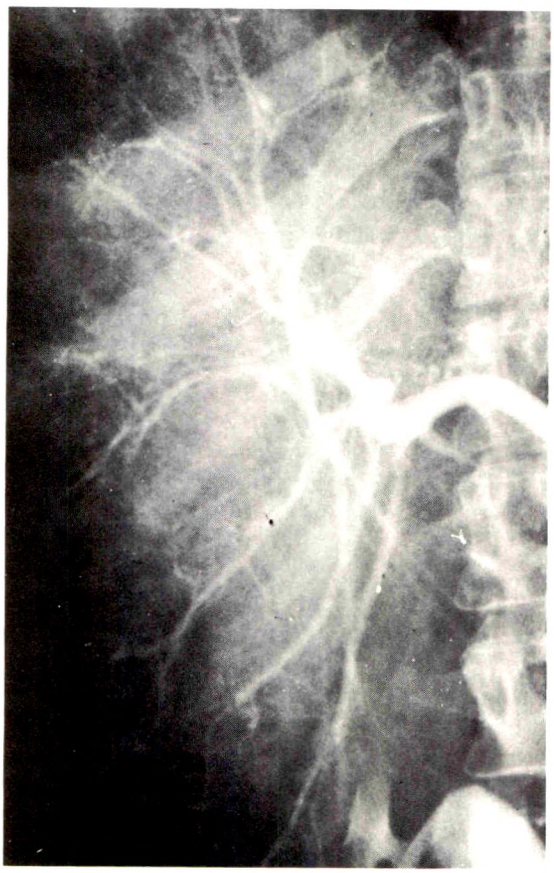


Fig. 9. *Fatty degeneration; early cirrhosis* (biopsy proven).

The liver is diffusely enlarged. The vessels throughout are diffusely and symmetrically elongated. No evidence of coiling or other changes of liver atrophy are seen.

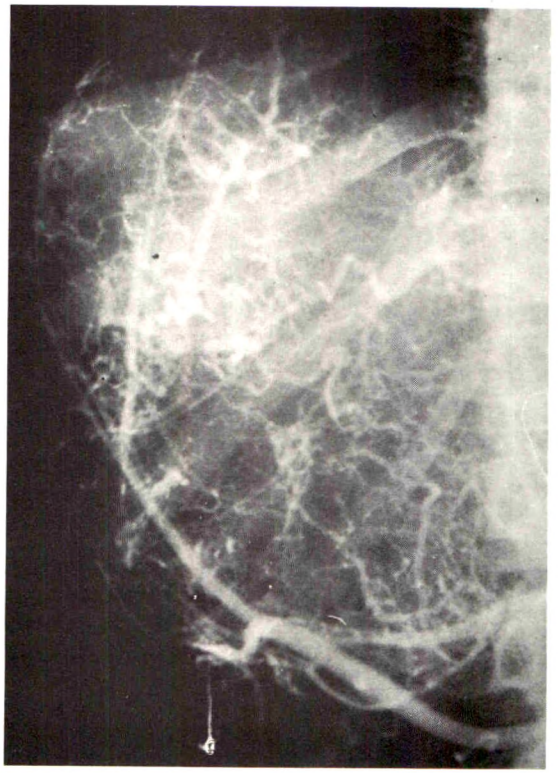


Fig. 10. *Hepatoma.*

Large hepatoma demonstrating diffuse vascular displacement and neovascularity.

cular, may offer some degree of difficulty in the diagnosis. Both the scan and the arteriographic phase will demonstrate negative defects. In the latter, however, the vessels will be obviously displaced and bowed around the masses. The hepatographic phase is irregular and nonhomogeneous in contrast to that observed in the regenerating nodule.

In many ways, focal nodular hyperplasia histologically resembles the regenerating nodule of cirrhosis. Although its origin is not completely understood, it is nevertheless always encountered in a normal liver. The vessels supplying the area of focal nodular hyperplasia are abnormal. They are large, tortuous, and irregular and in distinction to carcinoma and the regenerating nodule, surround the lesion and supply it from the periphery.[6,8] The lesion produces an intense stain during the capillary phase. This is well circumscribed and becomes quite evident during the portal venous phase. The radionuclide scan frequently demonstrates uptake within the mass, since reticuloendothelial cells are present.

Peripheral circumferential vessels have also been described in liver adenomas and probably represent the capsular vessels feeding the tumor.[9] The angiographic features of hemangiomas and other vascular malformations offer no difficulty in the differential diagnosis.[10] All of these fail to show any uptake of the colloidal radioisotope.

SUMMARY

The radiologic features and the differential diagnosis of the large macro-regenerating nodule in the cirrhotic liver are described as visualized in 7 patients, 6 of whom had large nodular livers (Table 1).

Two coexisting patterns predominate: (1) the cirrhotic pattern with its tortuous, compressed and corkscrew appearance; and (2) the pattern associated with the regenerating nodules demonstrating large stretched hepatic artery vessels containing a few side branches.

The hepatic scan most often demonstrates an irregular distribution with decreased to normal activity throughout the liver. The findings depend on the distribution of the underlying diseased state.

Correlation of the scan and the hepatic arteriogram are necessary to avoid falsely diagnosing space-occupying masses in either study.

TABLE I

RADIOLOGIC DIFFERENTIAL DIAGNOSIS OF THE ENLARGED NODULAR LIVER

Lesion	Scan Findings	Angiographic Findings	Hepatogram
Nodular regeneration in cirrhotic liver	Mixed areas of inactivity and activity	1. Crowded vessels, corkscrew appearance, cirrhosis 2. Stretched vessels, no neovascularity, nodule	1. Nonhomogeneous cirrhosis 2. Homogeneous nodule
Hepatoma	Poor to no activity over tumor	Stretched, displaced, neovascularity	Nonhomogeneous
Alcoholic hepatitis	Poor to no activity diffuse	Stretched vessels, smudging and pooling of opaque medium	Nonhomogeneous
Focal nodular regeneration	Normal activity	Neovascularity on periphery of lesion	Homogeneous
Metastasis	Multiple areas of inactivity	Displaced and bowed vessels	Nonhomogeneous

The findings then recognized are relatively specific. Since the cirrhotic liver is capable at one time of forming either large regenerating nodules or hepatoma, it is important to recognize the presence of a regenerating nodule in an enlarged liver, in order to avoid unnecessary surgery.

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REFERENCES

1. Anderson, W. A. D. Pathology. Volume II. Fifth edition. C. V. Mosby Company, St. Louis, 1966, pp. 887–923.
2. Bieler, E. U., Meyer, B. J., and Jansen, C. R. Liver scanning as method for detecting primary liver cancer: report on 100 cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 709–716.
3. Boyd, W. Textbook of Pathology. Seventh edition. Lea & Febiger, Philadelphia, 1964, pp. 796–805.
4. Florey, H. General Pathology. Third edition. W. B. Saunders Company, Philadelphia, 1962, pp. 595–602.
5. Kreel, L., Jones, E. A., and Tavill, A. S. Comparative study of arteriography and scintillation scanning in space-occupying lesions of liver. *Brit. J. Radiol.*, 1968, *41*, 401–411.
6. McLoughlin, M. J., Colapinto, R., Gilday, D. L., Hobbs, B., Korobkin, M., McDonald, P., and Phillips, M. J. Focal nodular hyperplasia of liver. *Radiology*, 1973, *107*, 257–263.
7. McLoughlin, M. J., and Gilday, D. L. Angiography and colloid scanning of benign mass lesion of liver. *Clin. Radiol.*, 1972, *23*, 377–391.
8. McMullen, C. T., and Montgomery, J. L. Arteriographic findings of focal nodular hyperplasia and review of literature. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 380–387.
9. Palubinskas, A. J., Baldwin, J., and McCormack, K. R. Liver-cell adenoma. *Radiology*, 1967, *89*, 444–447.
10. Pollard, J. J., Nebesar, R. A., and Mattoso, L. F. Angiographic diagnosis of benign diseases of liver. *Radiology*, 1966, *86*, 276–283.
11. Rossi, P., and Gould, H. R. Angiography and scanning in liver disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *96*, 553–562.
12. Rourke, J. A., Bosniak, M. A., and Ferris, E. J. Hepatic angiography in "alcoholic hepatitis." *Radiology*, 1968, *91*, 290–291.
13. Schiff, L. Diseases of the Liver. Second edition. J. B. Lippincott Company, Philadelphia, 1963, pp. 1–42.

LOCALIZED DEFORMITY OF THE GALLBLADDER SECONDARY TO HEPATIC MASS LESIONS*

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THE normal gallbladder is a more or less pear-shaped organ usually situated in the right upper quadrant of the abdomen on the undersurface of the right lobe of the liver and closely related to the proximal duodenum and hepatic flexure region of the colon. Variations in configuration are relatively common and the gallbladder may be round, oval, long and narrow in shape or exhibit the well-known "phrygian cap" deformity. Variations in position of an otherwise normal gallbladder may also occur and it may be partly or completely embedded in the liver substance or may be midline or left-sided in position even in the absence of situs inversus.

Apart from these normal variations in position and configuration, displacement and/or deformity of the gallbladder may result from dilatation of adjacent structures such as the duodenum or colon (Fig. 1, *A* and *B*), or may be produced by mass lesions in the right upper quadrant, particularly those arising in the liver or porta hepatis region. Since deformity of the gallbladder may be the first or only clue to the presence of an hepatic mass lesion, the radiologist plays an important role in its detection.

In the present paper some illustrative examples of localized and discrete defects of the gallbladder secondary to a variety of hepatic mass lesions are reported.

REPORT OF CASES

CASE I. A 47 year old white female, R.E., complained of nausea, vomiting and vague epigastric distress. Physical examination was normal. An upper gastrointestinal series showed no abnormality, but oral cholecystography revealed a fairly sharply circumscribed pressure defect on the lateral aspect of the gallbladder. This was present with the patient in the supine (Fig. 2*A*) and upright (Fig. 2, *B* and *C*) positions, and persisted following the administration of a fatty meal (Fig. 2*D*). A liver scan was obtained but this was considered normal (Fig. 3, *A* and *B*). Approximately 3 months later the patient presented complaining of headache, vomiting and vertigo. On this occasion, physical examination revealed signs suggestive of a posterior fossa lesion. This was confirmed by vertebral angiography and subsequent craniotomy revealed an ependymoma of the 4th ventricle. A ventriculo-cisternal shunt was performed. The patient died approximately 8 months following surgery and autopsy examination revealed a 5×5×3 cm. cavernous hemangioma arising from the undersurface of the right lobe of the liver and closely applied to the lateral aspect of the gallbladder which was intrinsically normal.

CASE II. A 42 year old white female, S.B., presented with hematuria and hypertension. Intravenous urography revealed changes typical of polycystic kidneys. Her liver was palpable 3 fingerbreadths below the costal margin. An oral cholecystogram showed multiple discrete defects on the superior aspect of the gallbladder (Fig. 4*A*). These contour defects did not change in appearance with a change in position of the patient. An upper gastrointestinal examination revealed similar discrete contour defects along the anterior aspect of the stomach (Fig. 4*B*). A radionuclide scan showed numerous large defects in the liver (Fig. 4*C*). Subsequent autopsy confirmed the presence of polycystic disease of the kidneys and liver.

CASE III. A 67 year old white male, T.N., complained of abdominal pain and constipation. A barium enema examination revealed a carcinoma of the sigmoid colon. At this time an oral cholecystogram was normal (Fig. 5*A*). A partial colectomy was performed and the patient remained well and symptom free for a period of 2 years when he complained of right upper quadrant pain and weight loss. The liver was

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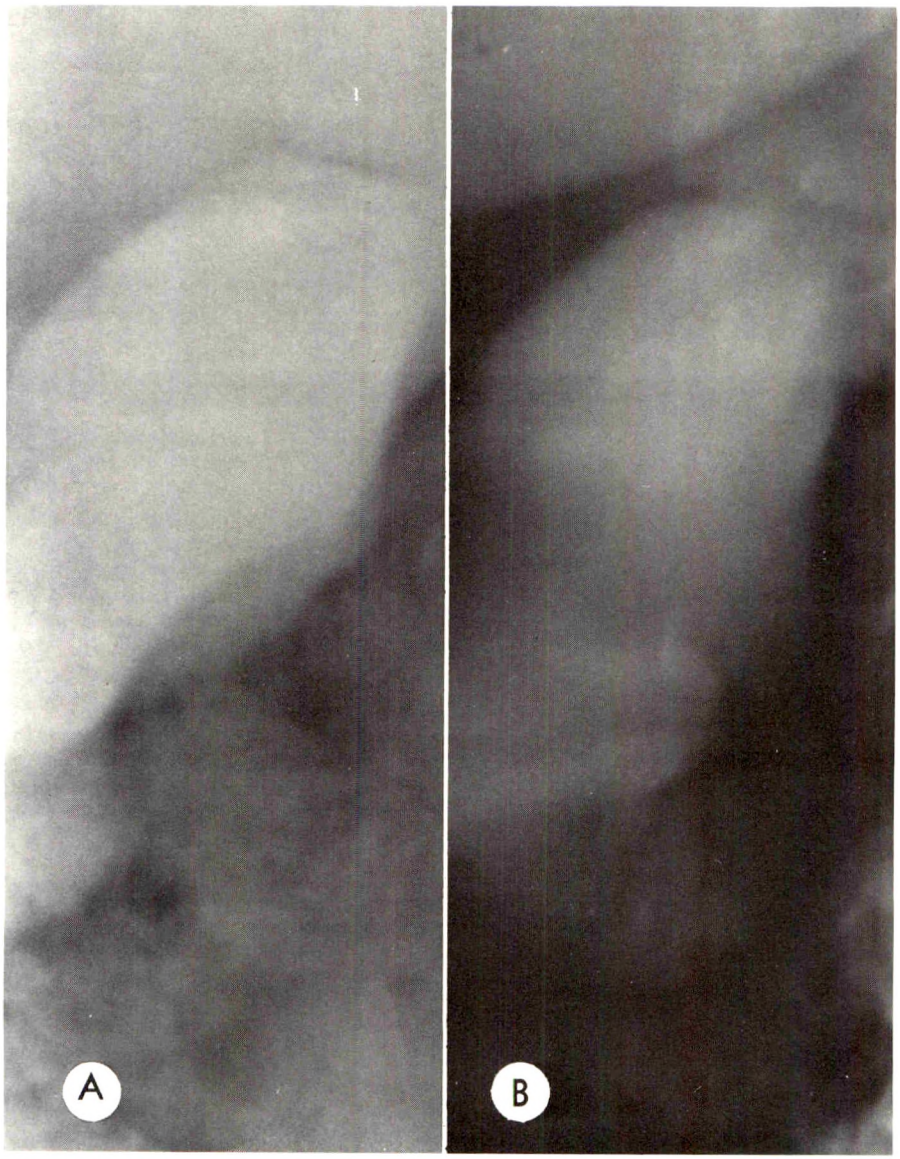


Fig. 1. (*A* and *B*) Localized extrinsic pressure defect on medial aspect of gallbladder produced by fecal-loaded, distended colon. Note variability in appearance with change in position.

just palpable below the costal margin. An oral cholecystogram revealed a contour defect on the superior aspect of the gallbladder (Fig. 5*B*). With the patient in the upright position the defect was again noted (Fig. 5*C*). Surgical exploration revealed a metastatic lesion in the liver closely adherent to the gallbladder.

Case iv. A 53 year old white male, H.L., complained of right upper abdominal pain, anorexia and weight loss. Physical examination revealed a palpable liver but no jaundice. A liver scan showed a large defect in the right lobe of the liver. An oral cholecystogram revealed a localized pressure defect on the lateral aspect of the gallbladder (Fig. 6). This defect was present with the patient in the supine, upright and lateral decubitus positions. Surgical exploration and subsequent autopsy examination revealed a hepatoma in the right lobe of the liver closely adherent to but not invading the gallbladder.

Case v. A 52 year old white female, E.N.,

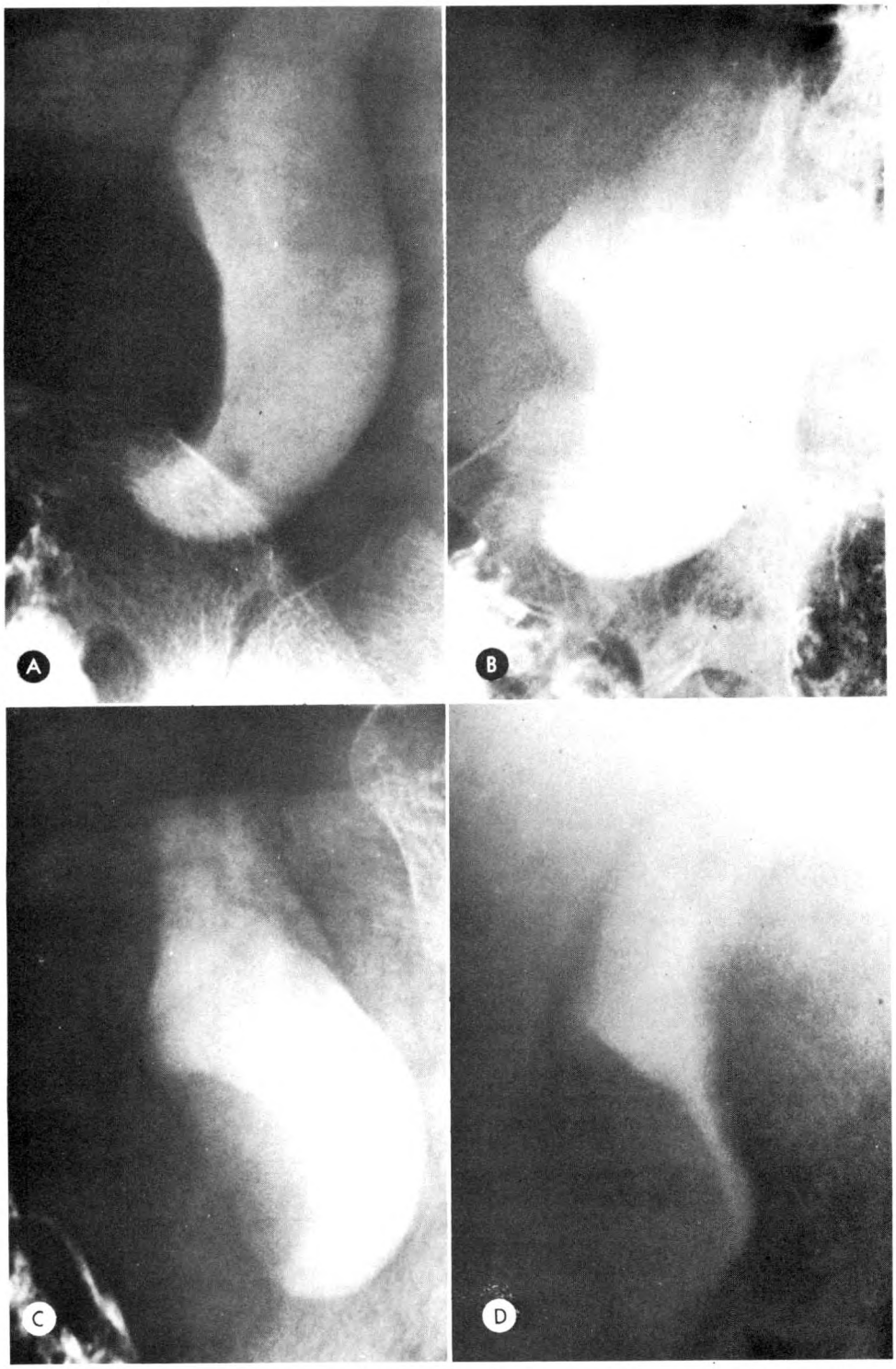


FIG. 2. Case 1. (*A*) Supine roentgenogram showing sharply circumscribed pressure defect on lateral aspect of gallbladder. (*B*) Upright roentgenogram showing persistence of defect. (*C*) Upright roentgenogram in different degree of obliquity. Note "pad effect" suggesting extrinsic nature of defect. (*D*) Note persistence of defect following contraction of gallbladder induced by fatty meal.

complained of vague epigastric distress and bloating of several months' duration. One year previously a hysterectomy had been performed for a large fibroid uterus. At this time a cyst, approximately 6 cm. in diameter, was discovered arising from the inferior aspect of the right lobe of the liver. Needle aspiration of this cyst revealed serous fluid and cytologic examination was unremarkable. No attempt was made to resect the cyst. On her present admission, physical examination was normal as were laboratory data including liver function studies. An upper gastrointestinal series showed an extrinsic pressure deformity on the greater curve aspect of the pyloric antrum (Fig. 7*C*). An oral cholecystogram revealed a concave pressure defect involving the fundus region of the gallbladder (Fig. 7, *A* and *B*). Surgical resection of the hepatic cyst was recommended but the patient refused operation.

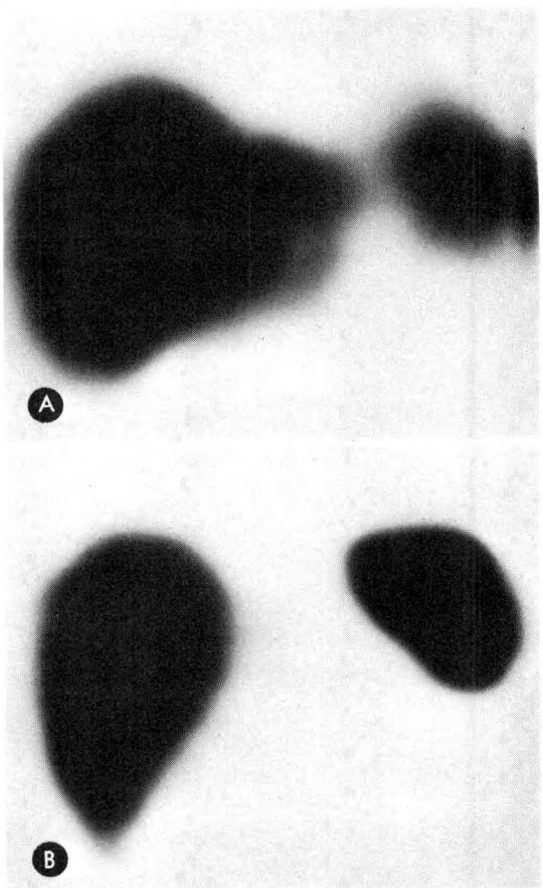


Fig. 3. Case 1. (*A*) Anterior and (*B*) posterior radionuclide scans. Technetium 99m sulphur colloid fails to demonstrate any definite intrahepatic mass lesion.

DISCUSSION

Mass lesions in the liver may produce deformity of the contour of the gallbladder and roentgenologic recognition of such deformity may be the initial clue to the presence of an hepatic lesion (Case 1) or may provide confirmatory evidence in patients with suspected liver disease (Cases II, III, and IV). Isolated examples of deformity of the gallbladder secondary to polycystic disease of the liver,[5,7] hepatoma,[2] hydatid cyst,[8] nonparasitic cyst,[3] metastatic malignancy[4] and hepar lobatum due to tertiary syphilis[4] have been reported.

It may be assumed that any mass lesion adjacent to the gallbladder is capable of displacing it or deforming its contour. In our experience discrete localized defects most commonly result from mass lesions arising in the liver. However, since extrahepatic masses in the right upper quadrant may produce similar defects, careful correlation of the cholecystographic findings with clinical and laboratory data as well as roentgenologic changes in adjacent structures such as the stomach, duodenum, colon and kidney is essential in the evaluation of the nature and significance of the gallbladder deformity.

Deformity of the gallbladder secondary to a gas-distended duodenum or fecal-loaded colon (Fig. 1, *A* and *B*) is generally variable and inconstant in appearance and the cause of the deformity is usually obvious. Defects of the gallbladder contour produced by hepatic mass lesions are constant and reproducible and generally persist despite a change in position of the patient (Fig. 2, *A–C*; and 5, *B* and *C*). Occasionally a change in appearance of the contour defects may occur with change in position.[5] In all of our patients the deformity was demonstrable in roentgenograms obtained both before and after the administration of a fatty meal.

Barium studies of the upper and/or lower gastrointestinal tract may be helpful in confirming the presence of a right upper quadrant mass (Fig. 4, *A* and *B*; and 7*C*) or may demonstrate a primary duodenal or

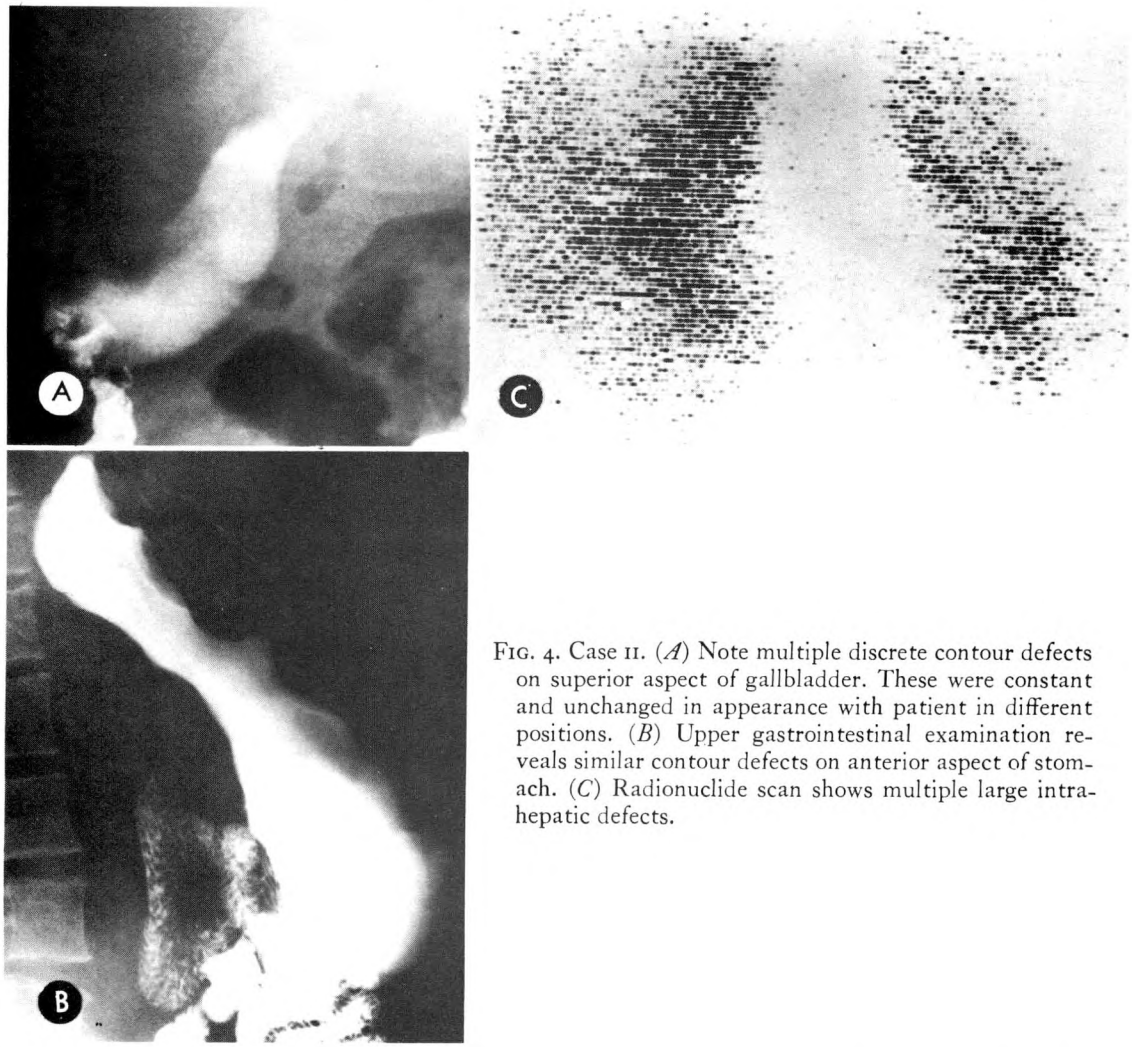


FIG. 4. Case II. (*A*) Note multiple discrete contour defects on superior aspect of gallbladder. These were constant and unchanged in appearance with patient in different positions. (*B*) Upper gastrointestinal examination reveals similar contour defects on anterior aspect of stomach. (*C*) Radionuclide scan shows multiple large intrahepatic defects.

colic lesion. Intravenous urography is essential in suspected cases of polycystic disease of the kidneys and liver. Radionuclide scanning of the liver probably provides the simplest method for the demonstration of a hepatic mass lesion, but as illustrated by Case I a normal scan does not entirely exclude a primary hepatic cause for the gallbladder deformity, especially when such lesions arise from the inferior aspect of the liver surface. In such cases hepatic angiography may provide confirmatory evidence of a mass lesion and may, moreover, aid in differentiating a vascular process such as hepatoma or hemangioma from an avascular lesion such as a cyst, abscess, granuloma or metastatic neoplasm.

Localized pressure defects of the gallbladder secondary to hepatic mass lesions must be differentiated from primary gallbladder disease. In this respect the only relatively common lesion of the gallbladder likely to provide a differential diagnostic problem is the localized form of adenomyomatosis referred to as an "adenomyoma" which almost invariably occurs at or near the fundus of the gallbladder producing a flattening or invagination of the wall of the fundus, frequently accompanied by a contrast-filled dimple or numerous intramural diverticula or sinuses in the region of the fundal deformity. It is only when a hepatic mass lesion produces a sharply circumscribed pressure defect on the fundus of the



Fig. 5. Case III. (*A*) Oral cholecystogram shows normal appearance of gallbladder. (*B*) Supine roentgenogram from oral cholecystography 2 years later reveals defect on superior aspect of gallbladder. (*C*) Upright roentgenogram demonstrates persistence of contour defect.

gallbladder (Fig. 7, *A* and *B*) that any differential diagnostic difficulty is likely to occur. Other primary diseases of the gallbladder such as cholelithiasis, papillomata and cholesterol polyps should provide no problem in differential diagnosis, while pri-



Fig. 6. Case IV. Note localized pressure defect involving lateral aspect of gallbladder contour. This was present with patient in upright and recumbent positions.



FIG. 7. Case v. (*A* and *B*) Note discrete concave pressure defect involving the fundus region of the gallbladder. No contrast-filled dimple or intramural diverticula are demonstrated. (*C*) Upper gastrointestinal examination reveals extrinsic pressure deformity involving the greater curve aspect of the pyloric antrum.

mary adenocarcinoma is almost invariably accompanied by failure of opacification of the gallbladder. Discrete pressure defects of the gallbladder contour secondary to hepatic mass lesions may simulate primary submucosal, intramural lesions of the gallbladder. The latter, however, are extremely rare, although intramural cysts and 1 unusual case of an intramural granuloma have been reported.[6] Primary submucosal intramural neoplasms are similarly very rare, but occasionally metastatic melanoma or carcinoma may involve the gallbladder wall.[1,9]

SUMMARY

Mass lesions in the liver may produce localized deformity of the contour of the gallbladder. Recognition of such defects may be the initial clue to the presence of a hepatic mass lesion or may provide confirmatory evidence in patients with suspected liver disease.

Illustrative examples of localized and discrete defects of the gallbladder secondary to a variety of hepatic mass lesions are presented, including hemangioma, polycystic disease, metastatic carcinoma, primary hepatoma and simple nonparasitic cyst. The roentgenologic differential diagnosis is briefly discussed.

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REFERENCES

1. Botting, A. J., Harrison, E. G., Jr., and Black, B. M. Metastatic hypernephroma masquerading as polypoid tumor of gallbladder, and review of metastatic tumors of gallbladder. *Proc. Staff Meet. Mayo Clin.*, 1963, *38*, 225–232.
2. Brust, R. W., Jr., and Conlon, P. C. Roentgenologic manifestations of primary hepatoma with particular reference to some unusual cholecystographic findings. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1962, *87*, 777–786.
3. Caplan, L. H., and Simon, M. Nonparasitic cyst of liver. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *96*, 421–428.
4. Fisher, M. S. Hepar lobatum and other less exotic causes of gall-bladder deformity. *Radiology*, 1968, *91*, 308–309.
5. Gambill, E. E., and Hodgson, J. R. Polycystic disease of liver with unusual cholecystographic manifestations: report of case. *Gastroenterology*, 1960, *38*, 1003–1004.
6. Ochsner, S. F. Intramural lesions of gallbladder. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 1–9.
7. Sandy, R. E. Cholecystography in presence of polycystic disease of liver: report of case and review of literature. *Radiology*, 1965, *85*, 895–897.
8. Schlanger, P. M., and Schlanger, H. Hydatid disease and its roentgen picture. Am. J. Roentgenol. & Rad. Therapy, 1948, *60*, 331–347.
9. Willis, R. A. The Spread of Tumors in the Human Body. Second edition. Butterworth & Co., Ltd., London, 1952, pp. 218–219.

PITFALLS IN THE ARTERIOGRAPHIC DIFFERENTIATION OF INTRAHEPATIC AND EXTRAHEPATIC MASSES*

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SELECTIVE abdominal arteriography has been established as a useful and reliable method to evaluate masses in the upper abdomen. The value of this method in the assessment of benign and malignant hepatic disease has also been documented extensively.[1,2,4,5,7,9] Arteriographic differentiation, however, between intrahepatic and extrahepatic masses has been based on criteria primarily for intrahepatic lesions, presented in a single report.[8] Almost no emphasis has been given to the lack of specificity of these findings; our experience has provided examples in which these criteria were unreliable.

This report is of 5 patients who had extrahepatic masses, but in whom the arteriographic criteria for intrahepatic masses were present. All 5 patients were believed to have intrahepatic masses clinically, and radioisotopic study of the liver suggested an intrahepatic mass in the 4 patients in whom that study was performed. Our purpose is to present the arteriographic patterns that could lead to the false positive diagnosis of an intrahepatic mass; precise understanding of these patterns should permit more accurate arteriographic interpretation.

REPORT OF CASES

CASE 1. A 42 year old man was hospitalized because of weakness, weight loss, and pain in the back and upper abdomen. Physical examination revealed hepatomegaly. A liver scan using Au^{198} colloid revealed a large area of diminished uptake in the right lobe. Two liver biopsies failed to provide sufficient material for diagnosis, but thoracocentesis yielded serosanguineous fluid containing malignant cells.

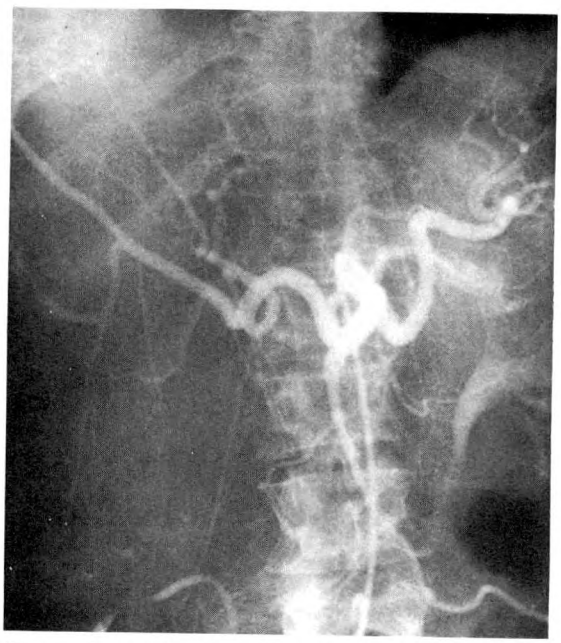


FIG. 1. Case 1. Arterial phase of celiac arteriography shows stretching of the branches of the intrahepatic arteries and displacement of the gastroduodenal artery to the left. No neovascularity is evident.

Celiac and selective hepatic arteriography demonstrated stretched intrahepatic artery branches and displacement of the gastroduodenal artery to the left (Fig. 1). Superior mesenteric and right renal arteriograms showed no abnormal findings.

Laparotomy for open liver biopsy disclosed a normal liver stretched over a huge retroperitoneal carcinoma of the right adrenal gland; no gross or microscopic invasion of the liver was found.

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† Research Fellow in Diagnostic Radiology, supported by Grant GM-01272 from the National Institute of General Medical Sciences, National Institutes of Health, USPHS.

Case II. A 22 year old woman was hospitalized because of abdominal pain and a palpable mass in the right upper quadrant of the abdomen. She had been in an automobile accident 2 years previously, but there was no evidence of an abdominal injury at that time. A Tc^{99m} sulfur colloid scintiphotogram revealed a large space-occupying lesion in the right side of the liver (Fig. 2, *A–D*). The clinical diagnosis was an intrahepatic tumor.

Celiac arteriograms showed displacement of the celiac axis and superior mesenteric artery to the left. In addition, the branches of the right hepatic artery were stretched and attenuated (Fig. 3*A*), and in the parenchymogram phase, a lucency with a dense superior rim was noted in the middle of the right lobe of the liver (Fig. 3*B*). The portal vein phase of the selective splenic arteriogram demonstrated marked stretching of the right portal veins (Fig. 4). The arteriographic diagnosis was hepatic abscess or hepatic cyst.

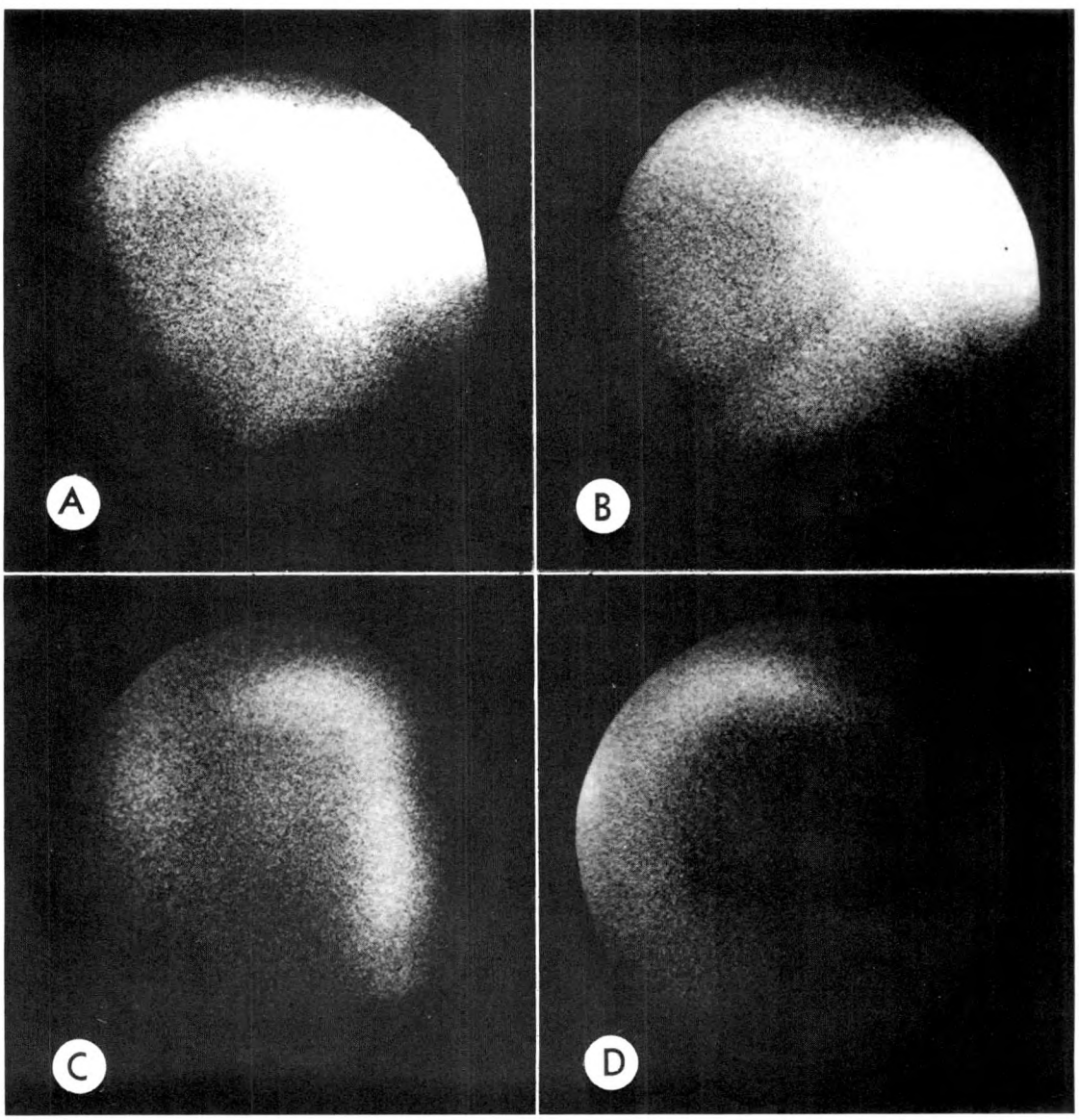


Fig. 2. Case II. Tc^{99m} sulfur colloid scintiphotograms demonstrate a large space-occupying lesion in the right lobe of the liver. (*A*) Right anterior, (*B*) right anterior with right costal marker, (*C*) right lateral, and (*D*) right posterior views.

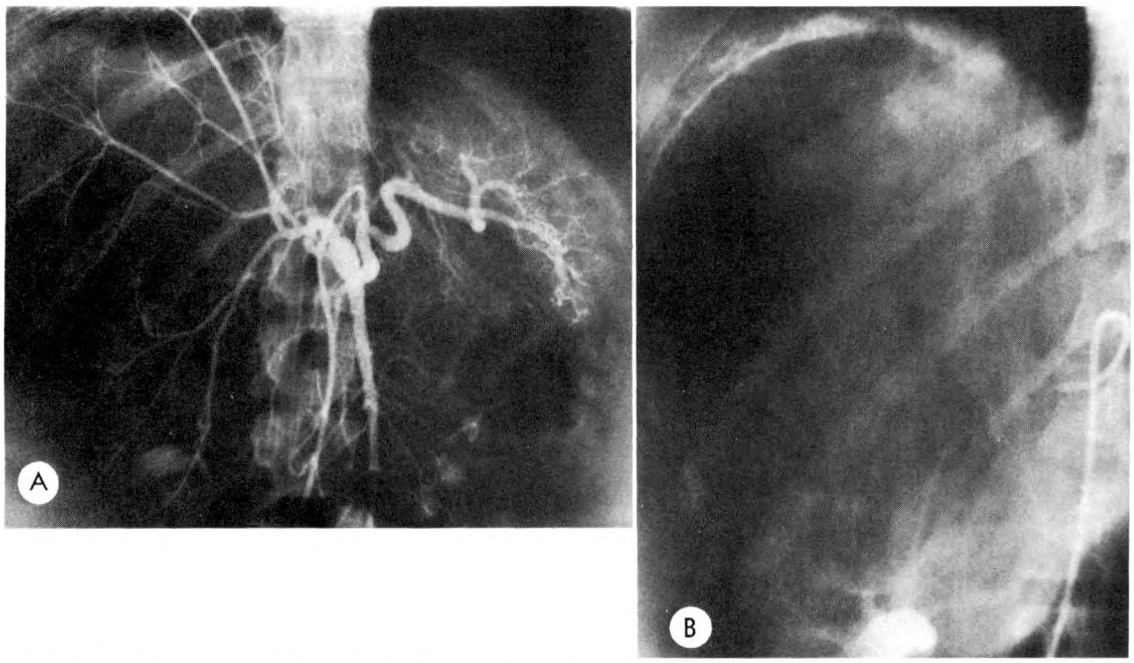
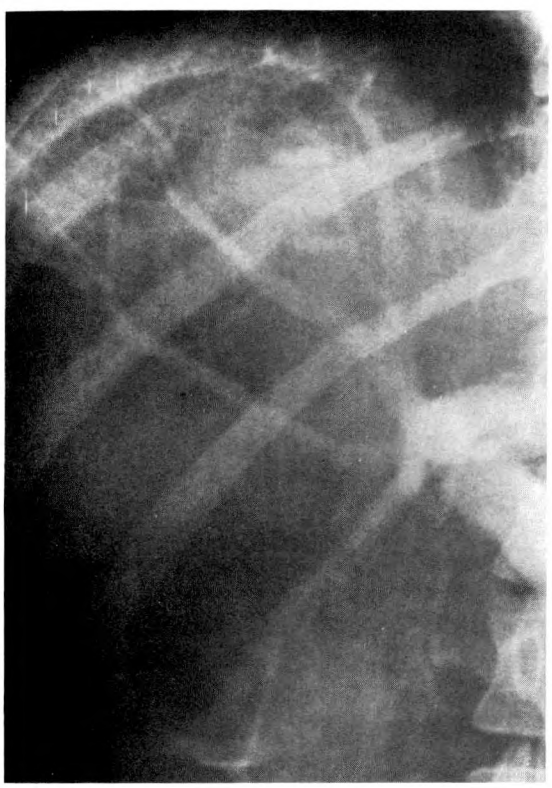


FIG. 3. Case II. (*A*) Arterial phase of celiac arteriography shows displacement of the celiac axis and superior mesenteric artery to the left. The branches of the right hepatic artery are markedly stretched and attenuated. No neovascularity is apparent. (*B*) Parenchymogram phase of hepatic arteriography shows an avascular area with a hypervascular, superior rim in the right lobe of the liver. The right kidney is displaced downward.



Exploratory laparotomy revealed a large cystic mass behind the liver, which was compressing the liver against the anterior abdominal wall. Histologic examination showed a benign, adrenal cyst.

CASE III. A 4 year old girl had a left nephrectomy for Wilms' tumor. At the time of the nephrectomy, the tumor had extended into the inferior vena cava to the level of the diaphragm. The patient had received postoperative irradiation and chemotherapy.

Although clinically the patient had apparently been free of metastatic disease, she was hospitalized 2 years later because follow-up examination revealed increasing hepatomegaly. A Tc^{99m} sulfur colloid scintiphotogram revealed an area of decreased labeling in the middle of the liver. Celiac arteriography was requested in order to evaluate the extent of the possible metastatic disease to the liver.

FIG. 4. Case II. Venous phase of selective splenic arteriography shows marked stretching of the portal veins to the right lobe of the liver.

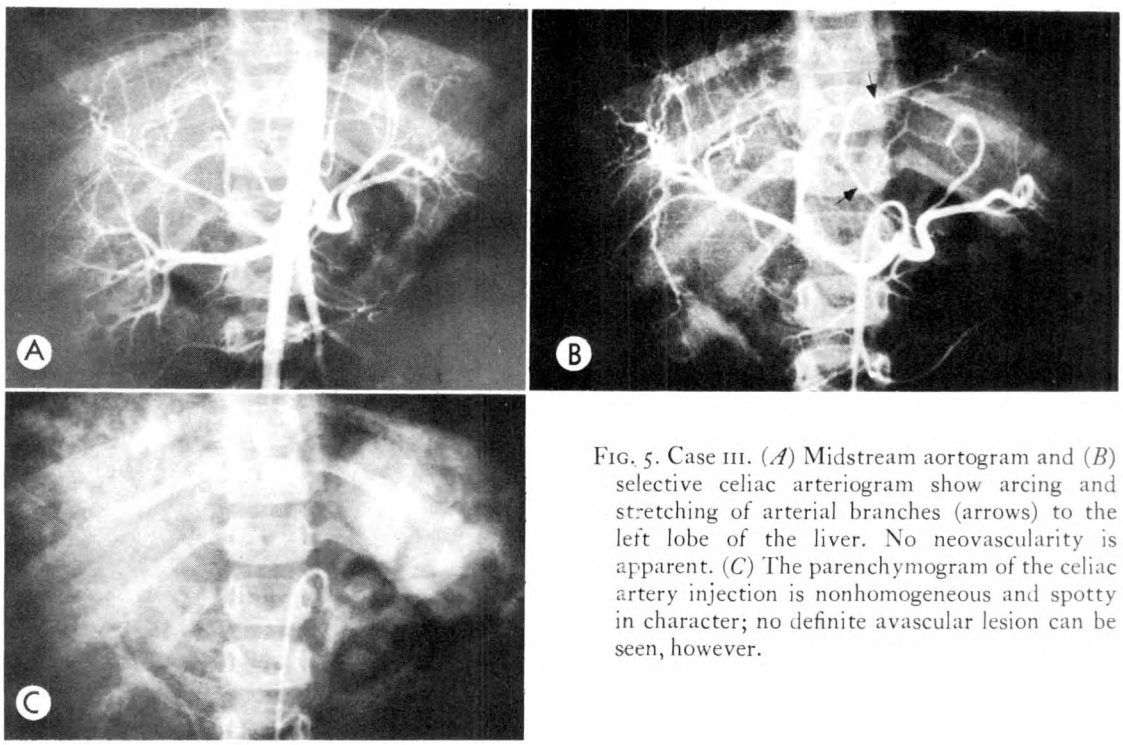


FIG. 5. Case III. (*A*) Midstream aortogram and (*B*) selective celiac arteriogram show arcing and stretching of arterial branches (arrows) to the left lobe of the liver. No neovascularity is apparent. (*C*) The parenchymogram of the celiac artery injection is nonhomogeneous and spotty in character; no definite avascular lesion can be seen, however.

Aortic and selective celiac arteriograms showed arcing and stretching of arterial branches to the left lobe of the liver (Fig. 5, *A* and *B*). No abnormal vessels were noted. The liver appeared nonhomogeneous in the parenchymogram phase (Fig. 5*C*), but no definite avascular lesion was noted in either lobe. The arteriographic impression was diffuse, spotty metastatic disease in the right lobe and a large site of metastasis in the left lobe.

Exploratory laparotomy revealed no metastatic disease to the liver, but a large, metastatic retroperitoneal mass was found posterior to and separable from the left lobe of the liver.

CASE IV. An asymptomatic 60 year old man was hospitalized because a mass in the right upper quadrant of the abdomen was palpated on routine physical examination. A Tc^{99m} sulfur colloid scintiphotogram showed a huge space-occupying lesion in the right lobe of the liver. The clinical diagnosis was an hepatic tumor or, less likely, an indolent hepatic abscess.

Celiac arteriography was performed and revealed marked displacement of the celiac axis to the left by a huge soft tissue mass in the right upper quadrant of the abdomen (Fig. 6). A splenic arteriogram showed a normal spleen, and displacement of the head of the pancreas to the left (Fig. 7*A*). The splenic vein was normal but the portal vein was displaced superiorly (Fig. 7*B*). The portal veins to the right lobe were stretched and attenuated; the left lobe portal veins were relatively normal in appearance. An hepatic arteriogram (Fig. 8, *A* and *B*) showed arterial branches in the right lobe of the liver stretched and attenuated by a large, apparently intrahepatic, mass. No displacement

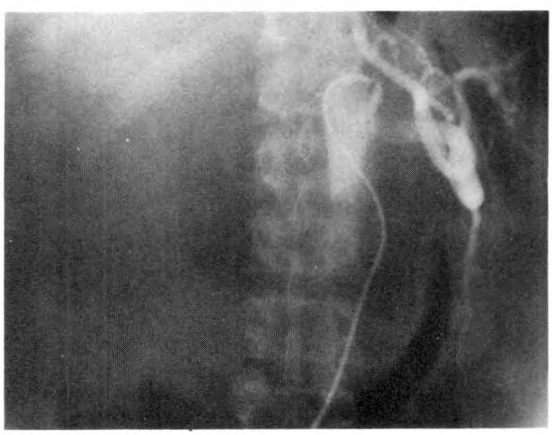


FIG. 6. Case IV. Celiac arteriogram demonstrates marked displacement of the celiac axis to the left by a huge mass of soft tissue density.

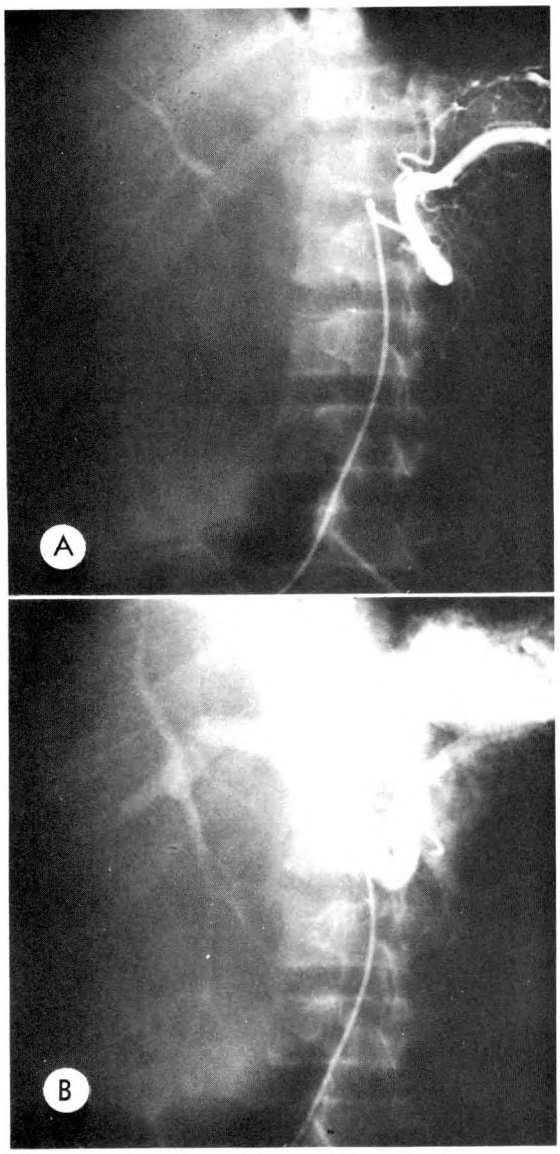


FIG. 7. Case IV. (*A*) Selective splenic arteriogram (with "flash-filling" of hepatic artery branches) shows vessels to the pancreas displaced toward the left. (*B*) Venous phase of splenic arteriogram shows a normal splenic vein and upward displacement of the main portal vein. The portal veins to the right lobe are stretched and attenuated but the left lobe portal veins are more normal in appearance.

of arterial branches outlining a mass was apparent—contrary to what one would expect from such a large intrahepatic lesion.

Consequently, a right renal arteriogram was obtained; it showed marked separation of the arterial branches to the upper pole from the other branches of the renal arteries (Fig. 9*A*). The branches to the upper pole were directed vertically and draped around a mass. Abnormal vessels were noted, and pooling of the contrast medium was also evident at the base of the mass (Fig. 9*B*). The arteriographic diagnosis was a large, cystic, renal carcinoma.

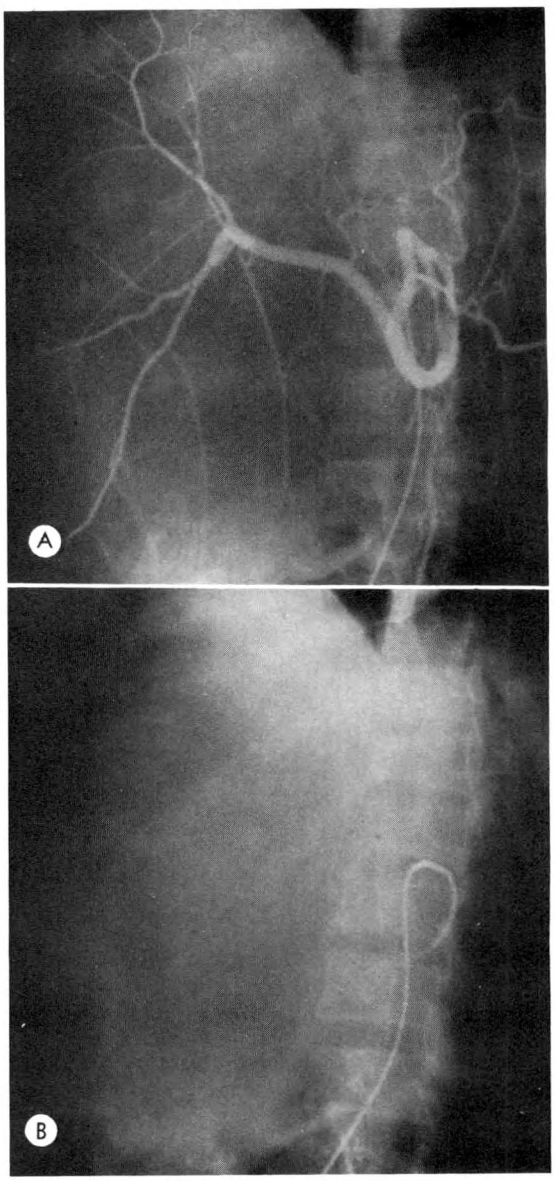


FIG. 8. Case IV. (*A*) Selective hepatic arteriogram shows arterial branches in the right lobe which are stretched and attenuated. No neovascularity is seen. The left lobe appears to be enlarged, but the vessels retain some of their tortuosity. (*B*) A lucency is seen within the right lobe during the parenchymogram phase.

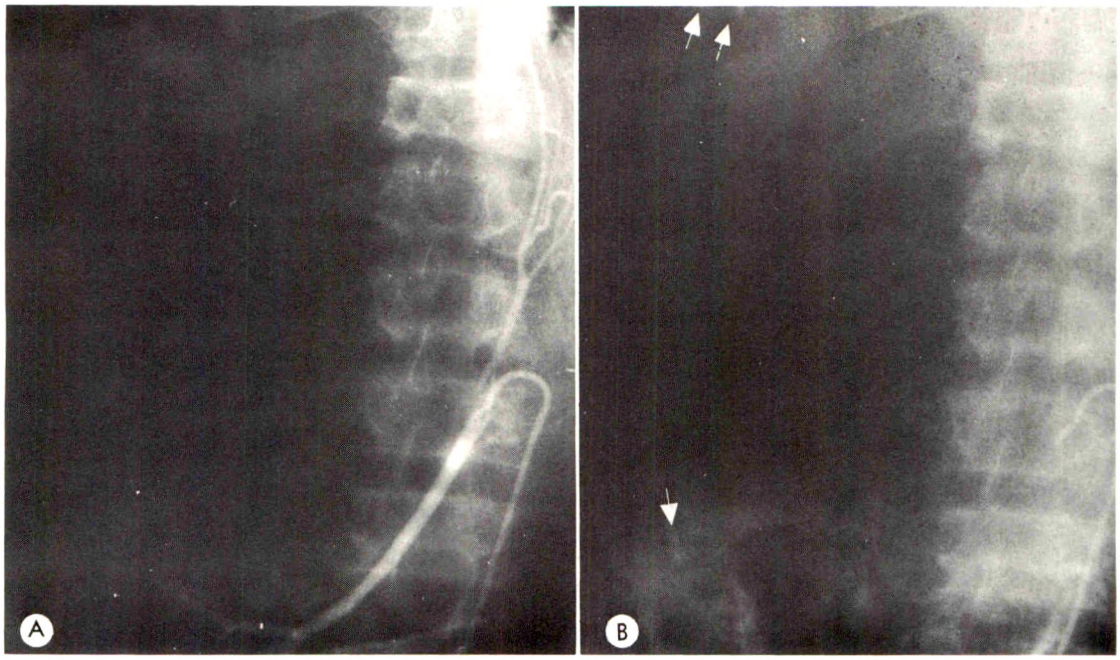


Fig. 9. Case iv. (*A*) Selective right renal arteriogram shows marked separation of the arterial branches to the upper pole from the other branches of the renal artery. (*B*) Late roentgenogram from selective right renal arteriography shows pooling of contrast medium (arrows) at the base of the mass and in the right upper quadrant of the abdomen.

Exploratory laparotomy uncovered a large cystic mass behind a stretched and thinned right lobe of liver. The mass had not invaded the liver and was easily separated from it. The mass was a cystic hypernephroma.

Case v. An 18 year old girl was hospitalized because of sudden onset of epigastric pain. She had no history of alcoholism or drug abuse. She had tenderness in the upper abdomen and laboratory tests suggested pancreatitis.

Examination of the upper gastrointestinal tract showed normal findings. Celiac arteriograms, however, showed stretching of vessels at the base of the right lobe of the liver and stretching of branches of the left hepatic artery (Fig. 10*A*). These findings combined with the somewhat nonhomogeneous appearance of the liver in the parenchymogram phase (Fig. 10*B*) suggested intrahepatic inflammation.

The patient's pain subsided and she was discharged. Six months later, however, the pain recurred. As before, laboratory tests suggested pancreatitis. Repeat arteriograms showed more normal appearance of the arteries to the base of the right lobe of the liver, but persistent stretching of the branches of the left hepatic artery (Fig. 11).

Late studies from injection of the hepatic artery showed dense opacification of the left hepatic vein; no right hepatic veins, however, were seen (Fig. 12). The splenic artery had changed in position from the prior study, and it was now arced inferiorly (Fig. 13*A*). The splenic vein was also arced inferiorly on this second study (Fig. 13*B*). The arteriographic diagnosis was a pseudocyst in the tail of the pancreas, located behind and inferior to the left lobe of the liver.

Exploratory laparotomy disclosed a large pancreatic pseudocyst arising from the superior border of the tail of the pancreas and situated behind the left lobe of the liver.

Discussion

Abnormal vessel(s) arising from the hepatic artery, distortion of hepatic artery branches, radiolucency within the liver in the parenchymogram phase, and extension of hepatic artery branches to the periphery of the liver are the arteriographic findings

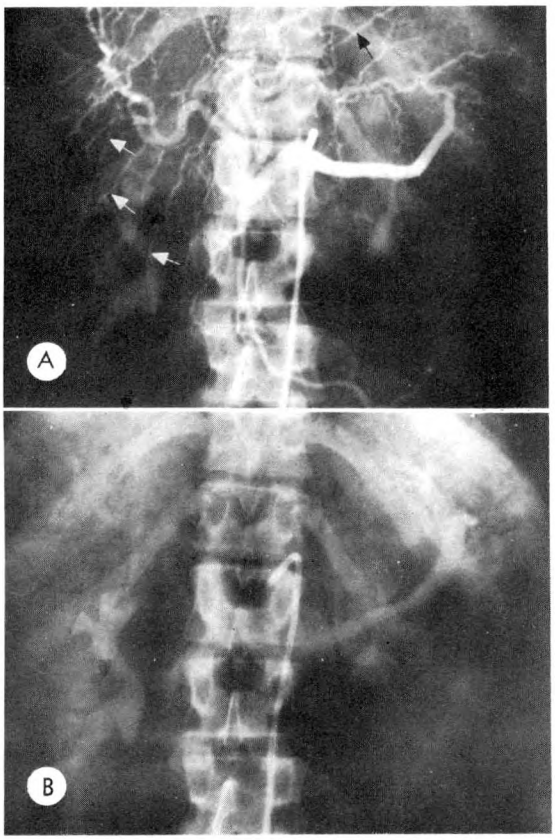


FIG. 10. Case v. (*A*) Celiac arteriogram shows minimal stretching of vessels at the base of the right lobe of the liver (white arrows) and stretching of some branches of the left hepatic artery (black arrow). (*B*) The venous phase of celiac arteriography shows a nonhomogeneous appearance of the liver parenchyma. Note position of splenic vein.

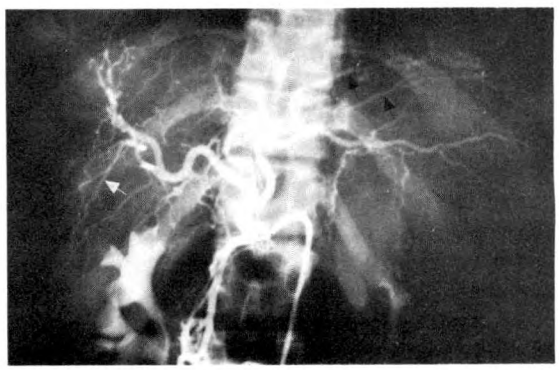


FIG. 11. Case v. Subsequent celiac arteriogram (6 months later) shows less stretching of branches to the right lobe of the liver (white arrow), but persistent stretching of the branches of the left hepatic artery (black arrows).

that Rossi and Ruzicka[8] believe should be considered in making the distinction between intrahepatic and extrahepatic masses. These arteriographic findings, however, were not sufficient to make this distinction in our group of cases with avascular or poorly vascularized masses directly superimposed upon the liver in the anteroposterior projection. Because each lesion was directly adjacent to the liver, indentation and compression of the parenchyma would have been expected.[3,6] Moreover, because the liver is quite pliable, its shape is easily altered by pressure from contiguous structures. It follows that the distribution of intrahepatic vasculature can also be altered. Despite the extrahepatic location of each lesion presented here, intrahepatic arterial branches were stretched and attenuated. In addition, because each lesion was relatively avascular, identification of a "feeding" vessel was not a helpful point to consider. The marked indentations made upon the liver by the masses of renal and adrenal origin, each created a large radiolucent area within the hepatic parenchymogram and, despite their size, the masses still allowed hepatic artery branches to extend beyond the mass to the periphery of the liver. Thus, it is apparent that despite the extrahepatic location of each of the lesions described in this report, some of the criteria

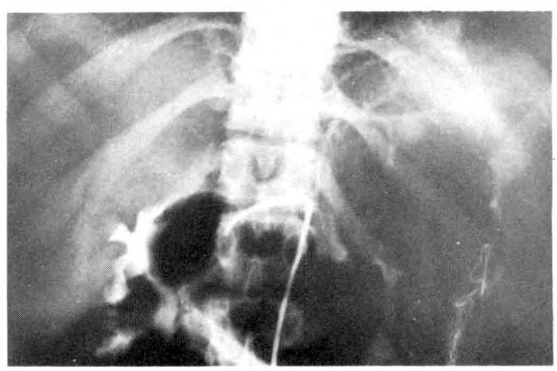


FIG. 12. Case v. Late film study from injection of the hepatic artery shows dense opacification of the left hepatic vein; no other hepatic veins are seen. (Reproduced with permission of the authors and *Radiology*.[4])

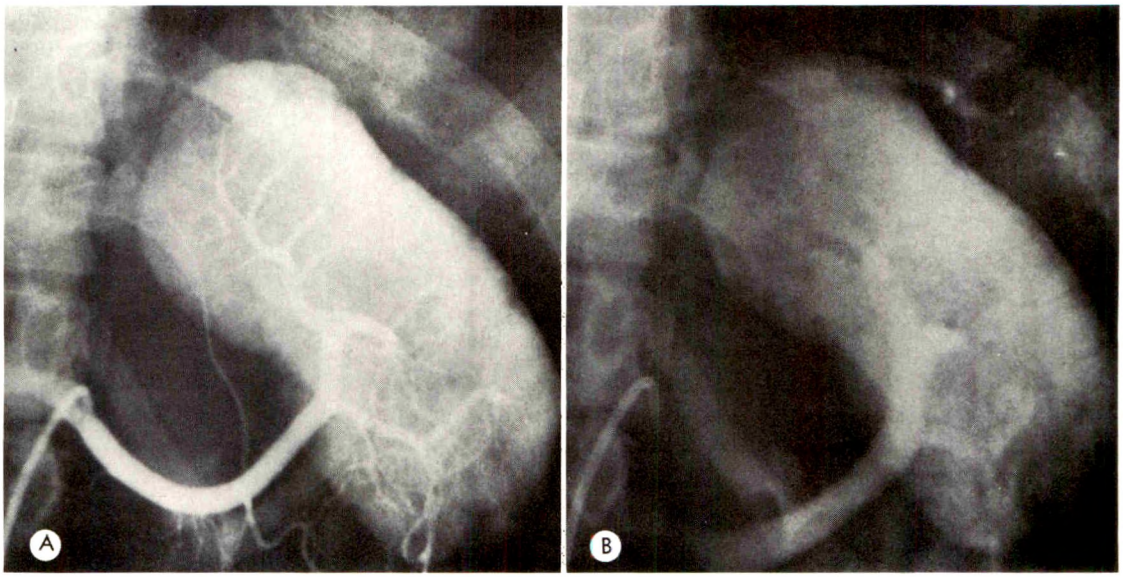


Fig. 13. Case v. (*A*) Selective splenic arteriogram shows arcing of the splenic artery downwards and stretching of an arterial branch arising from the superior portion of the splenic artery. (*B*) Venous phase shows similar downward displacement of the splenic vein. Both splenic artery and vein are changed from the previous study.

for intrahepatic location were present in each case.

Previous reports have emphasized the value of using more than one projection in performing celiac arteriography.[2,5,7,8] It is all too apparent from our cases that an anteroposterior and a series of either steep oblique or lateral roentgenograms would be extremely helpful in defining the location of a mass distorting the right lobe of the liver. The differentiation dilemma still exists, however, if the oblique or lateral view is not sufficiently tangential to the site of the compressed hepatic parenchyma to demonstrate the relationship of the liver margin to the mass. If the mass is superimposed upon the left lobe of the liver in the anteroposterior projection, neither craniocaudad oblique nor lateral projections can demonstrate its contour.

Accurate determination of the extrahepatic location of the mass was made by angiography in 2 of the 5 cases. The diagnosis was made by recognizing that the mass was probably extrahepatic on the hepatic arteriogram and then by injecting the artery supplying the organ of origin of the mass. Abnormalities relating to the mass in arteries of the organ of origin indicated the correct diagnosis. In all 5 cases, these abnormalities were considered by the members of the involved clinical services to have been important in determining the appropriate surgical approach to the mass. The distinction between intrahepatic and extrahepatic location of the mass may be impossible by any other diagnostic test, including liver scintiphotography. In Cases I, II, III, and IV, liver scans were interpreted as showing an intrahepatic mass. Liver scanning was not performed in Case V.

We believe that the distinction between intrahepatic and extrahepatic masses can be made by careful analysis of the hepatic vascular patterns on the arteriogram. A vascular intrahepatic mass presents no diagnostic problem because feeding arteries of hepatic origin can be identified. Distinction between an intrahepatic mass and a extrahepatic mass becomes difficult only when no neovascularity or feeding arteries are demonstrated. For example, intrahepatic cysts and many hepatic abscesses produce stretching, attenuation, and displace-

ment of hepatic artery branches without causing encasement of arteries or neovascularity. Hepatic cysts and abscesses, however, are spherical and contain no vasculature within the bulk of their mass. Instead, hepatic arteries and portal radicles are displaced about the circumference of the mass. Angiographically, arteries are arced superiorly and inferiorly about the mass. If the cyst or abscess is located deep within liver substance, hepatic arteries are also displaced anteriorly or posteriorly. These arteries may appear straightened and, in the anteroposterior projection, to course through the mass; nevertheless the superiorly and inferiorly arced arteries should permit identification of the location and nature of the mass. If the location still cannot be determined with confidence, a steep oblique or a projection with craniocaudad angulation of the roentgen-ray beam can show that the apparently straightened arteries are, in fact, arced anteriorly and posteriorly.

In the cases presented here, only Case II had hepatic arteries that were arced about the circumference of the mass in the anteroposterior projection. Even in this case, the arced arteries were only seen superior to the mass; inferiorly the hepatic arteries were straightened, not arced. The absence of arcing of hepatic arteries inferior to the mass should suggest that the inferior margin of the mass is outside the liver capsule and that the mass arose outside the liver.

An infiltrative intrahepatic mass that replaces rather than primarily displaces liver tissue could cause straightening of hepatic arteries without superior and inferior arcing of arteries about the mass. Most infiltrative hepatic masses, however, cause spasm or encasement of the hepatic arteries that would verify their intrahepatic location.

The angiographic finding of straightened, attenuated intrahepatic arteries without both superior and inferior arcing of arteries and without spasm or irregularity of arteries should suggest that the mass is extrahepatic. Oblique projections can then be obtained in an attempt to visualize the posterior margin of the liver parenchyma. Suspicion of the possible extrahepatic location of the mass should promote further investigation by intercostal, adrenal, pancreatic, or right renal arteriography.

SUMMARY

When a mass in the upper abdomen is suspected on clinical or routine roentgenographic examination, or a mass of the liver is detected by means of radioisotope study, selective abdominal arteriography has been suggested as the diagnostic method most helpful in differentiating whether the mass is intrahepatic or extrahepatic.

Our experience has shown that the arteriographic criteria suggested in the past are nonspecific and that, with careful analysis of intrahepatic arteries as well as additional oblique or lateral projections (whichever is more tangential to the site of compression), the false positive diagnosis of intrahepatic mass can often be avoided. Arteriography of adjacent organs can then be carried out to try to identify the origin and nature of the mass. The lack of superior and inferior arcing of intrahepatic arteries may serve as a sign of the extrahepatic location of a mass superimposed on the liver in the anteroposterior projection.

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REFERENCES

1. ALFIDI, R. J., RASTOGI, H., BUONOCORE, E., and BROWN, C. H. Hepatic arteriography. *Radiology*, 1968, *90*, 1136–1142.
2. FREEMAN, L. M., BERNSTEIN, R. G., KATZ, M. C., DERMAN, A., and MENG, C-H. Combined diagnostic approach of hepatic scanning and celiac angiography in investigation of liver disease. *J. Nuclear Med.*, 1969, *10*, 628–632.
3. FREEMAN, L. M., MENG, C-H., JOHNSON, P. M., BERNSTEIN, R. G., and BOSNIAK, M. A. False positive liver scans caused by disease processes in adjacent organs and structures. *Brit. J. Radiol.*, 1969, *42*, 651–656.
4. GLICKMAN, M. G., and HANDEL, S. F. Opacification of hepatic veins during celiac and

hepatic angiography. *Radiology*, 1972, *103*, 565–571.
5. Nebesar, R. A., Pollard, J. J., and Stone, D. L. Angiographic diagnosis of malignant disease of liver. *Radiology*, 1966, *86*, 284–291.
6. O'Donnell, T. A. Liver scanning for extrahepatic tumors. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 1063–1067.
7. Pollard, J. J., Nebesar, R. A., and Mattoso, L. F. Angiographic diagnosis of benign diseases of liver. *Radiology*, 1966, *86*, 276–283.
8. Rossi, P., and Ruzicka, F. F. Differentiation of intrahepatic and extrahepatic masses by arteriography. *Radiology*, 1969, *93*, 771–780.
9. Rossi, P., and Gould, H. R. Angiography and scanning in liver disease. *Radiology*, 1970, *96*, 553–562.

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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EDITORIAL

SEVENTY-FIFTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Seventy-fifth annual meeting of the American Roentgen Ray Society will be held at the San Francisco Hilton Hotel in San Francisco, California, Tuesday through Friday, September 24 through September 27, 1974. The Society last met in San Francisco in 1956. I think most everyone would agree that this beautiful city is a splendid place, not only for a meeting, but for a visit particularly at this time of year. The facilities are improved and expanded and there will be ample opportunity to become reacquainted with this interesting and elegant city on our West Coast.

Dr. and Mrs. Tom Fullenlove have generously accepted to serve as Chairpersons of the over-all Committee on Local Arrangements and Ladies' Committee, respectively. They are working with a fine Local Committee, and information concerning golf arrangements for the competition for the Willis F. Manges trophy (low net) and the Exhibitors' Trophy (low gross) will be available to you at a later date. There will also be arrangements for a tennis tournament, as this has developed into a popular portion of the social program. The Ladies' Local Arrangement Committee, chaired by Mrs. Tom Fullenlove, is in the process of organizing a very interesting program which will include a day's tour to the wine country of California on Monday, September 23, and during another day a tour of the harbor of San Francisco.

The Seventy-fifth Annual Meeting will begin formally on Tuesday at 8:30 A.M., September 24, and will be called to order by our President, Dr. William B. Seaman. This will be followed by the Installation of the President-Elect by Dr. Seaman and Dr. Harry Z. Mellins, Chairman of the Executive Council of the Society. A brief presidential address will be delivered by the incoming president, Dr. Colin B. Holman. The Scientific Program will begin at 9:00 A.M.

A total of 65 papers has been selected, and the format of the meeting has been changed slightly with respect to the last day of the meeting. On Friday, beginning at 8:30 A.M., there will be two simultaneous diagnostic sessions in order that the Instruction Courses for Friday may be started at 11:00 A.M. Past experience has shown that many members and guests have had to leave the meeting sometime shortly after noon on Friday. One of the diagnostic sections on Friday will be concerned with general radiologic topics. The second section, conducted simultaneously, will be devoted to the more specialized techniques. Members of the audience may wish to select the papers involving their own special interests and transfer from one section to the other as appropriate.

Last year the tradition was changed in that the Caldwell Lecture was delivered during prime time rather than in the evening. Again this year, it will be delivered at 11:30 A.M. on Wednesday. This year the Caldwell Lecture will be given by Dr. Collin S. MacCarty, Professor and Chairman of the Department of Neurologic Surgery at the Mayo Clinic, Rochester, Minnesota. Dr. MacCarty has always been a close friend to all radiologists and is especially interested in neuroradiology and in the programs we are developing in this

field. He has done much to develop a mutual understanding in respect to the relationships between radiology, neurosurgery and neurology. He is past president of the American Association of Neurological Surgeons (Cushing Society), past president of the Neurosurgical Society of America, and has held many other important positions in the national and international affairs of neurological surgery. His subject will be announced later and he will be introduced by a past president of our Society, Dr. C. Allen Good, former Professor and Chairman of the Department of Radiology at the Mayo Clinic. Dr. MacCarty is an extremely effective public speaker and is very much interested in continuing to develop helpful intercommunication between radiologists and neurosurgeons. It is interesting to note that his brother and his son are radiologists.

The Society will continue its custom of presenting Award Papers. This year these will include presentations from the American Radium Society, the Society for Pediatric Radiology, the American Society of Neuroradiology, and the Society of Gastrointestinal Radiologists.

Dr. Harold O. Peterson, the Director of the Instruction Courses, is again making arrangements for these valuable functions of our meeting. The number of courses has been expanded by scheduling some of these sessions on Monday, September 23, 1974, and as mentioned previously, the Friday courses will be held Friday morning from 11 o'clock through 12:30 P.M., in order to accommodate those who might otherwise miss a session on the last day of the meeting.

The Scientific Exhibits are always extremely interesting and a most important contribution to our meeting. Once again, they will be arranged by Dr. James Martin, together with his capable committee.

The Technical Exhibits under the supervision of Dr. George A. Kling will be on view from Monday through Friday and the locations of these exhibits will be available at a later date.

We are greatly indebted to the American College of Radiology and particularly to Ms. Sheila McGing for managing many of the general aspects of the meeting itself. The College will also put on an interesting program Wednesday afternoon.

The Executive Council at its last meeting decided that the Annual Banquet would be eliminated because it seemed to conflict with the evening plans of a large number of the members and guests attending the Annual Meeting. In its place, there will be a Presidential Reception on Thursday evening, from 6:30 P.M. through 9:00 P.M., to which all registered physicians and registered wives will be guests of the Society. A limited number of invitations will be issued to each of the staffs attending the Technical Exhibits. This reception is an effort on the part of the Society to make it possible for old friends to reunite and for all to be able to meet with the Officers and many contributors to the Seventy-fifth Annual Meeting.

Another innovation this year has to do with a change in the registration activities. It has been determined that the Society must now follow the lead of most other national societies in charging a modest registration fee to non-members and guests in order to partially defray the costs of the meeting which are very much greater than they have been in the past. There will be no registration fee for members of the American Roentgen Ray Society.

This issue of the JOURNAL contains printed registration forms which should be filled out and mailed as directed, together with a registration fee, if appropriate. The registration fee for non-members and guests is $25 and for *bona fide* residents training in radiology the registration fee will be $15. For those who have preregistered, programs, badges and tickets for the Presidential Reception will be available at stations so designated at the hotel. There will be provisions made for registration at the meeting, but it is hoped that most members and guests will take advantage of preregistration in order to

ADVANCE REGISTRATION

for the 75th annual meeting of the American Roentgen Ray Society in San Francisco September 23 to September 27, 1974, at the San Francisco Hilton Hotel.

If you plan to attend, please fill out the following forms, and mail each as indicated. Official badges and program booklets will be available at the registration desk in the San Francisco Hilton Hotel at the time of the meeting.

Preregistration by mail will be accepted only until September 5, 1974.

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20 North Wacker Drive, Chicago, Illinois 60606

Check one box, and include the proper fee in your envelope:

- ☐ Member of the American Roentgen Ray Society (no fee).
- ☐ Non-member (registration fee $25.00).
- ☐ Physician in training (registration fee $15.00). Be sure to include the certification on the reverse of this form.

Please print or type:

Name _____

Street _____

City _____

State or Province and zip _____

☐ Check here if you wish a program of events for ladies. Indicate the address to which this is to be sent.

Name _____

Street _____

City _____

State or Province and zip _____

MAIL TO THE SAN FRANCISCO HILTON

Ellis and Mason Streets, San Francisco, Calif. 94102
Attention: Front Office Manager

Please reserve for me:

MAIN BUILDING		TOWER	
Single	$21–40	Single	$31–48
Dble/twin	$30–49	Dble/twin	$39–58
Suites	$75 up	Suites	$95 up

Please circle your preference.

Name _____

Street _____

City _____

State or Province and zip _____

Arrival date and time _____

Departure date and time _____

FOR PHYSICIANS IN TRAINING:

_____ is in training in my department.

_____
Department Chief

_____ _____
Date Institution

avoid long delays and complications at the registration desks. This area will be open on Sunday, September 22, 1974.

Wives of members and guests may write to Mrs. Fullenlove (1200 California St., Apt. 23 D, San Francisco, CA 94109) or the American College of Radiology for information concerning the ladies' events and for a preregistration form. This form should be returned to her together with a check to cover the cost of whatever activities are chosen. It is most important to remember that the check must be made out exactly as Mrs. Fullenlove requests in her brochure as these funds cannot be co-mingled with the general funds of the American Roentgen Ray Society. Each lady registered will receive a free ticket inviting her to accompany her husband to the Presidential Reception, whether she participates in other activities or not.

All this seems a little complicated, but I am sure that with understanding on the part of the members and guests and the many devoted members of the staffs of our Society and of the American College of Radiology, it will all work out satisfactorily. It is with regret that the Society must change its traditional policy of not charging a registration fee, but during these days of rapidly increasing costs, it is no longer possible to develop and present a satisfactory program of this size and nature without some financial support from those taking part.

We expect the Seventy-fifth Annual Meeting to be a scientifically stimulating and a socially pleasant meeting in a delightful location. We sincerely urge all members of the Society, as well as all radiologists and others who are interested in the associated fields involved with the therapeutic and imaging functions of radiant energy in all its forms, to join us in San Francisco in September.

Colin B. Holman, M.D.
President-Elect
American Roentgen Ray Society

PRELIMINARY PROGRAM

SEVENTY-FIFTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY, SEPTEMBER 24–27, 1974 SAN FRANCISCO HILTON HOTEL, SAN FRANCISCO, CALIFORNIA

Tuesday, September 24, 1974

8:30 A.M.

Call to Order, Seventy-fifth Annual Meeting: William B. Seaman, M.D., New York, New York, President.

Installation of President-Elect, Colin B. Holman, M.D., Rochester, Minnesota, by William B. Seaman, M.D., New York, New York, President, and Harry Z. Mellins, M.D., Boston, Massachusetts, Chairman of the Executive Council.

Presentation of Past Presidents' Book to William B. Seaman, M.D., by Colin B. Holman, M.D.

Presidential Address: President Colin B. Holman, M.D.

9:00 A.M.

Presiding: Colin B. Holman, M.D.
Rochester, Minnesota

1. *Award Paper, The Society for Pediatric Radiology*. To be announced.
2. Gastrointestinal Perforations in Infancy. Arthur S. Tucker, M.D., Cleveland, Ohio, and *Leslie Soine, M.D., Cleveland, Ohio.
3. Ischemic Bowel in Neonates. *Howard Cockrill, M.D., Durham, N.C., *Herman Grossman, M.D., Durham, N.C., and *Frederick Meine, M.D., Durham, N.C.
4. A Distinctive Radiologic Appearance of *Mycoplasma* Pneumonia and Relationship to Immunologic Disorders. *Charles E. Putman, M.D., New Haven, Conn., *J. Bernard Gee, M.D., New Haven, Conn., and *Edward Druy, M.D., New Haven, Conn.
5. The Roentgen Manifestations of Melioidosis. *George P. Saba, II, M.D., Baltimore, Md., A. Everette James, Jr., M.D., Baltimore, Md., and *Maurice Reeder, M.D., Baltimore, Md.
6. Roentgenologic Observation of Pulmonary Arterial Hypertension in Association with Raynaud's Phenomenon and Systemic Sclerosis. Richard J. Steckel, M.D., Los Angeles, Cal., *Marshall Bein, M.D., Los Angeles, Cal., *Peter Kelly, M.D., Los Angeles, Cal., and *Vincent Ricchiuti, Los Angeles, Cal.
7. Current Concepts with Respect to Chondrocalcinosis and the Pseudogout Syndrome. *Pamela S. Jensen, M.D., New Haven, Conn., and *Charles E. Putman, M.D., New Haven, Conn.
8. Calcifications and Other Soft Tissue Stigmata in Systemic Lupus Erythematosus. *Joel A. Budin, M.D., New York, N.Y., and Frieda Feldman, M.D., New York, N.Y.
9. Primary Bone Tumors of the Clavicle and Scapula. *Julius Smith, M.D., New York, N.Y., *Daniel L. McLachlan, M.D., New York, N.Y., *Andrew G. Huvos, M.D., New York, N.Y., and *Norman L. Higinbotham, M.D., New York, N.Y.
10. The Navicular Fat Stripe. *Daniel William Terry, Jr., M.D., Milwaukee, Wis., and *James E. Ramin, M.D., Milwaukee, Wis.
11. Multicentric Reticulohistiocytosis: A Non-Inflammatory Polyarthritis with Distinctive Roentgenographic Features. *Richard H. Gold, M.D., Los Angeles, Cal., *Allen Metzger, M.D., Los Angeles, Cal., *Kate Killebrew, M.D., Los Angeles, Cal., and *Howard L. Weinberger, M.D., Los Angeles, Cal.
12. The Importance of Coronary Arteriography in the Evaluation of Patients with Aortic Valvular Disease. *Thomas A. Sos, M.D., New York, N.Y., and *Harold A. Baltaxe, M.D., New York, N.Y.
13. Coronary Reciprocating Flow: Its Value in Determining Myocardial Viability. *Hugo Spindola-Franco, M.D., Boston, Mass., *Douglass F. Adams, M.D., Boston, Mass., and Herbert L. Abrams, M.D., Boston, Mass.

Wednesday, September 25, 1974

8:30 A.M.

Presiding: Roderick L. Tondreau, M.D.
Meadowbrook, Pennsylvania

14. *Award Paper, The American Society of Neuroradiology*. To be announced.

* By invitation.

15. Early Diagnosis of Functioning Pituitary Gland Adenomas. Benjamin Kaufman, M.D., Cleveland, Ohio, *Olof H. Pearson, M.D., Cleveland, Ohio, *Jerald S. Brodkey, M.D., Cleveland, Ohio, and *Glenn F. Sykora, M.D., Cleveland, Ohio.
16. Metastatic Disease of the Sella Turcica. *Michael Kistler, M.D., Long Beach, Cal., and Henry W. Pribram, M.D., Long Beach, Cal.
17. The Radiographic Spectrum of Lesions Affecting the Sphenoid Sinus. (Sc. Ex.) Heun Y. Yune, M.D., Indianapolis, Ind., *Robert Holden, M.D., Indianapolis, Ind., and *John A. Smith, M.D., Indianapolis, Ind.
18. Angiography of Posterior Third Ventricular Masses. James C. Hoffman, Jr., M.D., Atlanta, Ga., and *Peter J. Sones, Jr., M.D., Atlanta, Ga.
19. Intracranial Hemangiopericytomas: Angiography, Pathology, and Differential Diagnosis. (Sc. Ex.) *Joseph A. Marc, M.D., Atlanta, Ga., M. M. Schechter, M.D., Atlanta, Ga., *Y. Tekai, M.D., Atlanta, Ga., and J. C. Hoffman, M.D., Atlanta, Ga.
20. Ultrasonic B-mode Scans of the Carotid Arteries. Richard D. Anderson, M.D., Birmingham, Ala.
21. Spinal Cord Microangiographic Changes in Experimental Transitory and Permanent Paraplegic Injuries. *William E. Allen, II, M.D., New Haven, Conn., *Charles G. D'Angelo, M.D., New Haven, Conn., and *E. Leon Kier, M.D., New Haven, Conn.
22. Radiologic Findings in Cerebellar Hematoma and Infarction Secondary to Hypertension. *James D. Massie, M.D., Memphis, Tenn., *Sergui Haussen, M.D., Memphis, Tenn., and Barry Gerald, M.D., Memphis, Tenn.
23. An Investigation for Improving Diagnostic Information Extraction from CAT Scans. *David F. Reese, M.D., Rochester, Minn., *Edwin C. McCullough, Ph.D., Rochester, Minn., *George W. Beeler, Jr., Ph.D., Rochester, Minn., and *Mr. Paul R. Gerding, Rochester, Minn.
24. Experimental Studies to Determine Application of EMI Scanning to the Human Body. Ralph J. Alfidi, M.D., Cleveland, Ohio, Thomas F. Meaney, M.D., Cleveland, Ohio, and *William J. MacIntyre, Ph.D., Cleveland, Ohio.

* By invitation.

Wednesday, September 25, 1974

11:30 A.M.

THE CALDWELL LECTURE

By

Collin S. MacCarty, M.D., Professor and Chairman, Department of Neurologic Surgery, Mayo Clinic
Rochester, Minnesota

Introduction by C. Allen Good, M.D., Past President, American Roentgen Ray Society; former Professor and Chairman, Department of Radiology, Mayo Clinic, Rochester, Minnesota.

Presentation of Caldwell Medal to Dr. MacCarty by President Holman.

THERAPY PROGRAM

Wednesday, September 25, 1974

8:30 A.M.

Presiding: *Felix N. Rutledge, M.D.
Houston, Texas

T1. *Award Paper, The American Radium Society*. To be announced.
T2. The Role of Radiation Therapy in the Treatment of Meningiomas. Harry Newman, M.D., San Francisco, Cal., *Jeannette Townsend, M.D., San Francisco, Cal., Glenn E. Sheline, M.D., San Francisco, Cal., *William M. Wara, M.D., San Francisco, Cal., and *Edwin B. Boldrey, M.D., San Francisco, Cal.
T3. Radiotherapeutic Management of Malignant Melanoma of the Eye. *Gunther Ehlers, M.D., Columbus, Ohio, Frank Batley, M.D., Columbus, Ohio, and *Mukund Kartha, Ph.D., Columbus, Ohio.
T4. The Experience with Well Differentiated Adenocarcinoma of the Endometrium. *Nathan Green, M.D., Los Angeles, Cal., *Roy Wilbur Melbye, M.D., Los Angeles, Cal., and *Jules Kernen, M.D., Los Angeles, Cal.
T5. A Retrospective Study of Mortality From Adenocarcinoma of the Endometrium. *A. Robert Kagan, M.D., Los Angeles, Cal., *Herman Nussbaum, M.D., Los Angeles, Cal., and *Harry Ziel, M.D., Los Angeles, Cal.
T6. Specimen Xerography in Radical Vulvectomy. (Sc. Ex.) *Eduard Friedrich, M.D.,

Milwaukee, Wis., and *John R. Milbrath, M.D., Milwaukee, Wis.

T7. Localization of Tc^{99m} Polyphosphate in Soft Tissue Malignancies. *Lawrence Steven Richman, M.D., Pittsburgh, Pa., *Lewis William Gumerman, M.D., Pittsburgh, Pa., and *Geoffrey Levine, M.S., Pittsburgh, Pa.

T8. Bone Scanning—Osteogenic Sarcoma: Correlation with Surgical Pathology. *Amy Beth Goldman, M.D., New York, N.Y., Melvin Harry Becker, M.D., New York, N.Y., *Philip Braunstein, M.D., New York, N.Y., *Kenneth C. Francis, M.D., New York, N.Y., *Nancy Branom Genieser, M.D., New York, N.Y., and *Hossein Firooznia, M.D., New York, N.Y.

T9. Selective Transcatheter Infusion of Vasopressin: A Method for Radiation Protection of Normal Small Bowel and Liver. *William J. Casarella, M.D., New York, N.Y., *Herbert Gerstein, M.D., New York, N.Y., Chu Chang, M.D., New York, N.Y., and *Marianne Wolff, M.D., New York, N.Y.

Thursday, September 26, 1974

8:30 A.M.

Presiding: William B. Seaman, M.D.
New York, New York

25. *Award Paper, The American Society of Gastrointestinal Radiology*. To be announced.

26. Angiography and the Localization of the Site of Chronic Gastrointestinal Bleeding. *Patrick F. Sheedy, II, M.D., Rochester, Minn., and *Richard E. Fulton, M.D., Rochester, Minn.

27. Embolic Control of Gastric Arterial Bleeding. Stewart R. Reuter, M.D., Eloise, Mich., and *Vincent P. Chuang, M.D., Eloise, Mich.

28. Catheterization Diagnosis and Therapy of the Bleeding Marginal Ulcer. *Alfred Rosenbaum, M.D., Bronx, N.Y., Stanley S. Siegelman, M.D., Bronx, N.Y., and Seymour Sprayregen, M.D., Bronx, N.Y.

29. Prostaglandin E_1 in Non-Thrombotic Mesenteric Ischemia. *Lloyd John Davis, M.D., Houston, Tex., *James Howard Anderson, Ph.D., Houston, Tex., Sidney Wallace, M.D., Houston, Tex., and *Eugene D. Jacobson, M.D., Houston, Tex.

30. Preoperative Arteriographic Localization of Islet Cell Tumors. *Richard E. Fulton, M.D., Rochester, Minn., *Patrick F. Sheedy, M.D., Rochester, Minn., *Donald C. McIlrath, M.D., Rochester, Minn., and *Deward O. Ferris, M.D., Rochester, Minn.

31. Diagnostic Accuracy of Endoscopic Pancreatography. Marcia K. Bilbao, M.D., Portland, Ore., and Charles T. Dotter, M.D., Portland, Ore.

32. Comparative Detection of Pancreatic Body-Tail Enlargement of the Supine Translateral and Axial Pancreatic Views: A Prospective Statistical Study. *Philip A. Sorabella, M.D., New York, N.Y., *William L. Campbell, M.D., New York, N.Y., *Andre Varma, M.D., New York, N.Y., and William B. Seaman, M.D., New York, N.Y.

33. Cystic Adenomatoid Malformation of the Lungs—Morphologic Analysis. (Sc. Ex.) *John E. Madewell, M.D., Washington, D.C., *John T. Stocker, M.D., Washington, D.C., and *Jack M. Korsower, M.D., Washington, D.C.

34. Observations of Splenic Circulation: Normal and Pathologic. (Sc. Ex.) James H. Scatliff, M.D., Chapel Hill, N.C., *Otis N. Fisher, M.D., Chapel Hill, N.C., and *W. Bonner Fuilford, M.D., Chapel Hill, N.C.

35. Benign or Minimal Deviation Hepatoma in Non-Cirrhotic Livers. *Shelby J. Galloway, M.D., New York, N.Y., William B. Seaman, M.D., New York, N.Y., and *Raffaele Lattes, M.D., New York, N.Y.

36. Current Perspectives on Giardiasis. (Sc. Ex.) *Charles H. Fisher, M.D., Baltimore, Md., *Kook Sang Oh, M.D., Baltimore, Md., *Theodore M. Bayless, M.D., Baltimore, Md., and Stanley S. Siegelman, M.D., Baltimore, Md.

37. Regional Enteritis of the Duodenum. *William M. Thompson, M.D., Durham, N.C., *Howard H. Cockrill, M.D., Durham, N.C., and Reed P. Rice, M.D., Durham, N.C.

38. Crohn's Disease of the Stomach—The Ram's Horn Sign. Jack Farman, M.D., Brooklyn, N.Y., David Faegenburg, M.D., Brooklyn, N.Y., and *Serge Dallemand, M.D., Brooklyn, N.Y.

39. Constrictive Pericarditis and Protein Losing Enteropathy—An Imitator of Intesti-

* By invitation.

nal Lymphangiectasia. *David A. Kumpe, M.D., Baltimore, Md., *Richard B. Jaffe, Baltimore, Md., *Thomas A. Waldmann, Baltimore, Md., and *Meredith A. Weinstein, Baltimore, Md.

40. Breast Cancer Screening Clinic—First Year's Experiences—Problems and Results. (Sc. Ex.) Thomas Carlile, M.D., Seattle, Wash., *Willis J. Taylor, M.D., Seattle, Wash., and *McClure H. Hall, M.D., Seattle, Wash.

Friday, September 27, 1974

8:30 A.M.–10:30 A.M.

SECTION I

Presiding: Walter M. Whitehouse, M.D.
Ann Arbor, Michigan

41. Diagnosis of Perirenal and Subcapsular Masses: Anatomic-Radiologic Correlation. Morton A. Meyers, M.D., New York, N.Y., *Joseph P. Whalen, M.D., New York, N.Y., and John A. Evans, M.D., New York, N.Y.
42. The Occult Duplex Kidney and the Polar-notch sign: An Important Finding in Subcapsular Nonfunctioning Upper Pole Ureteral Duplications. Richard C. Pfister, M.D., Boston, Mass., and *W. Hardy Hendren, M.D., Boston, Mass.
43. Urographic Findings in Adult Type Polycystic Renal Disease. *Jerald H. Pietan, M.D., Rochester, Minn., *Robert R. Hattery, M.D., Rochester, Minn., *Ross M. Tucker, M.D., Rochester, Minn., and *Glen W. Hartman, M.D., Rochester, Minn.
44. Urologic Manifestations of Sickle Hemoglobinopathies. *Sanford D. Minkin, M.D., Baltimore, Md., *K. Sang Oh, M.D., Baltimore, Md., *Samuel Charache, M.D., Baltimore, Md., *Roger C. Sanders, B.M., Baltimore, Md., and Stanley S. Siegelman, M.D., Baltimore, Md.
45. New Thoughts Concerning Xanthogranulomatous Pyelonephritis. (Sc. Ex.) *Stephen L. Gammill, M.D., Memphis, Tenn., *Robert Peace, M.D., Memphis, Tenn., *Elliot Himmelfarb, M.D., Memphis, Tenn., *Steve Sorgen, M.D., Memphis, Tenn., Jack Rabinowitz, M.D., Memphis, Tenn., and *Lowell Hurwitz, M.D., Memphis, Tenn.
46. The Specificity of Renal Vein Thrombosis. (Sc. Ex.) *Charles Mulhern, M.D., Philadelphia, Pa., Peter H. Arger, M.D., Philadelphia, Pa., and Wallace T. Miller, M.D., Philadelphia, Pa.
47. Evaluation of Pelvic Masses During Infusion Excretory Urography. *Thomas J. Imray, M.D., Milwaukee, Wis.
48. The Utilization and Efficacy of Pelvimetry. *Kevin M. Kelly, M.D., Los Angeles, Cal., *Dale A. Madden, M.S., Los Angeles, Cal., *Joseph S. Arcarese, M.S., Los Angeles, Cal., and *Reynold F. Brown, M.D., Los Angeles, Cal.

Friday, September 27, 1974

8:30 A.M.–10:30 A.M.

SECTION II (To be announced)

49. Angiotomography of the Liver. *James J. McSweeney, M.D., New York, N.Y., and Robin Caird Watson, M.D., New York, N.Y.
50. "BASMA"-Biplane Alternate Stereo Magnification Angiography. E. Ralph Heinz, M.D., Pittsburgh, Pa., *Richard L. Goldman, M.D., Pittsburgh, Pa., *Louis N. Scotti, M.D., Pittsburgh, Pa., *Donald Hardman, M.D., Pittsburgh, Pa., and *Ted Parrot, Pittsburgh, Pa.
51. Ultrasonically Guided Percutaneous Pancreatic Biopsy. *Soren Hancke, M.D., Boston, Mass., *Hans H. Holm, M.D., Boston, Mass., *Royal J. Bartrum, Jr., M.D., Boston, Mass., and *Edward H. Smith, M.D., Boston, Mass.
52. Echography in the Investigation of Renal Failure. *Roger Cobban Sanders, M.D., Baltimore, Md.
53. Clinical Feasibility Study Using an Acoustical Holography Medical Imaging System. (Sc. Ex.) *A. F. Metherell, Ph.D., Orange, Cal., *J. J. Flynn, M.D., Orange, Cal., and B. J. O'Loughlin, M.D., Orange, Cal.
54. Radionuclide Perfusion Lymphangiography Using Tc^{99m}-Serum Albumin. Richard J. Steckel, M.D., Los Angeles, Cal., *Stanley Furmanski, M.D., Los Angeles, Cal., *Roger Dunham, Los Angeles, Cal., *Nancy Ross, B.A., Los Angeles, Cal., *Norman Poe, M.D., Los Angeles, Cal., and *Harold Snow, D.V.M., Los Angeles, Cal.
55. A Comparison of an Air-Gap and a Grid in Roentgenography of the Chest. E. Dale

* By invitation.

Trout, D.Sc., Corvallis, Ore., *John P. Kelley, B.S., Corvallis, Ore., and *Vaughn L. Larson, B.S., Corvallis, Ore.

56. Adverse Reaction to Intravascularly Administered Contrast Media: A Comprehensive Study Based on the Results of a Prospective Survey. (Sc. Ex.) William H. Shehadi, M.D., Byram, Conn.

WORKSHOPS ON FEDERAL DIAGNOSTIC X-RAY STANDARD

Department of Health, Education, and Welfare

A FEDERAL standard for diagnostic x-ray equipment becomes effective August 1, 1974. This equipment standard primarily applies to manufacturers and assemblers, but users are also affected.

Under the standard, x-ray manufacturers are responsible for producing equipment and components that perform according to requirements of the standard. Assembler's primary responsibility is to install the system according to the manufacturer's specifications and to use the type of components called for by the standard. He must certify that these two conditions have been met by filing specified forms with the Food and Drug Administration's Bureau of Radiological Health, the State Radiation Control Agency, and the Purchaser.

One of the principal protection provisions of the standard requires machines to be capable of restricting the x-ray beam to the size of the film or fluoroscopic image receptor. The standard also contains provisions intended to make it possible for operators to reproduce more consistently a given image quality for given voltage, current, and time settings. This capability, in combination with good x-ray examination techniques, will tend to minimize film retakes and unnecessary exposure.

To familiarize persons who are affected by the new standard, especially commercial installers and users who may perform their own installations, with their responsibilities under the new regulations, workshops are being conducted by the Food and Drug Administration. These one-day sessions are being held in various parts of the U. S. Persons interested in attending are urged to contact the FDA Radiation Control Officer in their region for additional information. Workshops will also include discussions of proposed federal requirements involving resale of used x-ray equipment.

The following are the Regions, their Radiation Control Officers, their addresses, and their contact telephone numbers: *Region I*, Joseph Arnaudo (Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, Vermont), 585 Commercial Street, Boston, Massachusetts 02109, Telephone: 617-223-5859; *Region II*, James J. Cherniack (New Jersey, New York, Puerto Rico, Virgin Islands), 850 Third Avenue, Brooklyn, New York 11232, Telephone: 212-788-1451; *Region III*, Henry Kocol (Delaware, District of Columbia, Maryland, Pennsylvania, Virginia, West Virginia), 1204 U. S. Customhouse, Room 902B, Second and Chestnut Streets, Philadelphia, Pennsylvania 19106, Telephone: 215-597-4506; *Region IV*, Thomas R. Johnson, Jr. (Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, Tennessee), 880 West Peachtree Street N.W., Atlanta, Georgia 30309, Telephone: 404-526-3576; *Region V*, Richard H. Cooke (Illinois, Indiana, Michigan, Minnesota, Ohio, Wisconsin), Room A-1945, 175 West Jackson Boulevard, Chicago, Illinois 60604, Telephone: 312-353-5244; *Region VI*, Paul A. Dickson (Arkansas, Louisiana, New Mexico, Oklahoma, Texas), Room 470B, 500 South Ervay Street, Dallas, Texas 75201, Telephone: 214-749-2225; *Region VII*, Donald R. Tillery (Iowa, Kansas, Missouri, Nebraska), 1009 Cherry Street, Kansas City, Missouri 64106, Telephone: 816-374-3817; *Region VIII*, Walter R. Payne (Colorado, Montana, North Dakota, South Dakota, Utah, Wyoming), Room 500, U. S. Customhouse, Denver, Colorado 80202, Telephone: 303-837-4917; *Region IX*, Dale A. Stevenson (Arizona, California, Hawaii, Nevada, Guam, American Samoa), Federal Office Building, 50 Fulton Street, San Francisco,

California 94102; *Region X*, Wilbur F. Van Pelt (Alaska, Idaho, Oregon, Washington), Room 5003, Federal Office Building, 909 First Avenue, Seattle, Washington 98104, Telephone: 206-442-5740.

For additional information please contact Michael E. W. Shaffer, Public Information Officer, Bureau of Radiological Health, Food and Drug Administration, 5600 Fishers Lane (HFX-25), Rockville, Maryland 20852, Telephone: 301-443-3434.

NEWS ITEMS

THE AMERICAN COLLEGE OF RADIOLOGY

The Fifty-first Annual Meeting of the American College of Radiology was held at the Fairmont Roosevelt Hotel, New Orleans, Louisiana, from Sunday, March 31 to Friday, April 5, 1974.

The following officers were elected: *President*, Paul A. Riemenschneider, M.D., Santa Barbara, California; *Vice-President*, J. Richard Raines, M.D., Portland, Oregon; *Secretary-Treasurer*, Newton Hornick, M.D., Pittsburgh, Pennsylvania; *Chairman of The Board of Chancellors*, Robert E. Wise, M.D., Boston Massachusetts; *Vice-Chairman of The Board of Chancellors*, John M. Dennis, M.D., Baltimore, Maryland.

The Convocation ceremonies were held Tuesday at 5:00 P.M. in the Grand Ballroom, with Dr. Robert D. Moseley, Jr., President of the American College of Radiology, presiding. The degree of fellowship was conferred on 95 members of the College, who had been approved by The Board of Chancellors and elected by the votes of the Fellows. At these ceremonies, Gold Medals, the highest award of The American College of Radiology for "distinguished service to radiology," were presented to Drs. William E. Allen and Albert Jutras, and to Dr. J. Frank Walker, posthumously.

On Wednesday, at 6:30 P.M., the College had a Joint Reception in the Grand Ballroom with the Louisiana Radiological Society.

The Teachers Conference started Thursday with a Luncheon in the Grand Ballroom and continued Thursday afternoon and all day Friday in 6 consecutive Sessions. The topics of these Sessions were: I. The Proliferation of Postgraduate Education Programs; II. Innovation in Resident Assessment; III. The Position of Newer Techniques in Radiology Residency Programs; IV. Manpower Update: How Many Residents are Recognized?; V. The Impact of Public Law #92-603 (HR-1) on the Financing of Training Programs; Fee for Service *Versus* Cost Reimbursement in the Teaching Hospital; and VI. The Residency Matching Program: The Phoenix in Radiology.

There was an extensive and most attractive Ladies Program organized by Mrs. Lorna Buchtel and her able Committee. It consisted of: *Monday*, Strolling Tour of the French Quarter; *Tuesday*, The famous Garden District Tour; *Wednesday*, The Great River Road Plantation Tour; and *Thursday*, Vistas and Views of New Orleans.

The Fifty-second Annual Meeting of the College will be held at the Portland Hilton Hotel in Portland, Oregon, March 31–April 4, 1975.

This year's Annual Meeting was followed by the "1974 Jamaican Radiological Conference"; officially sponsored by the College, which was held April 6–13, 1974, at the Jamaica Holiday Inn in Montego Bay. The distinguished Faculty Members of this Conference were Drs. Milton Elkin, Theodore E. Keats, William B. Seaman, Jerome F. Wiot and Robert E. Wise.

An optional extension of the tour to Nassau, Bahamas, was scheduled for April 13–16, 1974.

THE ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

An outstanding program has been prepared for the Thirty-sixth Annual Midsummer Conference on the Rocky Mountain Radiological Society, which will be held at the Brown Palace Hotel in Denver on August 22, 23, and 24, 1974.

The list of speakers includes: Elias G. Theros, M.D., Registry of Radiologic Pathology, Armed Forces Institute of

Pathology; Joseph P. Whalen, M.D., Professor of Radiology, Cornell Medical Center; Jack Edeiken, M.D., Professor of Radiology, Jefferson Medical College; Donald W. Brown, M.D., Professor of Radiology, Division of Nuclear Medicine, University of Colorado Medical Center; Marvin L. Daves, M.D., Professor of Radiology, Department of Radiology, University of Colorado Medical Center; and Atis K. Freimanis, M.D., Professor and Chairman of Radiology, Medical College of Ohio at Toledo.

Application for Continuing Medical Education Category I Credit has been made.

Participants who plan to have a scientific exhibit should contact John Eule, M.D., Department of Radiology, Denver General Hospital, West 8th Avenue at Cherokee Street, Denver, Colorado 80204.

For further information please write to W. K. Absher, M.D., President, 124 West Pitkin, Pueblo, Colorado 81005.

INSTITUTE FOR PEDIATRIC RADIOLOGY

The Institute for Pediatric Radiology and Chas. E. Shopfner, M.D., will present another in the Current Status of Pediatric Radiology series of programs titled "Workshop in Pediatric Radiology," August 17–18, 1974, at the Denver Hilton Hotel, 1550 Court Place, Denver, Colorado 80202.

The registration is unlimited.

For additional information please write: Institute for Pediatric Radiology, 4148 North Cleveland Avenue, Kansas City, Missouri 64117.

POSTGRADUATE COURSE IN NEURORADIOLOGY HARVARD MEDICAL SCHOOL

There will be a Postgraduate Course in Neuroradiology sponsored by Dr. Juan M. Taveras and Dr. Paul F. J. New and Harvard Medical School from October 7–10, 1974.

This 4 day course will cover most topics in the field of Neuroradiology and the Faculty will consist of Neuroradiologists from the United States and Canada.

Please send inquiries to: Harvard Medical School, Department of Continuing Education, 25 Shattuck Street, Boston, Massachusetts 02115.

AMERICAN CANCER SOCIETY, INC.

National Conference on Advances in Cancer Management

The American Cancer Society—National Cancer Institute sponsors the following: Part I: Treatment and Rehabilitation, November 25–27, 1974, at the Waldorf-Astoria Hotel—New York, New York; and Part II: Detection and Diagnosis, May 1–3, 1975, at the Denver Hilton—Denver, Colorado.

The American Cancer Society's National Conference on Gynecologic Cancer will be held at Marriott Hotel—Philadelphia, Pennsylvania, September 18–20, 1975.

The Eighth National Cancer Conference of the American Cancer Society—National Cancer Institute will be held at Regency Hyatt Hotel—Atlanta, Georgia, September 20–22, 1976.

These professional education conferences will be acceptable for Credit Hours in Category I for the Physician's Recognition Award of the American Medical Association and for Elective Hours by the American Academy of Family Physicians.

Details regarding specific hours of accreditation and registration will be provided by Evadne Gerrard, Assistant to the Vice President for Professional Education, 219 East 42nd Street, New York, New York 10017.

INTERNATIONAL SOCIETY FOR PAEDIATRIC NEUROSURGERY

The International Society for Paediatric Neurosurgery will hold its annual meeting in London, England, from September 13 through 14, 1974.

Information concerning this meeting may be obtained from Kenneth Till, The Hospital for Sick Children, Great Ormond Street, London WC IN 3JH, England.

VII CONGRESO NACIONAL DE RADIOLOGIA

The VII National Congress of Radiology (Mexico) will be held April 27 to May 3, 1975, in the city of Merida, Yucatan, Mexico.

For further information please write to Dr. Augusto Reyes Bolio, Secretary General, Av. Perez Ponce No. 492 B. APDO. Post No. 121, Merida, Yucatan, Mexico.

DIAGNOSTIC ULTRASOUND IN PEDIATRICS

A comprehensive program on the value of diagnostic ultrasound as applied to pediatrics will be presented at The Johns Hopkins Hospital on September 5–6, 1974.

Both practical and theoretical considerations which apply specifically to the pediatric age group will be stressed.

The faculty, which has been selected for its contributions to the field of pediatric ultrasound, includes Donald Brascho, Richard Dallow, John Dorst, Barry Goldberg, Walter Henry, Daniel McNulty, Richard Meyer, Daniel Pieroni, David Sahn, Roger C. Sanders, Sumio Uematsu, Roberta Williams, and others.

Further details can be obtained from Roger C. Sanders, M.D., Department of Radiology, The Johns Hopkins Hospital, Baltimore, Maryland 21205.

BOOK REVIEW

RADIOLOGY OF THE NEWBORN AND YOUNG INFANT. By Leonard E. Swischuk, M.D., Professor of Radiology and Pediatrics, The University of Texas Medical Branch, Galveston, Texas. Cloth. Pp. 602, with many illustrations. Price, $36.50. Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, 1973.

The incredible growth of pediatric radiology has led to specialization into pediatric newborn radiology, as well as subspecialization in genitourinary, gastrointestinal, cardiac and pulmonary disorders. Dr. Swischuk has taken on the difficult task of providing a single-authored text involving all neonatal and infant radiology. He has done this in a single volume text of almost 600 pages. The bibliography in each section is extensive and remarkable, being both up-to-date and including most interesting references to surgical and pediatric articles as well as to prior radiologic studies.

This reviewer had mixed feeling about the results. The positive points far outweigh the negative. The important areas are done well and include an extensive use of "special" procedures (*i.e.*, angiograms, isotopic studies) where applicable. An over-all view is maintained stressing conventional studies.

The chest section is quite good. This confusing area of "wet" lungs, "drowned" lungs, transient tachypnea, "reticulonodular" *versus* "reticular" patterns has always defied a definitive answer. Dr. Swischuk tries gamely but fails. He has no reason to be ashamed.

On the debit side, the reproduction of radiographic studies is often less than ideal. This was most noted in the genitourinary section. Dr. Swischuk's known interest in congenital heart disease and neurologic disease perhaps accounts for the extensive coverage (twice the pages given to genitourinary topics) and large size illustrations in an area of evaluation largely limited to teaching hospitals with busy cardiac and neuroradiologic "teams."

Another area of possible disagreement is the author's inclusion of innumerable entities rarely if ever seen in newborns and young infants with text, several references, but no illustrations and the repeated comment that the "following is rarely or uncommonly encountered" in this age group.

Is it fair for this reviewer to compare it to "Caffey"? Since this will inevitably be done, some comments are in order. Swischuk's work is much more limited in scope (although not enough as noted), physically much easier to use (with a superb table of contents). One is spared the long though rewarding search through the index. In depth of coverage and thinking on the varied areas of pediatric radiology it cannot be said to be up to "Caffey." I hope Dr. Swischuk will reduce the scope in the next edition and improve the quality of the reproductions. Overall, this is an excellent work and a supplement, rather than a replacement for "Caffey"—it is, incidentally, considerably less expensive as well.

WALTER E. BERDON, M.D.

BOOKS RECEIVED

THE YEAR BOOK OF RADIOLOGY, 1974. Radiologic Diagnosis: edited by Walter M. Whitehouse, M.D.; associate editors, Joseph J. Bookstein, M.D., Trygve O. Gabrielsen, M.D., John F. Holt, M.D., William Martel, M.D., and John R. Thornbury, M.D. Radiation Therapy: edited by Howard B. Latourette, M.D.; associate editors, A. Curtis Hass, M.D., and Robert T. Guthrie, M.D. Cloth. Pp. 435, with some illustrations. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill. 60601, 1974.

HUMAN RADIATION BIOLOGY. By Kedar N. Prasad, Ph.D., Associate Professor of Radiology; Chief, Section of Radiation Biology, University of Colorado Medical Center, Denver, Colo. Cloth. Pp. 524, with 162 illustrations. Price, $25.00. Harper & Row, Publishers, Inc., 2350 Virginia Avenue, Hagerstown, Md. 21740, 1974.

TUMORS OF THE EXTRA-ADRENAL PARAGANGLION SYSTEM (INCLUDING CHEMORECEPTORS). Atlas of Tumor Pathology, Second Series, Fascicle 9. By George G. Glenner, M.D., Chief, Section on Molecular Pathology, Laboratory of Experimental Pathology, National Institute of Arthritis, Metabolism, and Digestive Diseases, National Institutes of Health, Bethesda, Md.; and Philip M. Grimley, M.D., Head of Ultrastructural Pathology Section, Laboratory of Pathology, National Cancer Institute, National Institutes of Health, Bethesda, Md. Paper. Pp. 90, with many figures. Price, $4.50. Published by the Armed Forces In-

stitute of Pathology, Washington, D. C. under the auspices of Universities Associated for Research and Education in Pathology, Inc., Bethesda, Md. For sale by the American Registry of Pathology, Armed Forces Institute of Pathology, Washington, D. C. 20306, 1974.

Clark's Positioning in Radiography. Volume 1: Routine Radiography. Ninth edition. Revised by James McInnes, F.S.R., T.E., F.R.P.S. Cloth. Pp. 468, plus index, with many illustrations. Ilford Limited. William Heinemann Medical Books Ltd. Distributed by Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill. 60601, 1973.

How to Build and Use Electronic Devices Without Frustration, Panic, Mountains of Money, or an Engineering Degree. By Stuart A. Hoenig, Ph.D., Professor of Electrical Engineering, University of Arizona, Tucson; and F. Leland Payne, M.S.E.E., Design Engineer, Burr-Brown Research Corporation, Tucson. Paper. Pp. 192, with many figures. Price, $9.95. Little, Brown and Company, 34 Beacon Street, Boston, Mass. 02106, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Southampton Princess Hotel, Southampton, Bermuda, May 4–9, 1975.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Portland-Hilton Hotel, Portland, Oregon, March 31–April 4, 1975.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Chicago, Ill., June 15–19, 1974.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next year. New York, N. Y., June 16–22, 1974, New York Hilton Hotel; and Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers June 22, 1974.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1974 was September 30, 1973.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

The third examination is scheduled for Saturday, Sept. 21, 1974. Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017. The deadline for receipt of completed applications is June 1, 1974.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D. C. 20010. Annual Meeting.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting: Sonesta Beach Hotel, Key Biscayne, Fla., Oct. 30–Nov. 4, 1974.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

FOURTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Rio de Janeiro, Brazil in 1977.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aéreo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Meeting in June, 1975 in Edinburgh, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting: Chicago, Ill., June 22–23, 1974.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Richard H. Greenspan, Dept. of Radiol., Yale University School of Medicine, 333 Cedar St., New Haven, Conn. 06510.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Charles J. Masters, Maj. MC, Department of the Air Force, United States Air Force Hospital, Wiesbaden (USAFE) APO New York 09220.

Blockley Radiological Society
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Giacomo Pantaleo, 96-13 Forest Ave., Brooklyn, N. Y. 11227. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Albert A. Franco, 45 Alexander St., Lockport, N. Y. 14094. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, 27 Hurd Circle, Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio

Central Texas Radiology Society
Secretary-Treasurer, Dr. William H. Roush, Radiology Service, Veterans Administration Hosp., Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. George N. Weston, 1515 Ygnacio Valley Rd., Walnut Creek, Calif. 94598. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Menzie H. Brown, 155 S. 18th St., Kansas City, Kans. 66102.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Richard E. Litt, 6125 S.W. 31st St., Miami, Fla. 33155. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting: Houston, Texas, July 7–11, 1974.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John R. Dehner, Reid Memorial Hosp., Richmond, Ind. 47374.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference: Century Plaza Hotel, Los Angeles, Calif., Jan. 31–Feb. 2, 1975.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Eli A. Etscovitz, 10 Stonewood Dr., Newton Centre, Mass. 02159.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Howard L. Copas, 827 South 131st St., Omaha, Neb. 68154. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

New England Roentgen Ray Society
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02119. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting: May 10, 1974.

New England Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference, New Orleans, La., Apr. 25–27, 1974.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 S. University Dr., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Mark D. Reiss, 2100 Rockwood Dr., Sacramento, Calif. 95825. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

NORTHWESTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 22–24, 1974.

SAN ANTONIO-CIVILIAN MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President, Dr. Leland E. Kellerhouse, 2466 First Ave., San Diego, Calif. 92101. Meets the first Monday of each month at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION OF RADIOLOGY, NATIONAL MEDICAL ASSOCIATION
Chairman, Dr. Roy G. Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broad-

way, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. 21st Annual Meeting: Town and Country Hotel, San Diego, Calif., June 10–14, 1974.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 311 Medical Arts Bldg., Chattanooga, Tenn. 37402. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada
First European Course in Chest Radiology at the Royal College of Surgeons, London, W.C.1., May 20–23, 1974.
Hon. Secretary, Dr. Ian H. Kerr, 156 Lambeth Rd., London, S.E.1.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton; St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 8952 Bellfonte Rd., Richmond, Va. 23229.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Isaiah J. Seligman, 16 Guion Pl., New Rochelle, N. Y. 10801. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Francisco Miranbell S., Apartado 3352, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1 Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32

Welbeck St., London, W1M/7PG, England. Annual General Meeting: May 15–18, 1974.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St. Catherine St. West, Montreal 107, Que., Canada. Thirty-eighth Annual Meeting.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, Lincolns Inn Fields, London WC2A 3PN, England. Annual General Meeting: May 15–18, 1974.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. J. Jääskeläinen, The Children's Hospital,

Stenväckinkatu 11, SF-00290 Helsinki 29, Finland. Annual Meeting.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting in Uppsala, Sweden in June 1974.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa 1-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

Vol. 121, No. 2

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Head

Gilday, D. L. *et al.:* Subdural hematoma—what is the role of brain scanning in its diagnosis? 456
Waxman, A. D. *et al.:* Differential diagnosis of brain lesions by gallium scanning 456
Vignaud, J. *et al.:* Venography of the orbit: an analytical report of 413 cases 456

Neck and Chest

Gaux, J. C. *et al.:* Arteriographic signs of primary hyperparathyroidism: report of 10 cases 457
Johnson, T. H. *et al.:* Acquired tracheomalacia 457
Olbert, F. *et al.:* Roentgen diagnosis of so-called intralobar sequestration of the lung 458
Zwintz, von E.: Results after resection of pulmonary metastases 458
Stoloff, I. L.: The prognostic value of bronchoscopy in primary lung cancer: a new perspective for an old procedure 459

Abdomen

Warter, P. *et al.:* Radiologic findings in duodenal hematoma 459
Schechner, S. A. *et al.:* "Innocent" pneumobilia: report of a case and review of the literature 459
Popovsky, J. *et al.:* Liver trauma: conservative management and the liver scan 460
Macgregor, A. M. C., and Hawkins, I. F., Jr.: Selective pharmacodynamic angiography in the diagnosis of carcinoma of the pancreas. 460

Genitourinary System

Peeters, F. L. M.: The renal angiogram in tuberculosis 460
Jackman, R. J., and Stevens, G. M.: Benign hemorrhagic renal cyst: nephrotomography, renal arteriography, and cyst puncture 461
Pollack, H. M., and Popky, G. L.: Roentgenographic manifestations of spontaneous renal hemorrhage 461
Jaconette, J. R. *et al.:* Pericapsular renal veins: recognition without angiography 462
Laurent, Y.: Multiple diverticula or diverticulosis of the ureter; a rare radio-clinical syndrome 462
Catalano, D.: Selective pharmaco-angiographic control of post-traumatic hematuria 463
Elkin, M. *et al.:* Ureteral obstruction in patients with uterine prolapse 463

Skeletal System

Govoni, A. F.: Osteophytes of the cervical spine and dysphagia 463
Willich, von E., and Englert, M.: The metacarpal sign 464

RADIATION THERAPY

Wellwood, J. M., and Jackson, B. T.: The intestinal complications of radiotherapy 464
Huguier, M. *et al.:* Surgical treatment of Crohn's disease: a review of 120 cases 465
Martini, N. *et al.:* Bronchiolar carcinoma: a review of 152 patients 465
Tabár, L. *et al.:* The significance of mammography, galactography, and pneumocystography in detecting occult carcinomas of the breast 466
Horowitz, A. L. *et al.:* The roentgen features of synovial sarcomas 466

ROENTGEN DIAGNOSIS

HEAD

GILDAY, DAVID L., COATES, GEOFFREY, and GOLDENBERG, DAVID. Subdural hematoma—what is the role of brain scanning in its diagnosis? *J. Nuclear Med.*, May, 1973, *14*, 283–287. (Address: David L. Gilday, Division of Nuclear Medicine, The Hospital for Sick Children, 555 University Avenue, Toronto 2, Ontario, Canada.)

Forty patients with subdural hematomas confirmed at surgery were examined by both technetium 99m brain scanning and biplane arteriography. The duration of the lesion was determined histologically in all patients. In 30/32 patients with unilateral subdural hematoma, the scan was abnormal. Twelve of these patients had acute lesions where no membrane was found at surgery, and all had abnormal scans; 18/20 patients with chronic lesions had abnormal scans. There was no relationship between the activity seen in the lesion, and the chronicity of the lesion. In 8 patients with bilateral subdural hematomas (16 lesions), 4 were less than 1 cm. size, and only 1 of these was seen. Of the remaining 12 lesions, 11 were seen on the scan. All patients had abnormal cerebral arteriograms.

In another group of 98 patients in whom the clinical course was reviewed, 87 had a pre-scan diagnosis of subdural hematoma. In this group, 15 were clearly positive, and were subsequently found to have subdural hematoma. Eleven others were also interpreted as abnormal, and were found to have other pathology. In 20 patients with suspicious scans, 15 were found to have other pathology and 5 had no definite disease. Two patients had normal scans, but subdural hematomas were found in 2. No disease was found in the remaining 50.

When the patient has a subdural hematoma and the scan is abnormal, the lesion is usually clearly seen. When the scan is normal, the probability of a lesion is low. In this study, the false negative rate was 4 per cent, while the false positive rate was 11 per cent.—*William H. Strauss, M.D.*

WAXMAN, ALAN D., LEE, GEORGE, WOLFSTEIN, RALPH, and SIEMSEN, JAN K. Differential diagnosis of brain lesions by gallium scanning. *J. Nuclear Med.*, Dec., 1973, *14*, 903–906. (Address: A. D. Waxman, Nuclear Medicine Section, LAC/USC Medical Center, 1200 North State Street, Los Angeles, Calif. 90033.)

Seventy-seven patients underwent brain scanning with both technetium 99m pertechnetate and gallium 67 citrate. The pertechnetate image was obtained 4 hours after injection; the gallium scanning was performed 48–72 hours after administration and within 4 days of the technetium examination. There were no patients who had an abnormal gallium scan, who had a normal technetium examination. In 23 patients with cerebral infarction diagnosed by angiography, the gallium scan was completely normal, while the technetium examination was abnormal.

In neoplasm, 9/28 patients revealed increased gallium accumulation in the lesion. This accumulation was greater than that for technetium when the uptake was compared to the activity in the calvaria. In 17/28 patients with neoplasm, the concentration in the technetium scan was equal to that of the gallium, and in 2, the technetium concentration was greater.

The combined use of both tracers can increase the specificity of brain scanning, and aid in the differential diagnosis of tumors from stroke.—*William H. Strauss, M.D.*

VIGNAUD, JACQUELINE, CLAY, CLAUDE, and BILANIUK, LARISSA T. Venography of the orbit: an analytical report of 413 cases. *Radiology*, Feb., 1974, *110*, 373–382. (Address: Jacqueline Vignaud, M.D., Service de Radiologie, Fondation Ophthalmologique Adolphe de Rothschild, 29, rue Manin, 75019 Paris, France.)

Orbital venography provides excellent topographic information regarding intraorbital tumors and is recommended when clinical findings (most frequently exophthalmos) suggest orbital disease or involvement of the orbit and cavernous sinuses. It provides little etiologic information; however, a definite diagnosis can be made in the small group of vascular malformations and hemangiomas that are opacified directly and in fistulas manifest by nonopacification and increased venous pressure. Venography is not helpful in isolated thrombosis of the central retinal vein and in palpebral abnormalities. Investigation of known endocrine exophthalmos usually provides no helpful information.

The authors' experience and results of orbital venography in 413 cases is reviewed. Specific aspects of technique, anatomy, and interpretation are emphasized. A percutaneous frontal approach was used in 398 patients with a success rate of 89 per cent. The posterior route (jugulography) was the next approach of choice, but is not as satisfactory for filling of the smaller orbital veins. No severe complications were encountered in the patients studied. Measurement of orbital venous pressure with a water manometer is especially important in nonopacification, venous dilatation, or impeded flow of contrast material at the level of the cavernous sinus. Normal pressure in the supine position is between 12 to 16 cm. of water; a value greater than 25 cm. is highly suggestive of an arteriovenous fistula.

Combination of magnification and subtraction

techniques is helpful in the study of orbital radiographic anatomy and pathology. If the inferior orbital vein is opacified, it is helpful in identifying and localizing the abnormality more precisely. The inferior orbital vein is especially helpful in differentiating between intraconic and extraconic lesions in the inferior portion of the orbit. Medial and lateral anastomoses between the superior and inferior ophthalmic veins, called connecting veins, aid in delineating the location and extent of a lesion. The abnormal findings in orbital venography include: (a) lack of opacification; (b) displacement; (c) modification of the caliber and contour of vessels; (d) stain; and (e) a change in the mode of filling.

Venography does not compete with arteriography, which gives less topographic, but often more etiologic information. The 2 examinations complement one another. It is preferable to start with venography because of the much smaller risk involved, especially in elderly patients.

In patients with tumor exophthalmos, venography aids in localizing the tumor with regard to the 3 dimensions of the orbit, guiding selection of the surgical approach and, on occasion, to provide etiologic information. In the authors' experience, good clinical-radiologic-surgical correlation was obtained in the localization of intraorbital space-occupying lesions.—*J. L. Montgomery, M.D.*

Neck and Chest

Gaux, J. C., Bismuth, V., Fritel, D., Brisset, J. M., Blery, M., Baglin, A., and Fendler, J. P. Les signes artériographiques de l'hyperparathyroïdisme primaire: à propos de 10 observations. (Arteriographic signs of primary hyperparathyroidism: report of 10 cases.) *Presse méd.*, Nov., 1973, *2*, 2733–2737. (Address: Pr. ag. V. Bismuth, Service de Radiologie de l'Hôpital Ambroise-Paré, 9, avenue Charles-de-Gaulle, F 92104 Boulogne, France.)

Since surgical excision is necessary for adequate treatment of primary hyperparathyroidism—and even with surgical exploration it may be difficult to find the adenoma(s)—the usefulness of arteriography in identification and localization is considered.

Ten patients were investigated by complete retrograde preoperative arteriography of the parathyroid glands. Seven of these patients had renal stones and none had bone lesions. The adenoma was directly opacified in 6, although 3 of these required photographic subtraction for identification of the hypervascular lesion. The late arterial phase was best for discrimination from thyroid vascularity.

It is believed that indirect signs, such as hypertrophy of the inferior thyroid artery to a width of at least 2.5 mm., or opening of its second loop, have only suggestive value in the clinical situation.—*Alan E. Oestreich, M.D.*

Johnson, Thomas H., Mikita, John J., Wilson, Robert J., and Feist, J. H. Acquired tracheomalacia. *Radiology*, Dec., 1973, *109*, 577–580. (Address: Thomas H. Johnson, M.D., Department of Radiology, University of Oklahoma School of Medicine, Oklahoma City, Okla. 73190.)

Tracheomalacia is a process characterized by weakness of the tracheal walls and supporting cartilage. Primary tracheomalacia is a congenital abnormality with weakness of the tracheal walls, present at birth. Secondary tracheomalacia, or acquired tracheomalacia, is a disease process with weakening of the tracheal walls developing in later life. The common etiology appears to be inhalation of chronic irritants such as cigarette smoke and industrial air pollutants. Acquired tracheomalacia has previously been overlooked due to the difficulty of diagnosis. It is almost impossible to recognize on static suspended motion chest roentgenograms unless ectasia is present. Fluoroscopy and cine and tape recording utilizing quiet respiration and coughing has proved to be the optimal means of evaluation for this diagnosis.

The optimal patient position is standing with evaluation by fluoroscopy and motion recording in the posteroanterior and left anterior oblique positions. The left anterior oblique position appears optimal for evaluation of minimal degrees of tracheal collapse.

Numerous fluoroscopic observations and 10 cine recording studies were made in normal patients. In the authors' own series and others', there is less than one-half caliber collapse of the trachea during coughing in the anterior and left anterior oblique positions.

Thirty-five cases of acquired tracheomalacia were evaluated and classified by cine motion recording. These evaluations were carried out in the posteroanterior and left anterior oblique positions with bronchography and also without contrast agent. With motion recording these studies can be done without contrast material. Fluoroscopic visualization alone is difficult without contrast material, but still possible.

From the study of these 35 cases, a classification for the degree of tracheomalacia was developed. First degree tracheomalacia is collapse of one-half to three-fourths of the trachea with coughing. Second degree is collapse of three-fourths to complete collapse of the trachea with coughing. Third degree is complete collapse of the trachea with coughing. In fourth degree tracheomalacia, there is complete collapse of the trachea with coughing and ectasia of the trachea during suspended respiration.

Focal tracheomalacia is a definite localized change in tracheal caliber out of proportion to the mobility of the remaining trachea. The increase in severity of chronic obstructive pulmonary disease appears to parallel the degree of tracheomalacia, which appears to progress with increased age. Focal tracheomalacia

is often associated with surgery (tracheostomy, intubation).

The complete meaning of this correlation between degree of tracheomalacia and progressive severity of chronic obstructive pulmonary disease is not understood at the present time and no logical method of classification of the degree of tracheomalacia has been previously suggested. With the classification of tracheomalacia set forth here, a close evaluation of future cases will possibly pinpoint the correlation between tracheomalacia and progressive pulmonary disease.

Treatment of tracheomalacia with strut operations has been useful in delaying the progression of obstructive pulmonary disease, but it has not been completely successful, as malacia then develops above and below the point of the strut. Perhaps development of a treatment for tracheomalacia will also be instrumental in preventing progressive pulmonary disease.—*William O. Havens, M.D.*

OLBERT, F., WICKE, L., and ZWINTZ, E. Zur Röntgendiagnose der sogenannten intralobären Sequestration der Lunge. (Roentgen diagnosis of so-called intralobar sequestration of the lung.) *Radiol. clin. et biol.*, 1973, *42*, 90–109. (Address: Dr. F. Olbert, Leiter der Röntgenstation des Angiologisch-Diagnostischen Institutes, der I. Chirurgischen Abteilung des Krankenhauses der Stadt Wien-Lainz, Wolkersbergenstrasse 1, A-1130 Wien 13, Austria.)

Intralobar pulmonary sequestration represents a hamartoma-like malformation. It appears as a separate pulmonary mass in the lower lung fields posteriorly and obtains its blood supply from the descending aorta either above or below the diaphragm.

There are 3 variations according to the roentgen appearance: (1) pseudotumorous form being well encapsulated; (2) pseudoabscess form with presence of a fluid level; and (3) bronchiectatic form with multilocular cysts through communication with the stomach or esophagus. Communication with the bronchial tree may also occur.

Lateral tomograms aid in obtaining a detailed outline of the mass and occasionally in demonstrating a band-like density of the aberrant artery. Bronchograms show a displacement of the bronchial tree as it is encroached upon by the mass, while a selective aortogram will actually succeed in filling of the supplying artery. Three cases are reported and illustrated.

The first patient was a 20 year old woman with a group of round foci in the left lower lung field posteriorly, discovered as an incidental finding on routine chest roentgenograms. At surgery, 3 supplying arteries were found to originate from the abdominal aorta.

The second patient was a 33 year old woman suffering from a gynecologic disorder. Routine chest roentgenograms disclosed confluent band-like densities in the right lower lung field posteriorly as in bronchiectases. Tomograms showed cyst-like radiolucencies in the same area. Only the selective aortogram revealed the full extent of the mass supplied by an aberrant artery measuring 6 mm. in diameter.

The third patient was a 30 year old man, who had suffered from bronchiectases since childhood and from repeated bouts of hemoptyses alternating with copious purulent sputum for the past 10 years. Chest roentgenograms showed an ill-defined abnormal density in the left lower lung field posteriorly. A selective aortogram disclosed a large aberrant artery originating in the abdominal aorta.

In all 3 cases the aberrant arteries were ligated followed by a lobectomy which resulted in complete recovery.— *Ernest Kraft, M.D.*

ZWINTZ, VON E. Ergebnisse nach Resektion von Lungenmetastasen. (Results after resection of pulmonary metastases.) *Wien. klin. Wchnschr.*, Sept., 1973, *85*, 604–606. (From: I. Chirugische Abteilung des Krankenhauses der Stadt Wien-Lainz, Wolkersbergenstrasse 1, A-1130 Wien, Austria.)

Pulmonary metastases may remain asymptomatic for prolonged periods of time and when detected are too widespread for a surgical approach. Nevertheless, even a bilateral involvement and recurrent metastatic seeding form no absolute surgical contraindication.

In a series of 232 patients with pulmonary seeding, 31 (14.2 per cent) could be considered for an operation. There were 7 pneumonectomies, 2 bilobectomies, 17 lobectomies, 4 segmental resections, and 3 extirpations among 18 men and 13 women. In 2 patients it was possible to perform a second operation for recurrences which had developed after the first operation.

In the first patient a right upper lobe lobectomy had been performed 6 years after a nephrectomy. Fifteen months later a left upper lobe lobectomy was done and still 6 months later a cerebral metastasis was operated on. The patient lived an additional 9 months or 2½ years after the first operation on the right lung.

The second patient was a woman who had been operated on for a meningeal hemangiopericytoma. Thirteen years later a lobectomy of the left upper lobe was performed and still 15 months later a rib lesion in the same location was resected.

Solitary metastases were found in 18 patients with epithelial tumors (kidneys 10 times, colon 4 times, breast 3 times, and uterus 1 time) and in 15 patients with mesenchymal tumors (extremities 7 times; uterus and brain, 2 each; breast, eye, neck, and spine, 1 each).

Follow-up studies of the 31 patients were possible up to 11 years. The 5 year survival rate was 10 per cent (3 patients). One patient was still alive after 11 years. This patient had had an amputation of the right leg for an osteogenic sarcoma and a lobectomy of the right middle lobe 2½ years later. No further metastatic spread was observed during the period of 11 years.

Operations for metastatic tumors are indicated after the primary tumor had been eradicated and no local recurrence had developed.—*Ernest Kraft, M.D.*

Stoloff, Irwin L. The prognostic value of bronchoscopy in primary lung cancer: a new perspective for an old procedure. *J.A.M.A.*, Jan., 1974, *227*, 229–230. (Address: Thomas Jefferson Medical College, 1025 Walnut Street, Philadelphia, Pa. 19107.)

A retrospective analysis of the 1,038 lung cancers detected with photofluorography by the Philadelphia surveillance unit was made. These tumors were divided into those that involved the stem bronchi, and those that involved the peripheral bronchi.

Of 1,038 primary lung cancers, bronchoscopic results were available for 860 cases. Among these 860 cases, 132 persons or 15 per cent were found to have a tumor in a stem bronchus; 141 (16 per cent) had peripheral bronchial cancers; 251 (29 per cent) had a normal bronchoscopy; and in 341 (40 per cent) some other bronchoscopic findings such as extrinsic compression or secretions were noted.

The 1, 2, and 5 year cumulative survival rates (life table method) of 132 patients with stem bronchial cancers were 12, 3.7, and 1.7 per cent, respectively. These survival rates are less than the survival for those with more peripheral cancers (32, 22, and 14 per cent, respectively).

Eighty-six cases (65 per cent) of the stem carcinomas were considered inoperable. Thoracotomies were performed on the other 46. Twenty-two were exploratory, 19 were palliative resections, and 5 were thought to be curative. Only 1 patient has survived for more than 5 years. His cancer was considered inoperable and he received "curative" irradiation to his primary tumor. Four patients were dead within 1 month of their surgery giving an operative mortality of 9.6 per cent.

The author suggests that due to the relatively high operative mortality and the poor prognoses, patients with invasive carcinoma in the stem bronchi should not be operated on.—*Frederick P. Stitik, M.D.*

Abdomen

Warter, P., Bernhard, J. D., Tongio, J., and Kempf, F. Les aspects radiologiques de l'hématome duodénal. (Radiologic findings in duodenal hematoma.) *J. de radiol., d'électrol. et de méd. nucléaire*, Oct., 1973, *54*, 703–709. (Address: Pr P. Warter, Travail du Service de Radiologie du Pavillon Chirurgical A, C.H.U., F 67000 Strasbourg, France.)

Multiple manifestations of duodenal hematoma can be recognized on barium examination, in addition to the familiar "coil spring" configuration. Seven patients are reported, illustrating a spectrum of the abnormality.

Duodenal hematoma is of traumatic origin in children, and often in adults. Other causes relate usually to pancreatic disease or coagulation disorders.

On the plain film roentgenogram, the right psoas margin may be obliterated due to associated bleeding. The stomach on barium examination is distended, with delayed evacuation.

There is partial or, rarely, complete narrowing of a portion of the duodenal loop. In the latter event, no specific diagnosis of hematoma can usually be made. In the partial form, the narrowing is usually long, segmental, and nonconcentric. It may appear as an external slightly convex impression, or as a filling defect resembling a benign tumor when seen *en face*. There is usually mucosal modification in the narrowed zone, with thickening, thumb printing, palisading, or "coil spring." Two of the cases demonstrated filling of a pseudoaneurysmal pocket communicating with the duodenal lumen in the region of the hematoma.—*Alan E. Oestreich, M.D.*

Schechner, Stephen A., Miller, Ira D., Ehrlich, Frank E., Knapp, Robert W., and Mullen, Joseph T. "Innocent" pneumobilia: report of a case and review of the literature. *A.M.A. Arch. Surg.*, Jan., 1974, *108*, 118–120. (Address: Stephen A. Schechner, M.D., Box 634, US Naval Hospital, Portsmouth, Va. 23708.)

Pneumobilia is an uncommon roentgenologic finding in a patient who has not had previous right upper quadrant abdominal surgery.

The presence of gas in the biliary system usually indicates an abnormal connection between the biliary and gastrointestinal tracts. In almost all cases, surgical intervention will be required to correct the underlying pathologic condition. If cholelithiasis or choledocholithiasis is not found at the operating table, care must be taken to inspect thoroughly the biliary tree, pancreas and gastrointestinal tract for other pathologic conditions.

A 31 year old woman was found to have pneumobilia during evaluation for right upper quadrant abdominal pain. Preoperative studies and surgical exploration failed to uncover any cause for the abnormal gas, and it was still present 1 year postoperatively. Incompetence of the sphincter of Oddi may be the only cause found to explain the pneumobilia. The authors recommend cholecystectomy in such cases

to prevent the subsequent development of ascending cholangitis— a complication resulting from infection in the relatively static pool of gallbladder bile.—*David Morse, M.D.*

POPOVSKY, JULIO, WIENER, STEPHEN N., FELDER, PETER A., BIRAMJE, ASPI, and REYDMAN, MELVIN. Liver trauma: conservative management and the liver scan. *A.M.A. Arch. Surg.*, Feb., 1974, *227*, 753–756. (Address: Julio Popovsky, M.D., 11811 Shaker Boulevard, Cleveland, Ohio 44120.)

Five patients with trauma ranging from blunt (auto accident) to penetrating (gunshot wound) were evaluated by liver scan.

The authors indicate that differentiation between laceration of the liver, intraparenchymal hematoma and subcapsular hematoma was possible. Confirmation by angiography was available in 3 of 5 cases. No normal livers were included.

A conservative (*i.e.*, nonoperative) therapeutic approach was utilized successfully in 4 of 5 patients.

The conclusion is reached that accurate diagnosis and close clinical observation are necessary in the conservative approach and that liver scanning can be helpful in patient evaluation.—*Donald P. Harrington, M.D.*

MACGREGOR, ALEXANDER M. C., and HAWKINS, I. F., JR. Selective pharmacodynamic angiography in the diagnosis of carcinoma of the pancreas. *Surg., Gynec. & Obst.*, Dec., 1973, *137*, 917–921. (From: The Departments of Surgery and Radiology, College of Medicine, University of Florida, Gainesville, Fla.)

Mortality rates for carcinoma of the pancreas have been rising progressively, largely because few patients are referred for surgery while their lesions are still small. By the time pain or jaundice becomes evident, the malignant lesion almost always has become inoperable because of local spread or distant metastases.

Earlier reports of pancreatic angiography were usually retrospective evaluations of celiac artery injections, utilizing some technique other than high volume rapid flow injections of contrast material. This resulted in an estimated preoperative diagnostic accuracy rate of 66 per cent with carcinoma of the pancreas.

In this study, the authors investigated 100 consecutive patients prospectively in terms of ability to catheterize pancreatic vessels: for the comparison of celiac or first order celiac artery branch injections with direct pancreatic artery injections; for the comparison of injections with vasodilators and those without; and for accuracy of roentgenologic diagnosis.

The accuracy of angiographic diagnosis of pancreatic disease was found to increase directly with the degree of visualization of the vessels within the pancreas itself. This, in turn, varied directly with the success in selectively catheterizing pancreatic vessels. Celiac artery stenosis prevented catheterization of either the celiac axis or its branches in 9 per cent of patients. Injections of first order branches, hepatic or splenic arteries, were obtained in 86 per cent of the patients, while placement of the catheter in the gastroduodenal, dorsal pancreatic, great pancreatic artery, or inferior or superior arcade was achieved in 50 per cent.

If direct catheterization of the pancreatic arteries could not be achieved, similar results could be obtained by the use of Priscoline to dilate the pancreatic arterial inflow or epinephrine to reduce the peripancreatic arterial flow, forcing contrast material into the more proximal part of the pancreatic vessels. All of these methods required high volume rapid flow injection of the contrast material.

The diagnostic criteria used were modified from the experience of previous authors. They emphasize the importance of intrapancreatic vessel encasement, arterial occlusion, and lack of increased vascularity in differentiating carcinoma from pancreatitis, in which vascularity is diffusely increased and the vessels, although irregular or beaded, are intact.

Use of this selective pharmacodynamic method resulted in a 92 per cent diagnostic accuracy rate, including 2 patients with 1.5 cm. diameter carcinomas in the head of the pancreas. No false negative diagnoses were made. Two false positive diagnoses were made, 1 in a patient with delayed rupture of the spleen and associated small vessel changes in the tail of the pancreas due to a previous operation, and 1 in a patient with a chronic pericolic abscess in relation to the transverse mesocolon.

In summary, the authors feel that from a surgical standpoint the availability of a technique which offers a 92 per cent diagnostic accuracy rate of an otherwise inaccessible and ill-defined organ is a considerable advance. Combining a zero false negative rate with a false positive rate of 2 to 6 per cent, and an incidence of significant complications of less than 1 per cent warrants additional encouragement of its routine use in the evaluation of carcinoma of the pancreas.

The criterion of utilization should be suspicion of the disease.—*Donald M. Monson, M.D.*

GENITOURINARY SYSTEM

PEETERS, F. L. M. The renal angiogram in tuberculosis. *Radiol. clin. et biol.*, 1973, *42*, 488–499. (Address: Pauluslaan 13, Nijmegen, The Netherlands.)

Hematogeneous dissemination of a tuberculosis infection to the kidneys implies that the renal

parenchyma is the first to be involved by the tubercle bacillus. The initial tissue response is one of proliferation, in which small nodules (tubercles) are formed.

Five stages in the dissemination of renal tuberculosis are postulated. The first stage is cortical tuberculosis, with or without caseation or coalescence of several tubercles into a single large focus. The second stage involves dissemination into the papillae and the minor calyces; this is also ulcero-cavitary tuberculosis. The first and, in particular, the second stage involve bacteriuria, with a grave risk of ureteritis and cystitis. The third and fourth stages are characterized by caseation and fibrosis. The caseation can lead to cavitation; the fibrosis causes strictures, particularly in the tissues surrounding the renal pelvis. Strictures may occur also in the ureter, but more frequently the ureter becomes a rigid, wide tube with a thickened wall. The fifth stage is loss of renal function, be it due to hydronephrosis as a result of obstruction, or as a result of extensive fibrosis with atrophy.

The primary angiographic findings, in order of frequency, were: reduced vascularization of areas involved; irregular contours of the arteries; occlusion of smaller vessels, giving rise to defects in the nephrogram; and signs of an expanding process, *e.g.*, displacement of vessels.

Secondary angiographic findings included: evidence for parenchymal scarring and retraction, with vascular occlusion, suggestive of infarction; filling of the capsular artery which denotes the original size of the kidney in cases where there has been complete destruction of the renal parenchyma; and dilatation of tortuous ureteral arteries. The latter, however, is a very nonspecific finding, occurring with various ureteral inflammatory processes, benign and malignant tumors, and reduced blood flow in the renal artery or the aorta.

On a theoretic basis the renal angiogram should be able to detect small cortical tuberculous lesions before the excretory urogram becomes positive. On a practical basis patients either do not have angiograms during this first stage, or the small vessel detail is such that these early lesions cannot be detected on standard angiograms. Magnification techniques are suggested for further evaluation.

For the time being the value of angiography in the diagnosis of renal tuberculosis does not seem to lie in the possibility of diagnosing this disease as such in an earlier stage. The principal merit of angiography is that it can help to establish the possible cause of renal nonfunction and eliminate the possibility of a malignant tumor.—*Donald M. Monson, M.D.*

Jackman, Roger J., and Stevens, G. Melvin. Benign hemorrhagic renal cyst: nephrotomography, renal arteriography, and cyst puncture. *Radiology*, Jan., 1974, *110*, 7–13. (Address: Roger J. Jackman, M.D., Palo Alto Medical Clinic, 300 Homer Avenue, Palo Alto, Calif. 94301.)

Six case reports of benign hemorrhagic renal cysts are presented.

Benign hemorrhagic renal cyst is defined in this study as a cyst containing degenerating hemorrhagic material without evidence of malignancy. Nephrotomography (5 cases) and renal angiography (3 cases) showed findings consistent with a renal cyst. In each of the 6 cases at cyst puncture there was hemorrhagic aspirate containing degenerating hemorrhagic material. None had positive cytology and 1 had fat present. The cyst cavity was irregular in 3 and smooth in 3. In 2 of the cases with irregular cyst cavities the entire mass seen at nephrotomography could not be accounted for. Surgical confirmation of benign hemorrhagic cyst was obtained in 2 patients with irregular cyst cavities and at autopsy in the other patient with an irregular cyst cavity. The other 3 patients are being followed with a presumptive diagnosis of benign hemorrhagic cyst.

In 13 other cases of cyst puncture there was hemorrhagic aspirate. In all of these only fresh blood was present, usually a small amount at the beginning of the aspiration. In none were there degenerating erythrocytes, epithelial cells, or malignant cells. These were all felt to be secondary to traumatic cyst punctures and no further studies were done.

The following recommendations are therefore made. In the presence of hemorrhagic aspirate at cyst puncture, traumatic causes can be eliminated by gross inspection and cytology. In those having hemorrhagic aspirate with tumor vascularity on the arteriogram or positive cytologies, surgery is recommended. In the patient having a degenerating hemorrhagic aspirate with a typical cyst pattern at nephrotomography and angiography as well as a smooth cyst cavity and the entire mass seen by nephrotomography accounted for, a presumptive diagnosis of benign hemorrhagic cyst can be made. These patients may be followed. In those patients with a similar combination of findings, but having an irregular cyst cavity or being unable to account for the entire renal mass, exploration is recommended. Gross inspection as well as biopsy should be done. In these patients nephrectomy can thus be avoided.—*A. K. Teaford, M.D.*

Pollack, Howard M., and Popky, George L. Roentgenographic manifestations of spontaneous renal hemorrhage. *Radiology*, Jan., 1974, *110*, 1–6. (Address: Howard M. Pollack, M.D., Department of Radiology, Episcopal Hospital, Front Street and Lehigh Avenue, Philadelphia, Pa. 19125.)

Although "spontaneous" renal bleeding (bleeding without a history of trauma) usually manifests itself as hematuria, there are occasions when some or all of

the blood does not communicate with the collecting system. Under such circumstances, and especially if the bleeding is arterial, a hematoma may develop.

Twenty-two cases were analyzed for radiographic signs and underlying etiologies. Ten tumors were included and 5 cases were due to bleeding diathesis.

Intrarenal hematomas exhibit the urographic characteristics of a radiolucent renal mass without neovascularity at angiography. Complete resorption in the uncomplicated case should occur in several weeks to months. Incomplete resorption should raise the possibility of tumor. One case each of renal infarction and bleeding diathesis caused the intrarenal hematomas in this series.

When the source of bleeding is close to the renal capsule, the hematoma may dissect the capsule away from the renal parenchyma. This form of bleeding is usually self-limited due to tamponade. Enlargement of the renal outline may be the only urographic sign. Flattening, usually involving the outer border, is characteristic at arteriography or nephrotomography. Elevation of the renal capsule may be seen at angiography. Five of the 9 cases of subcapsular hematomas were associated with neoplasms. Only 1 neoplasm was seen at arteriography. Four radiographically undetected lesions ranged in size from 5 to 10 mm. in diameter. Bleeding diathesis, renal infarction and 1 case of unknown etiology made up the 4 remaining cases. Although surgery is usually employed in these cases, the authors recommend meticulous inspection for tumor because of the high incidence of occult neoplasms at arteriography in their series.

When the hematoma breaks through the renal capsule, it gains access to the perirenal space. This sequence is frequently seen with trauma, but less commonly in spontaneous hematoma. A large soft tissue mass is seen in the abdomen, and the outline of the kidney and upper psoas margins are obliterated. The kidney may be displaced and may show impairment of renal function. Two neoplasms and 3 cases of drug polyarteritis comprised this group. Arteriography was not performed in the cases of neoplasms.

Three tumors, 2 cases of bleeding diathesis, and 1 case of polyarteritis presented as combined patterns of hematomas.—*Jon F. Bergstrom, M.D.*

JACONETTE, J. RICHARD, MCGINNIS, KENNETH D., and ZESKIND, H. JAY. Pericapsular renal veins: recognition without angiography. *Radiology*, Feb., 1974, *110*, 283–287. (Address: J. Richard Jaconette, M.D., Department of Radiology, Mount Carmel Mercy Hospital, Detroit, Mich. 48235.)

Dilated pericapsular renal veins occur with an incidence of 57 per cent in renal cell carcinoma. A weighted retrospective study done by the authors using 47 cases of renal cell carcinoma, 47 cases of renal cysts, 47 cases of pyelonephritis, and additional cases of miscellaneous neoplastic disease and cases of tumor simulating conditions such as hydronephrosis, renal carbuncle and perinephric abscess documents the high incidence of dilated pericapsular renal veins. The conclusion from the study was that these dilated veins occur with great frequency in renal cell carcinomas, but rarely in benign disease.

The dilated pericapsular renal veins present as irregular serpiginous soft tissue density shadows lying in the perirenal fat. The veins may lie parallel to the margin of the kidney or may lie in the fat adjacent to the associated renal mass. The authors recommend the use of high dose urography to better delineate these abnormal veins. Tomography was used as an adjunct in the study of a number of cases. Generally, there was better demonstration of the dilated pericapsular veins with contrast material.

The demonstration of collateral pericapsular veins has been described previously in renal cell carcinoma with involvement of the renal vein by thrombus or neoplasm. The authors indicate that abnormal pericapsular veins are seen more frequently in renal cell carcinoma when the vein is involved. However, they found abnormal pericapsular veins in 7 of the cases without vein invasion and in 6 cases of benign disease. Because of these 13 cases without renal vein involvement, the authors concluded that renal vein involvement is not necessary to produce the dilatation of pericapsular renal veins. Dilatation may be simply the result of hypervascularity.

In summary, distended pericapsular veins are found rarely in renal cysts. The presence of pericapsular veins is supportive evidence of renal cell carcinoma, although a suppurative process should also be considered. Abnormal dilated pericapsular veins do not necessarily suggest renal vein compression or invasion by tumor or thrombus.—*J. P. Eberts, M.D.*

LAURENT, Y. Les diverticules multiples ou diverticulose de l'uretère; un syndrome radioclinique rare. (Multiple diverticula or diverticulosis of the ureter; a rare radio-clinical syndrome.) *J. belge de radiol.*, Sept.–Oct., 1973, *56*, 383–392. (Address: Chef de Service à la Clinique César de Paepe, Maître de Stage à l'Université Libre de Bruxelles, 186, avenue de Messidor, B-1180 Bruxelles, Belgium.)

Several small diverticula of one or both ureters have rarely been noted in patients with urinary tract infection or enlarged prostate. All previously reported examples were found on retrograde pyelography except for 1 case on intravenous urography. Usually the ureter is of normal over-all caliber.

Two patients, men about 50 years old, were found on intravenous urography to have a few very small diverticula in the lumbar portion of the right ureter.

In the first case, who had urinary tract infection, the diverticula were noted only while external compression was applied to the ureters. The other patient had a prostatic adenoma; his diverticula were seen on a perfusion intravenous urogram with no external compression.

Such miniscule diverticula are possibly overlooked even when present. The etiology remains unproven. —*Alan E. Oestreich, M.D.*

Catalano, Dino. Controllo farmacoangiografico selettivo di ematuria post-traumatica. (Selective pharmaco-angiographic control of post-traumatic hematuria.) *Radiol. med.*, Sept., 1973, *59*, 800–802. (Address: Ospedale S. Gennaro, 25 via S. Gennaro dei Poveri, I-80136 Napoli, Italy.)

Well known is the value of selective angiography in the demonstration of hemorrhages from various internal organs. Recently, this procedure has found a new important role in the control of the hemorrhage by selective vasoconstrictor infusion into the bleeding artery. The author has obtained complete control of a renal hemorrhage by selective pharmacoangiography.

A 37 year old man was admitted to the Division of Urology because of hematuria following severe trauma. After the first emergency therapeutic procedures, it was decided to perform selective angiography of the right kidney, where the symptomatology indicated the lesion to be present. This study revealed no gross distortion of the renal parenchyma, but showed rupture of at least 3 segmental intraparenchymal arteries with extravasation. The kidney was nonfunctioning. Because of the extent and type of the pathologic changes, it was decided to postpone the surgical intervention and to try selective vasoconstrictor infusion through the catheter left *in situ*. A 1 to 1,000 solution of adrenalin was used and 500 μg. was introduced. At the end of the infusion, only the main renal arteries were opacified by the contrast medium. Because of the satisfactory result, a second infusion of adrenalin in the same amount and solution was carried out. An angiogram performed 30 minutes after this second infusion demonstrated a normal opacification of the intrarenal circulation and no evidence of extravasation. The catheter was then withdrawn and the patient kept under strict observation.

Multiple vesical washings resulted in the elimination of the vesical blood clots, and 24 hours later there was no evidence of hematuria.

Excretory urography performed 2 days after admission showed normal excretion and concentration bilaterally, without evidence of gross pathologic changes in the morphology of the collecting system of the right kidney. This return to normal function and morphology has persisted in several control studies at longer periods of time.—*A. F. Govoni, M.D.*

Elkin, Milton, Goldman, Stanford M., and Meng, Chien-Hsing. Ureteral obstruction in patients with uterine prolapse. *Radiology*, Feb., 1974, *110*, 289–294. (Address: Milton Elkin, M.D., Department of Radiology, Albert Einstein College of Medicine, Bronx, N. Y. 10461.)

Procidentia as a cause of upper urinary tract dilatation in elderly patients is discussed by the authors using 19 separate cases. Intravenous urography, including study in the prone position, was performed on all patients, with erect views obtained when possible. All but 1 of the 19 patients had third degree prolapse of the uterus. The 1 patient without prolapse was 30 years post hysterectomy; however, she had severe vaginal prolapse and a large cystocele. The average age was 72 years.

All cases showed some degree of upper urinary tract dilatation. The erect position studies demonstrated the prolapse of the bladder with caudal displacement of the trigone the best. One patient who was 3 years and 3 months post LaFort's colpocleisis demonstrated less dilatation on a follow-up study.

Since 74 per cent demonstrated urinary tract infection, excretory urograms are recommended in any patient with procidentia. If significant dilatation is found, the authors recommend corrective surgery. The caudal displacement of the trigone with resultant mechanical deformity of the meatus is postulated by the authors as the cause of the low ureteral obstruction.—*Robert F. Kuhnhein, M.D.*

Skeletal System

Govoni, Antonio F. Osteofiti della colonna cervicalle e disfagia. (Osteophytes of the cervical spine and dysphagia.) *Radiol. med.*, Sept., 1973, *59*, 769–780. (Address: Queens Hospital Center, 82–68 164th Street, Jamaica, N. Y. 11432.)

Hypertrophic spondylosis of the cervical spine and the frequent association with neurologic symptomatology, related to the pathologic changes in the cervical vertebrae involved, are well known. Rather infrequent are, however, the reports on dysphagia produced by spondylosis deformans involving the cervical vertebrae.

The author's report deals with 12 cases in which osteophytes of the anterior margins of 2 or more vertebrae resulted in dysphagia by direct pressure on the cervical esophagus.

Reviewed are the most accepted theories on the development of these osteophytes and analyzed is the anatomic relationship between the esophagus and the cervical spine, which would explain the symptomatology presented by the patients.

The decades most frequently affected, as it appears from an analysis of the 70 cases previously reported, and of the author's cases, are the sixth, seventh and fifth. The vertebral bodies mostly involved are C6–C7, C5–C6 and C4–C5. There is a greater incidence in the male sex.

The treatment is palliative and only in 15 cases was the symptomatology such as to require the surgical removal of the osteophytes.

Numerous illustrations demonstrate the roentgen findings presented by the patients, whose histories are reported in detail.—*E. Frederick Lang, M.D.*

WILLICH, VON E., and ENGLERT, M. Das Metakarpalzeichen. (The metacarpal sign.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Oct., 1973, *119*, 443–450. (Address: Priv.-Doz. Dr. E. von Willich, Universitäts-Kinderklinik, Röntgenabteilung, Hofmeisterweg 1–9, 6900 Heidelberg 1, Germany.)

The metacarpal sign relates to the length of the fourth metacarpal bone. The relative length is determined by a line drawn tangentially to the fourth and fifth metacarpal heads. When this line passes through the third metacarpal head, the metacarpal sign proves positive, as the fourth metacarpal bone is now considered "short." A borderline sign exists, when the tangential line also forms a tangent to the third metacarpal head, while normally the line bypasses this bone distally. Short fourth metacarpal bones are usually encountered in cases of gonadal dysgenesis.

The metacarpal sign was tested in the left hand of 1,000 children who had been hospitalized for various disorders. There were 558 boys and 442 girls, ranging in age from 3 days to 17 years. The average age was 5 years and 9 months. The metacarpal sign proved positive in 60 boys and 45 girls (10.4 per cent) including 37 children with borderline findings. The positive sign was noted to increase in frequency with advancing age.

An abnormal maturation could not be related to the metacarpal sign as 39 children presented with a retarded growth and 8 with an accelerated development. The most common clinical findings in this group included brain damage, obesity, retarded growth, and convulsive disorders, while endocrine diseases could not be observed.

In a separate group of 14 girls with gonadal dysgenesis the metacarpal sign could only be found twice. This inconclusive result could be attributed to the early age of the patients as the positive metacarpal sign was reported much more frequently in adolescents. Therefore, it is believed that it could develop at or after puberty.

Since the metacarpal sign was mostly noted in the general population, it could be attributed to a normal variant of a familial trait.—*Ernest Kraft, M.D.*

RADIATION THERAPY

WELLWOOD, J. M., and JACKSON, B. T. The intestinal complications of radiotherapy. *Brit. J. Surg.*, Oct., 1973, *60*, 814–818. (From: St. Thomas's Hospital, London, England.)

The authors reviewed 38 patients seen with intestinal complications following irradiation of the abdomen at the St. Thomas Hospital. All but 1 patient had malignant disease, and most patients had a pelvic neoplasm.

Proctocolitis, rectal ulcers, steatorrhea, large and small bowel strictures, perforations, and fistulas were noted. Many of these complications occurred together, and one complication often progressed to another. Colitis, ulcers, and steatorrhea were usually early changes compared with strictures. Perforation, fistulas, and anal strictures were seen as late complications.

The progressive nature of these complications is stressed, *i.e.*, proctocolitis preceded the development of colorectal strictures by up to 4½ years.

Clinically, abdominal pain usually indicated stricture, and pain on defecation usually indicated rectal ulcer or stricture. Patients with diarrhea had malabsorption of fat, vitamin B_{12}, and calcium.

The histologic changes in the operative specimens all showed findings consistent with the post radiation changes described in the literature. In addition, the authors noted an increase in the submucosal fibrous tissue, well developed arterial sclerosis, and marked lymphatic ectasia.

The radiologic findings included mucosal abnormalities, strictures, and nodular filling defects. Some of the strictures had many features to suggest malignancy, and others simulated the findings in the terminal ileum seen in regional enteritis. The nodular filling defects were very difficult to distinguish from metastases. Mucosal folds were found to be thickened and there was dilution of the barium in some cases.

The authors found that late intestinal complications followed irradiation from a few months to many years. In the past, radiation dose, pelvic adhesions, and vascular changes have been considered in the etiology of post radiation changes.

In this series vascular changes seemed to fit with the time period involved in late complications, and explained, in part, the group of patients who developed bowel ischemia many years following irradiation.

Complications involving bowel-to-bowel anastomosis were very high, and the authors suggest avoiding primary anastomosis.

Post radiation changes led to death in 14 patients (37 per cent), and 9 of these patients had no evidence of recurrent tumor at postmortem examination.

The early diagnosis of these complications involved recognizing the clinical signs of diarrhea and colic.

The roentgenologic diagnosis was confusing because the changes mimicked other diseases. In some cases nodular filling defects very closely resembled metastatic nodules.—*Daniel P. Link, M.D.*

Huguier, M., Malafosse, M., Malta-Bey, G., and Loygue, J. Le traitement chirurgical de la maladie de Crohn: revue de 120 cas. (Surgical treatment of Crohn's disease: a review of 120 cases.) *Presse méd.*, 1973, *2*, 1931–1934. (Address: M. Huguier, Hôpital Rothschild, 43, Bd. de Picpus, F 75012, Paris, France.)

The authors present a review of 120 cases operated on between 1953 and 1970. Clinically, the patients presented one or a combination of the following symptoms: abdominal pain (108 cases), sometimes of obstructive type (22 cases); diarrhea (108 cases), sometimes profuse (12 cases) or accompanied by hemorrhage (18 cases); and loss of weight (96 cases). Other less prevalent symptoms included arthralgia (17 cases), ocular signs (7 cases), or cutaneous signs (2 cases).

The disease involved the terminal ileum in 42 cases; the ileum and the right colon in 54 cases (in 31 of these the whole colon was diseased); the left colon in 14 cases; and various other sites in 10 cases (duodenum in 3 cases, widespread jejuno-ileum in 7 cases).

A single or repeated intestinal resection was performed on 111 patients, among these 11 cases consulted for a recurrence following a previous resection performed elsewhere. Among the first group of 100 patients, recurrences were observed 19 times, and false recurrences, attributable to pathologic tissue left in place during the first intervention, were encountered 11 times. Among the second group of 11 patients, recurrences were noted 9 times and false recurrences twice. In more than half of these cases, the disease recurred within a 2 year period following resection. Late recurrences were rare in this series (3 cases out of 23 cases followed for more than 5 years).

The cases afflicted by a recurrence were submitted to medical or surgical treatment. Two subsequent reinterventions were performed in 7 cases, and a third was necessary for 1 of them.

Of their total of 111 cases, the authors reported 3 deaths, which is a low rate compared to the rate of 14 per cent obtained in their surgically treated group of ulcerative colitis.—*H. P. Lévesque, M.D.*

Martini, Nael, Khafagy, Medhad M., Melamed, Myron R., Golbey, Robert B., and Beattie, Edward J., Jr. Bronchiolar carcinoma: a review of 152 patients. *Clin. Bull.*, 1973, *3*, 98–101. (From: Memorial Sloan-Kettering Cancer Center, New York, N. Y. 10021.)

From 1961 to 1968, 1,645 cases of primary carcinoma of the lung were treated at Memorial Sloan-Kettering Cancer Center. Carcinomas arising from the broncho-alveolar tree and having a glandular growth pattern were classified as adenocarcinomas or broncho-alveolar carcinomas from their growth pattern and the amount of tissue destruction. Broncho-alveolar carcinomas were more peripheral and progressed multifocally along alveolar septa with minimal tissue destruction. Adenocarcinomas were more proximal and usually progressed in large single masses with tissue destruction. There were 152 broncho-alveolar carcinomas (9 per cent of the 1,645). Male to female ratio was approximately 2:1; 7 per cent of the males and 48 per cent of the females were nonsmokers. At detection, 32 per cent had no symptoms, 50 per cent had symptoms less than 3 months in duration, and the remainder had symptoms for periods longer than 3 months.

The radiologic findings included: mass density; parenchymal infiltrate; and pleural effusion. One hundred patients had a mass density (single in 91, multiple in 9); 15 patients had infiltrates simulating pneumonia, but only 1 patient was febrile; 37 patients had pleural effusion, and in 16 of these, it was the only radiologic finding. Thoracentesis in 29 patients showed malignant cytology in all cases. Carcinomas were on the right in 88 patients and on the left in 51 patients. Bilateral lesions were found in 13 patients. Most of the tumor masses were small, and only 5 were greater than 6 cm. in diameter.

Diagnosis was established by sputum cytology (20 per cent), pleural cytology (20 per cent), biopsy of metastatic lesions (20 per cent), and thoracotomy in the remainder.

Treatment regimens included: surgery; external irradiation; and chemotherapy. Sixty-eight resectable tumors were found out of 89 patients who were treated, primarily by surgery. Regional lymph node dissection was carried out in 12 patients in addition to the local resection. There were 6 postoperative complications and 4 deaths. Fifty-five patients received external irradiation. A tumor dose of less than 4,000 rads was given to half of the patients; the other half received more than 4,000 rads. Systemic chemotherapy was given only in the presence of pleural effusion or distant metastases. Prolonged pleural effusions were treated with one or more of the following: radioactive phosphorus; chemotherapy; external irradiation and pleurectomy.

Survival of the 152 patients was 18 per cent at 3 years and 11 per cent at 5 years. All of the long-term survivors had resectable lesions and were treated with surgery alone. Patients on other treatment regimens had a much more rapid course, and none of the patients who presented with pleural effusion survived longer than 23 months. Twenty-three per cent of the patients, who were detected before symptoms, survived 5 years or longer. Postmortem studies in 57 patients revealed generalized metastatic disease

in 36 and single organ metastases in 16 (in order of frequency: brain, liver and bone).

Survival from broncho-alveolar carcinoma was shown to be better than for primary lung cancer in general. Disease with regional lymph node involvement was tolerated better than disease with pleural involvement. Long-term survival only occurred with resectable lesions which were treated by surgery alone.

The authors believe that the spectrum of clinical and radiologic features in their cases classified as broncho-alveolar carcinoma, histologically, may reflect a group of neoplasms rather than a single entity. —*Daniel P. Link, M.D.*

TABÁR, L., KÁDAS, I., MÁRTON, Z., NÉMETH, Á., and KOSARAS, B. The significance of mammography, galactography, and pneumocystography in detecting occult carcinomas of the breast. *Surg., Gynec. & Obst.*, Dec., 1973, *137*, 965–970. (From: The Radiological Clinic, Medical University, The Departments of Pathology and Surgery, Baranya County Hospital, Pathological Institute, and Institute of Anatomy, Histology, and Embryology, Medical University, Pécs, Hungary.)

Most breast lesions are discovered by the patient and about 65 per cent of these have axillary metastases at the time of discovery. Disease without metastases is considered to be early in its course. Mammography can reveal disease before there are clinical findings in many cases. In the proper clinical setting, pneumocystography and galactography may also aid in the detection of early lesions. From May 1969 to November 1972, 1,710 patients were seen. There were 1,920 mammographies, 56 galactographies, and 59 pneumocystographies; 258 malignant and 420 benign lesions were detected, including 22 malignant lesions which were not detectable clinically. Mammography also proved useful in detecting a clinically occult primary lesion in the presence of metastases.

In the patients with an abnormal nipple discharge, galactography showed early interductal lesions which were not apparent clinically and could not be demonstrated by mammography. Cystic lesions may contain papillary carcinomas in the wall which can be found on pneumocystograms.

The authors stress close cooperation by the surgeon, pathologist and radiologist. They believe that by utilizing mammography along with galactography and pneumocystography, the mortality for carcinoma of the breast can be reduced.—*Daniel P. Link, M.D.*

HOROWITZ, ALFRED L., RESNICK, DONALD, and WATSON, ROBIN CAIRD. The roentgen features of synovial sarcomas. *Clin. Radiol.*, Oct., 1973, *24*, 481–484. (From: Department of Diagnostic Radiology, Memorial Hospital for Cancer and Allied Diseases, 424 East 68th Street, New York, N. Y. 10021.)

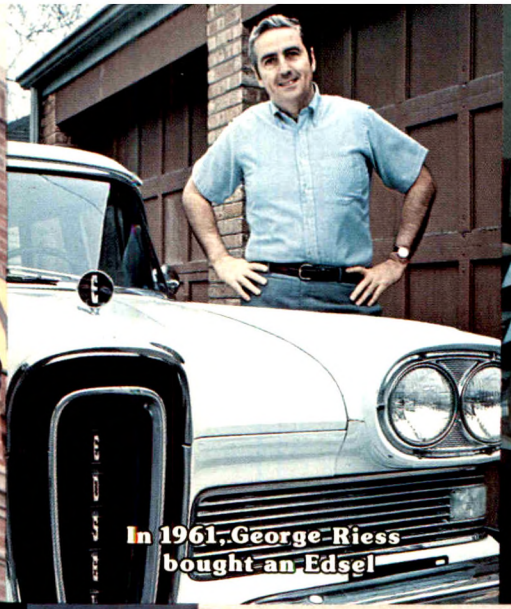
Reviewing the experience at Memorial Hospital between 1966 and 1970, 45 proven cases of synovial sarcomas are recorded. Only 15 patients had roentgenograms available for review and formed the basis for this radiologic report.

Apparently all 15 of the cases had some roentgenologic abnormality. In 10 of the cases a soft tissue mass was visible, having no specific diagnostic features. Three of the tumors showed soft tissue calcification—an incidence of 20 per cent. This compares with 32 per cent previously published in a larger series by Cadman. Three cases showed bone abnormality and in this series and others it generally took the form of erosive changes due to pressure by the adjacent mass. Actual bone invasion, however, can occur with localized areas of rarefaction, showing little active sclerosis unless treatment has already been given.

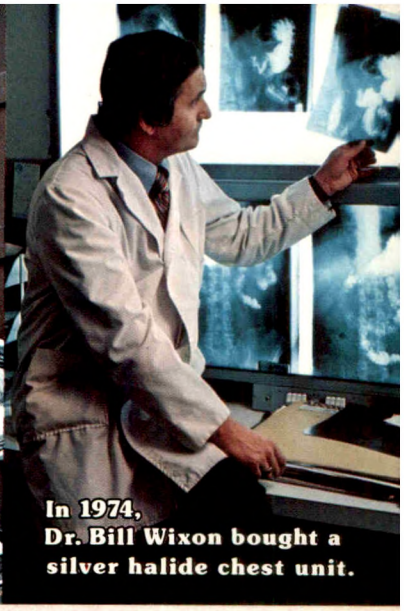
It is emphasized that synovial sarcomas are uncommon malignancies. They need not arise within a joint capsule, but may appear along the tendon sheath anywhere in an extremity. There is a predilection for lower extremity involvement. Although the average age of the patients in this series is 34, the ages in fact range from 9 to 64 years.—*R. H. Troupin, M.D.*



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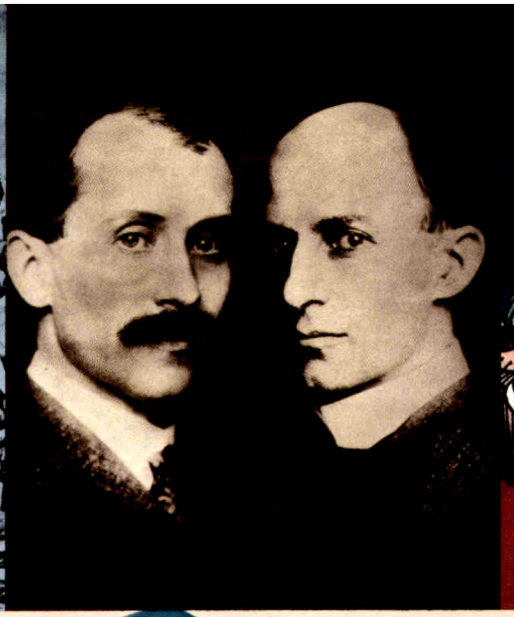
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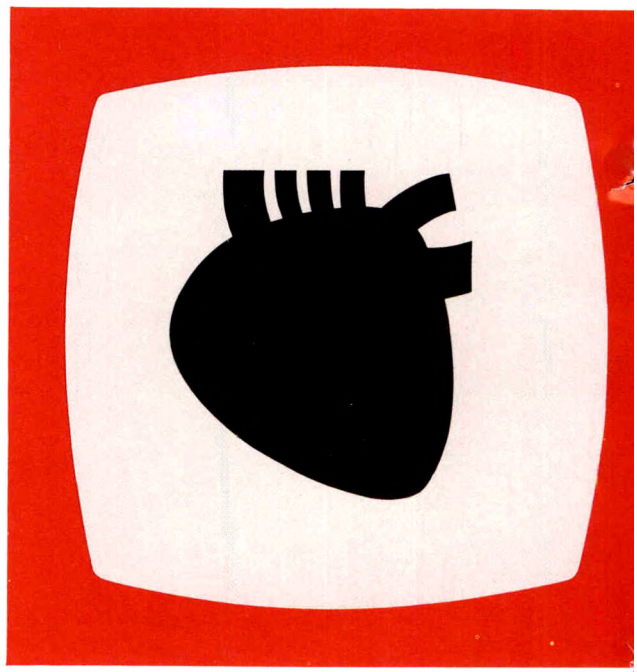
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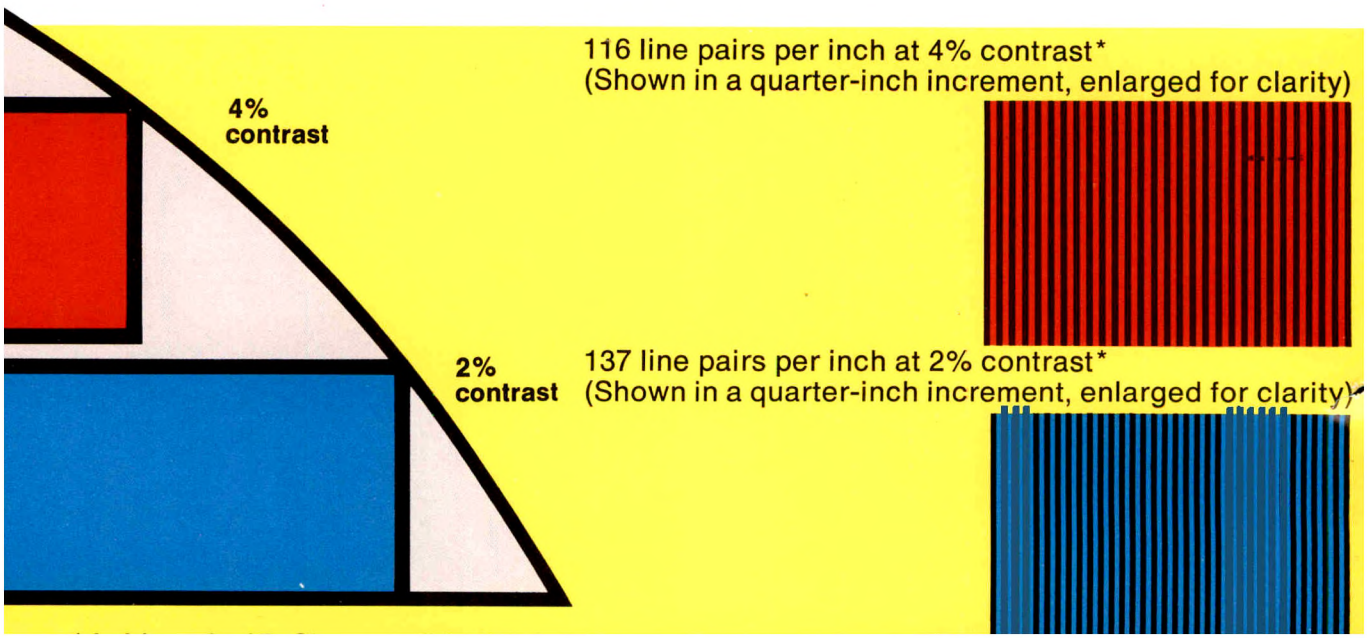
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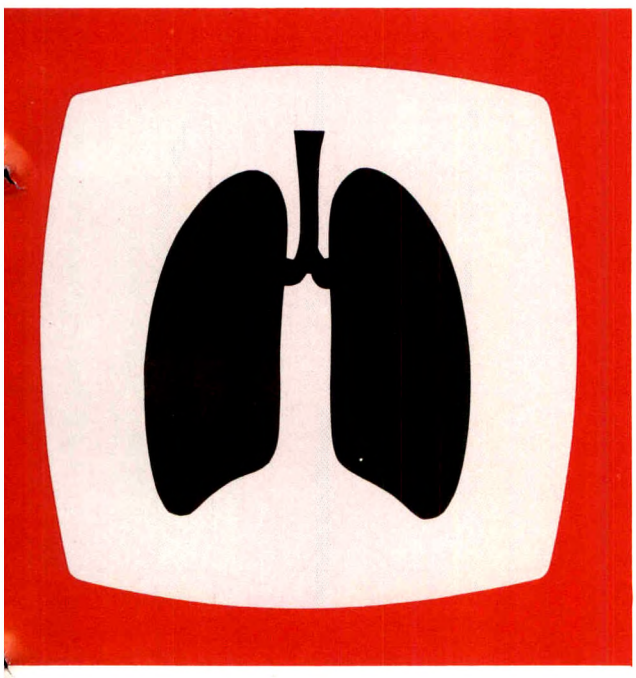
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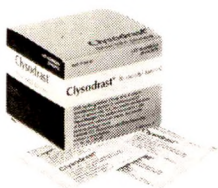
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MODERN THIN-SECTION TOMOGRAPHY. *Edited by* **Arnold Berrett,** *New York Medical College, New York City;* **Sam Brunner,** *Gentofte Hospital, Copenhagen, Denmark; and* **Galdino E. Valvassori,** *Univ. of Illinois, Chicago. Foreword by* **Solve Welin.** *(21 Contributors)* Information includes the history of tomography, the physical principles involved, and descriptions of many of the machines employed. The following chapters deal with tomographic anatomy, technical factors and a description of the clinical applications of the method in various parts of the body. *'73, 352 pp. (7 x 10), 496 il., 17 tables, $26.50*

THE BIOLOGICAL AND CLINICAL BASIS OF RADIOSENSITIVITY. *Edited by* **Milton Friedman,** *Instituto Regina Elena, Rome, Italy. (35 Contributors)* Integrated in every radiobiological experiment and in the treatment of every tumor is the concept of radiosensitivity. Outstanding authorities in the fields of radiobiology, radiation pathology, preclinical radiobiology and experimental clinical radiotherapy explain their specialized investigations in terms of radiosensitivity and chemotherapy. In this manner the book fills the gap in information about radiosensitivity between the laboratory and clinical schools by clarifying and consolidating pertinent material, defining the unproven, and eliminating the irrelevant. *'74, about 589 pp. (7 x 10), 373 il., 67 tables*

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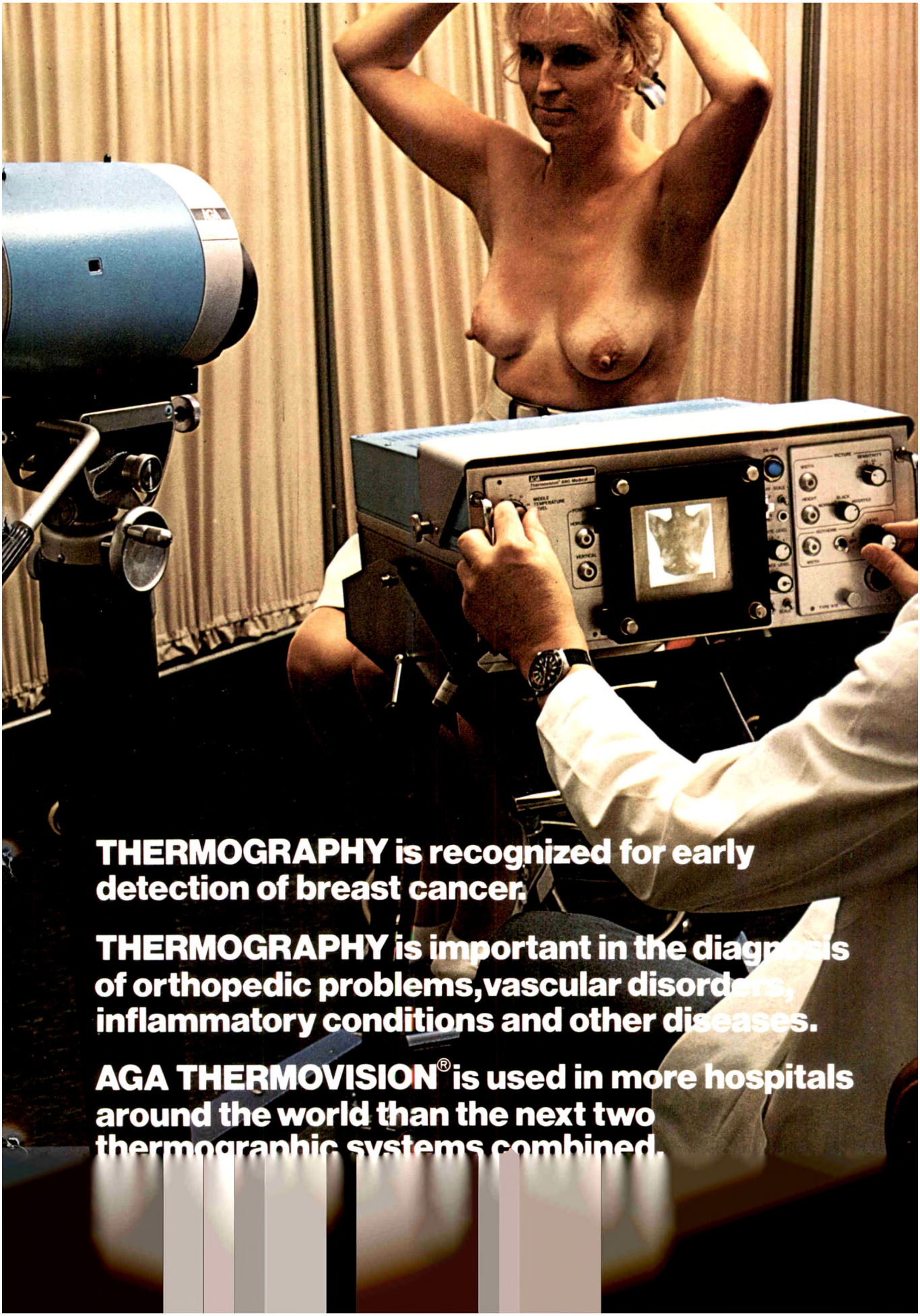
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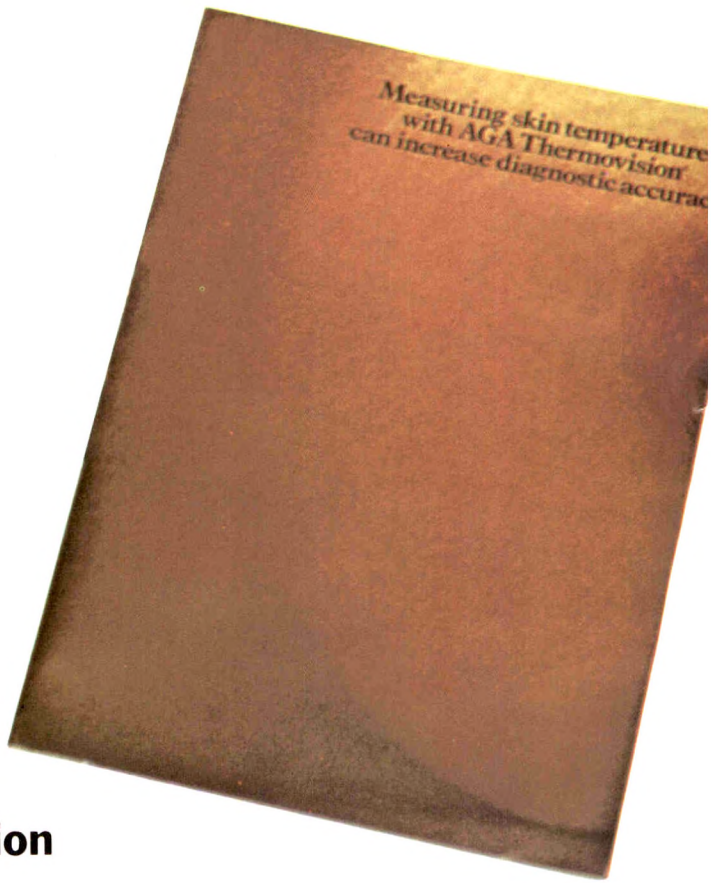
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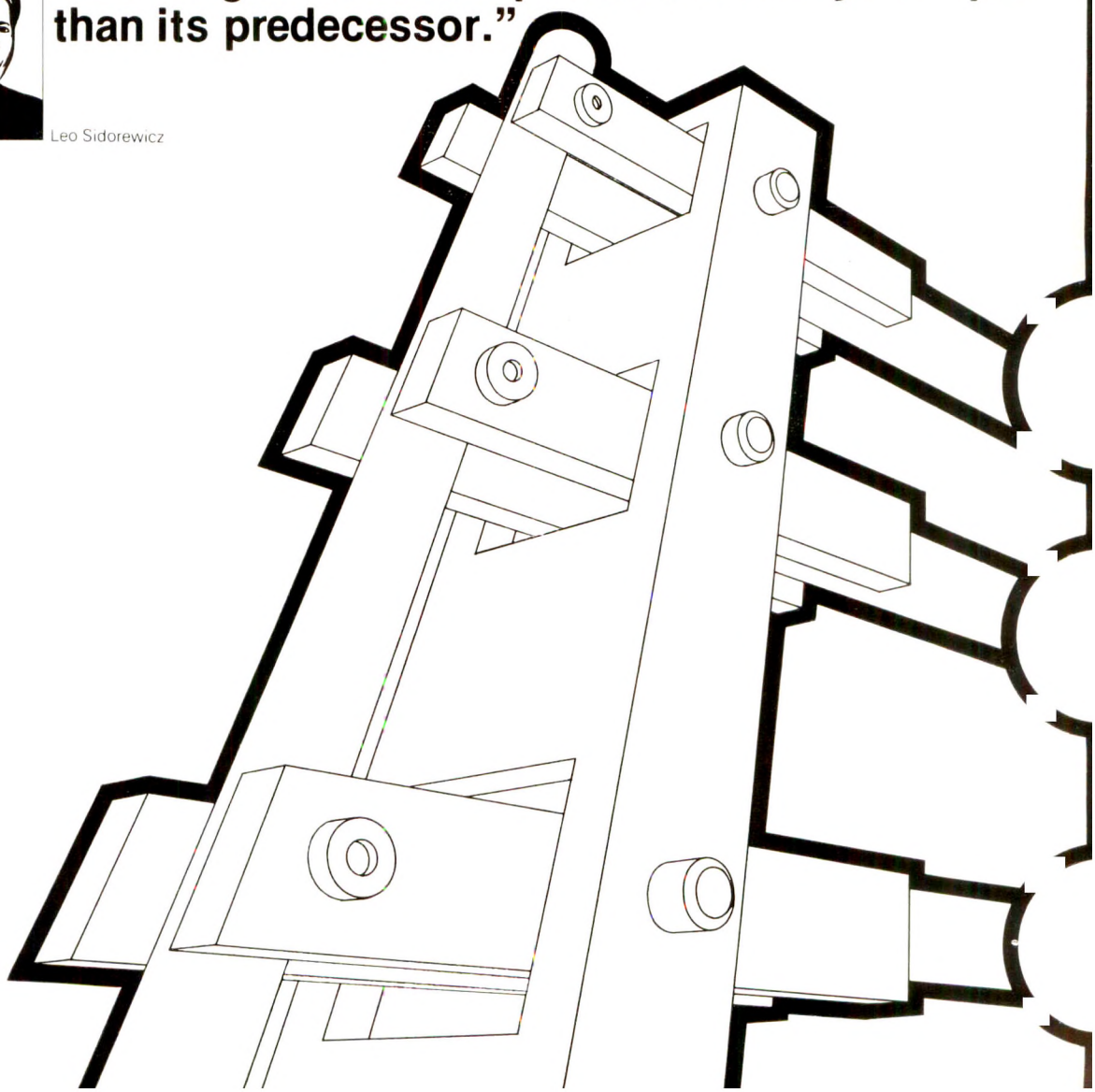
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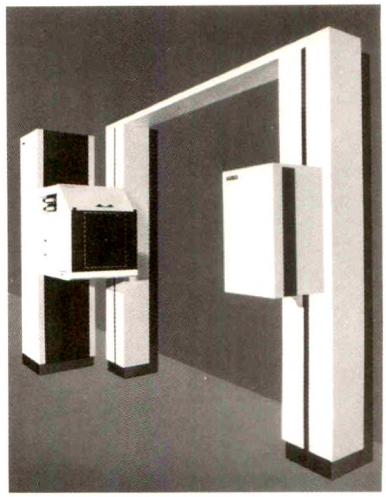
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Description: Hypaque-Cysto is a radiopaque medium which contains approximately 141 mg. organically-bound iodine per ml. It is a 30 per cent (weight/volume) sterile aqueous solution of the meglumine salt of 3,5-diacetamido-2,4,6-triiodobenzoic acid ($C_{18}H_{26}I_3N_3O_9$). The viscosity is 1.94 cps at 25° C. and 1.42 cps at 37° C. The pH is adjusted with hydrochloric acid or meglumine solution. Hypaque-Cysto is clear and colorless to pale yellow. It should be protected from strong light. The solution is relatively thermostable and may be autoclaved. Calcium disodium edetate 1:10,000 has been added as a sequestering stabilizing agent.

Action: Hypaque-Cysto is a diagnostic radiopaque medium which provides visualization of the lower urinary tract.

Indication: Hypaque-Cysto is indicated for retrograde cystourethrography.

Adverse Reactions: No irritation or other undesirable effects have been reported following the administration of Hypaque-Cysto in clinical investigations.

Dosage and Administration: After the bladder is emptied, Hypaque-Cysto is gently instilled without force, often beyond the first desire to micturate, but not beyond the point of urgency or mild discomfort. The volume required to fill the bladder to slightly less than capacity may vary from patient to patient.

Bladder capacity in normal adults is generally 200 to 300 ml., and rarely, up to 600 ml. Capacity at birth is 20 to 50 ml., and increases about 400 per cent in the first year. In children 3 to 5 years old, bladder capacity is 150 to 180 ml. In children older than 8 years, it is in the low adult range.

In disease, bladder capacity in adults may vary from 50 ml. in neurogenic "reflux" bladder to over 1000 ml. in autonomous bladder or chronic lower urinary tract obstruction.

Repeat examination may be required to detect reflux, or in function studies.

The concentration varies with technique and equipment used. Hypaque-Cysto may be diluted with sterile water or 5 per cent dextrose solution, as indicated in the following table.

To Make		Add	Final Solution Contains
Final conc.	Final volume	Sterile water or 5% dextrose solution	Iodine
30 %	250 ml.	—	141 mg./ml.
25 %	300 ml.	50 ml.	118 mg./ml.
21.4%	350 ml.	100 ml.	101 mg./ml.
20 %	375 ml.	125 ml.	94 mg./ml.
18.8%	400 ml.	150 ml.	88 mg./ml.
16.7%	450 ml.	200 ml.	78 mg./ml.
15 %	500 ml.	250 ml.	71 mg./ml.

How Supplied: Calibrated 500 ml. dilution bottles containing 250 ml. Hypaque-Cysto; rubber stoppered, with inner removable seal and screw neck.



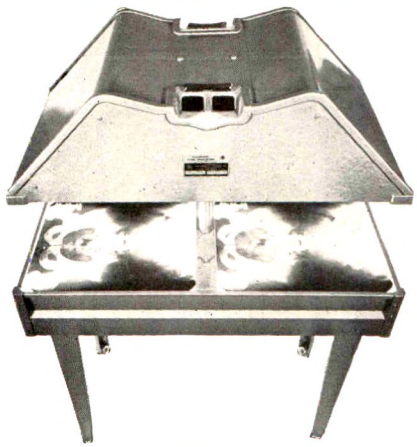
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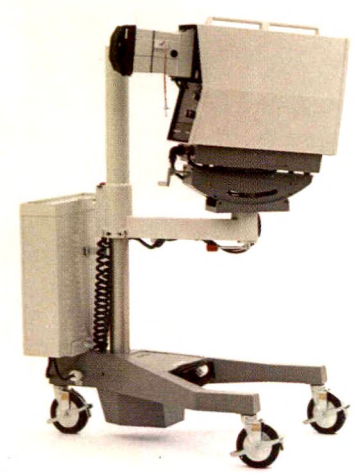
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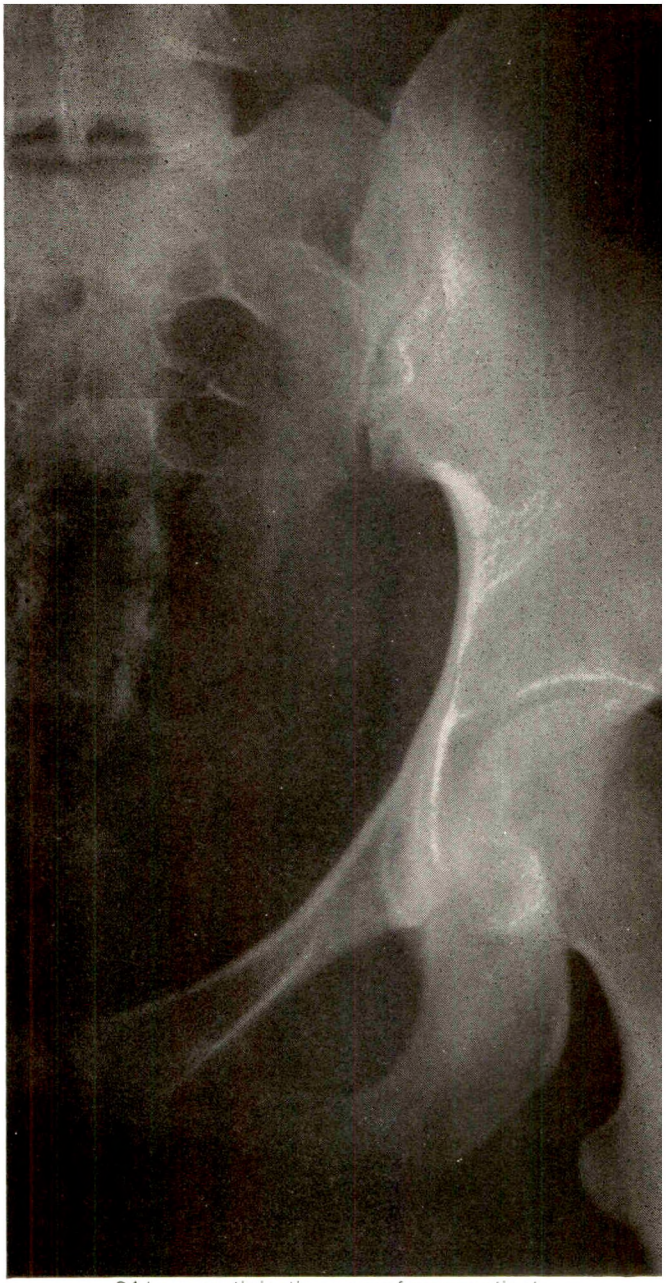
In recent years, mounting evidence has shown that successful radiotherapy is dependent on the accuracy and precision with which the radiation is delivered to the patient. If unusual or difficult treatments are undertaken, additional dosimetric studies should be carried out, some of which are best done with a RANDO Phantom.

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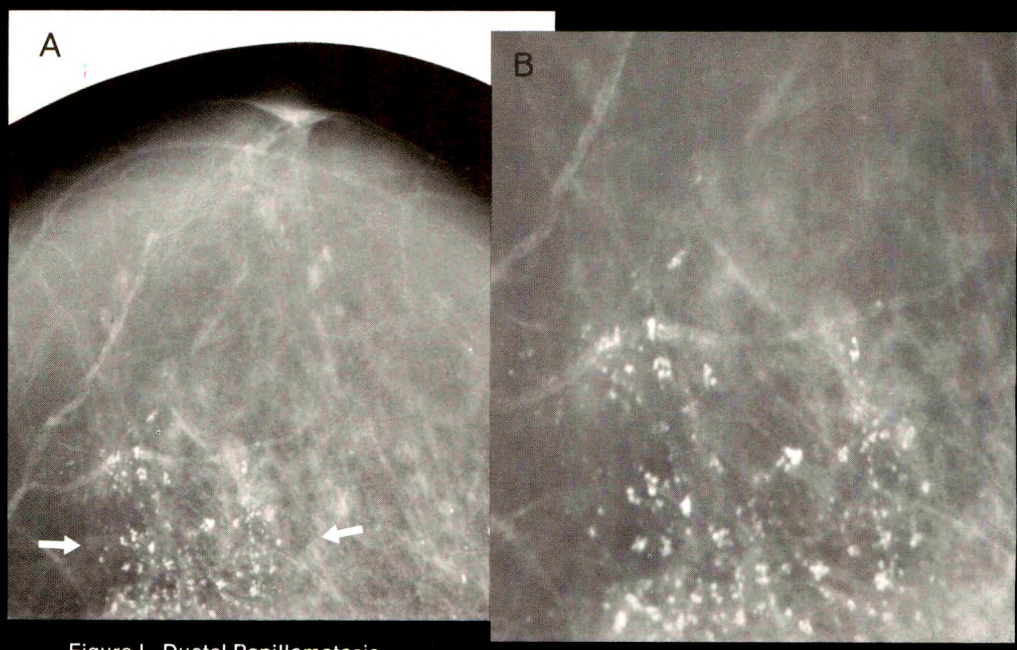


Figure I—Ductal Papillomatosis
A. Craniocaudal view reveals calcifications of varying size and configuration (arrows), seen to better advantage in Figure B, which look very much like the calcification seen in breast cancer. Clinically, the patient had no complaints. The breast was operated upon because of the mammographic suggestion of possible intraductal carcinoma. Biopsy revealed fibrocystic disease with proliferative changes including papillomatosis and adenosis.

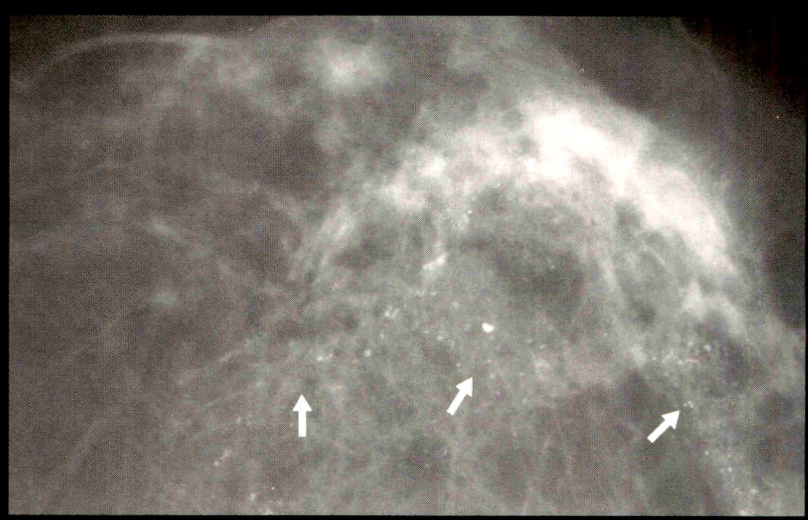


Figure II—
Fibrocystic Disease
This breast, too, was removed surgically because of innumerable punctate calcifications (arrows) considered cancer mammographically. Breast biopsy revealed diffuse fibrocystic disease with calcifications in and around dilated ducts.

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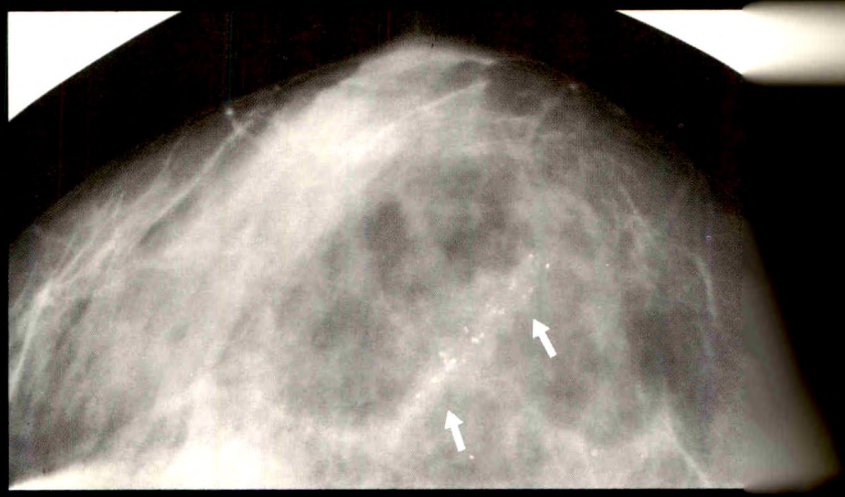
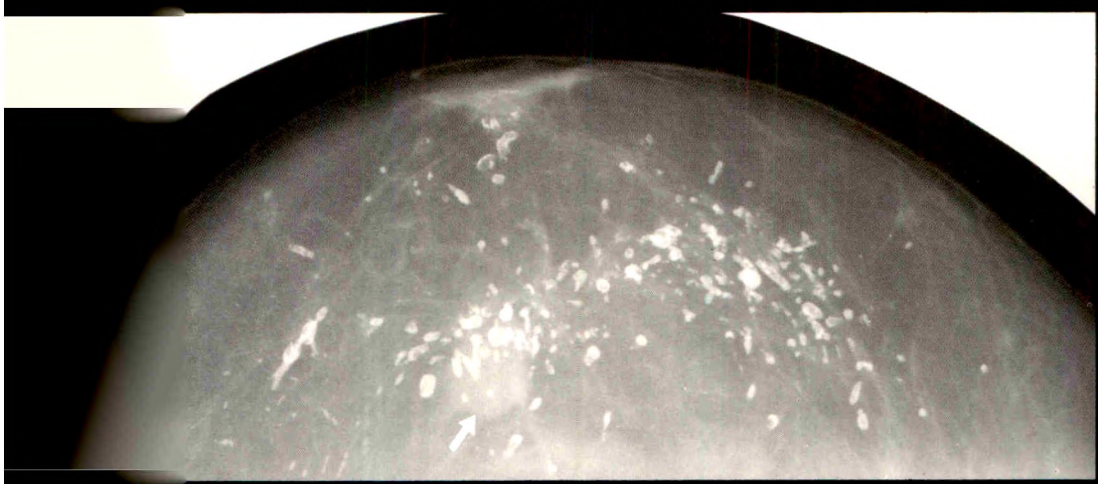


Figure IV—Comedomastitis Plus Carcinoma
Craniocaudal view reveals innumerable coarse linear and somewhat round calcifications distributed throughout most of the breast. The patient was known to have a small palpable mass in the center of this breast. On close inspection, a modular density may be seen (arrow), which is partially obscured by the benign calcifications that surround it. The nodule proved to be a small (1.5 cm) carcinoma; the rest of the breast revealed extensive mammary duct ectasia with calcifications in the wall of the ducts. The experienced observer remembers that sclerosing adenosis, ductal papillomatosis, and fibrocystic disease may reveal tiny punctate calcifications that mimic cancer.



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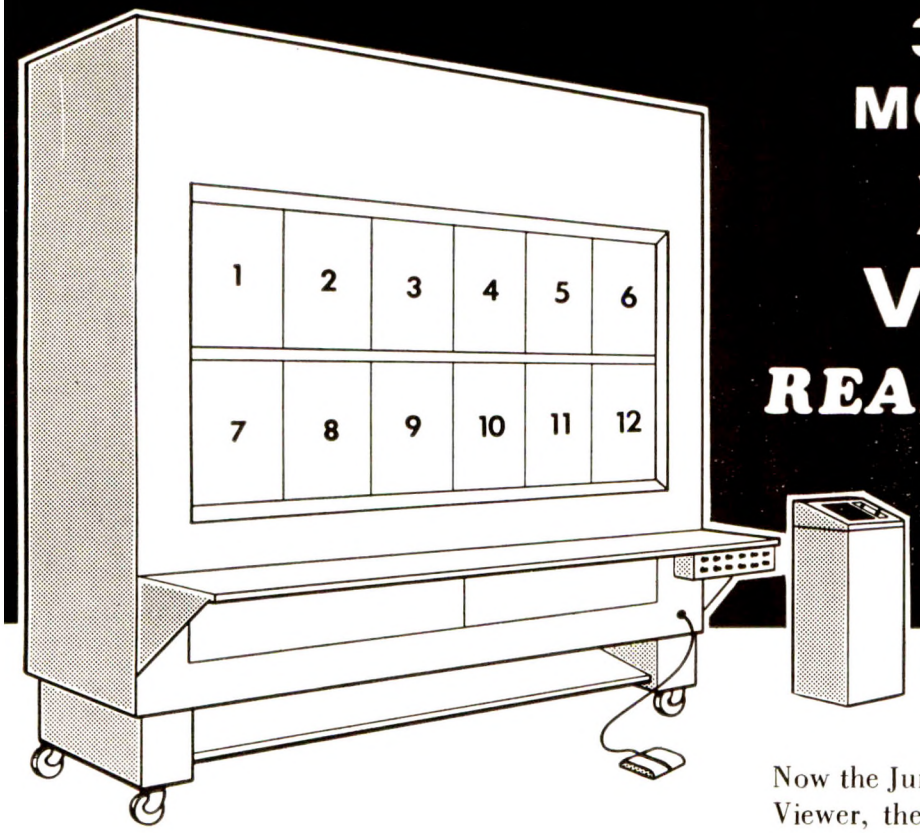
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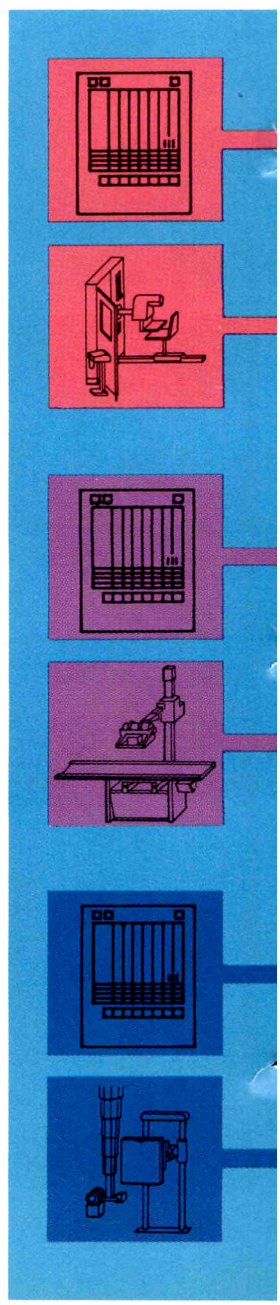


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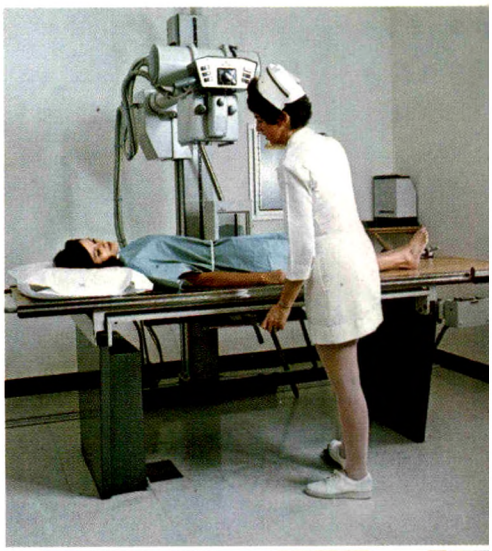
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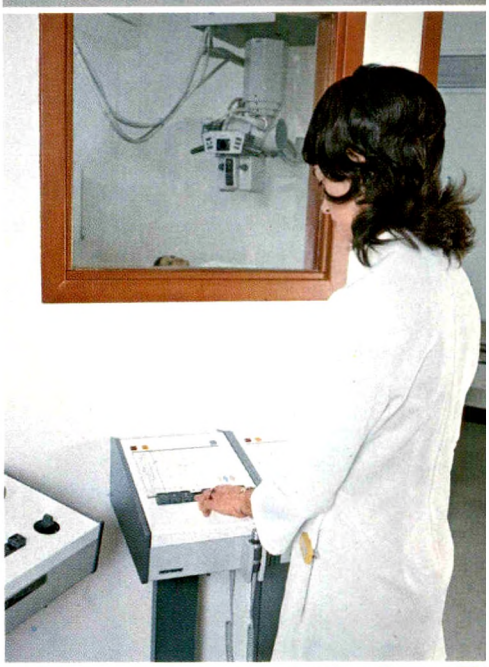
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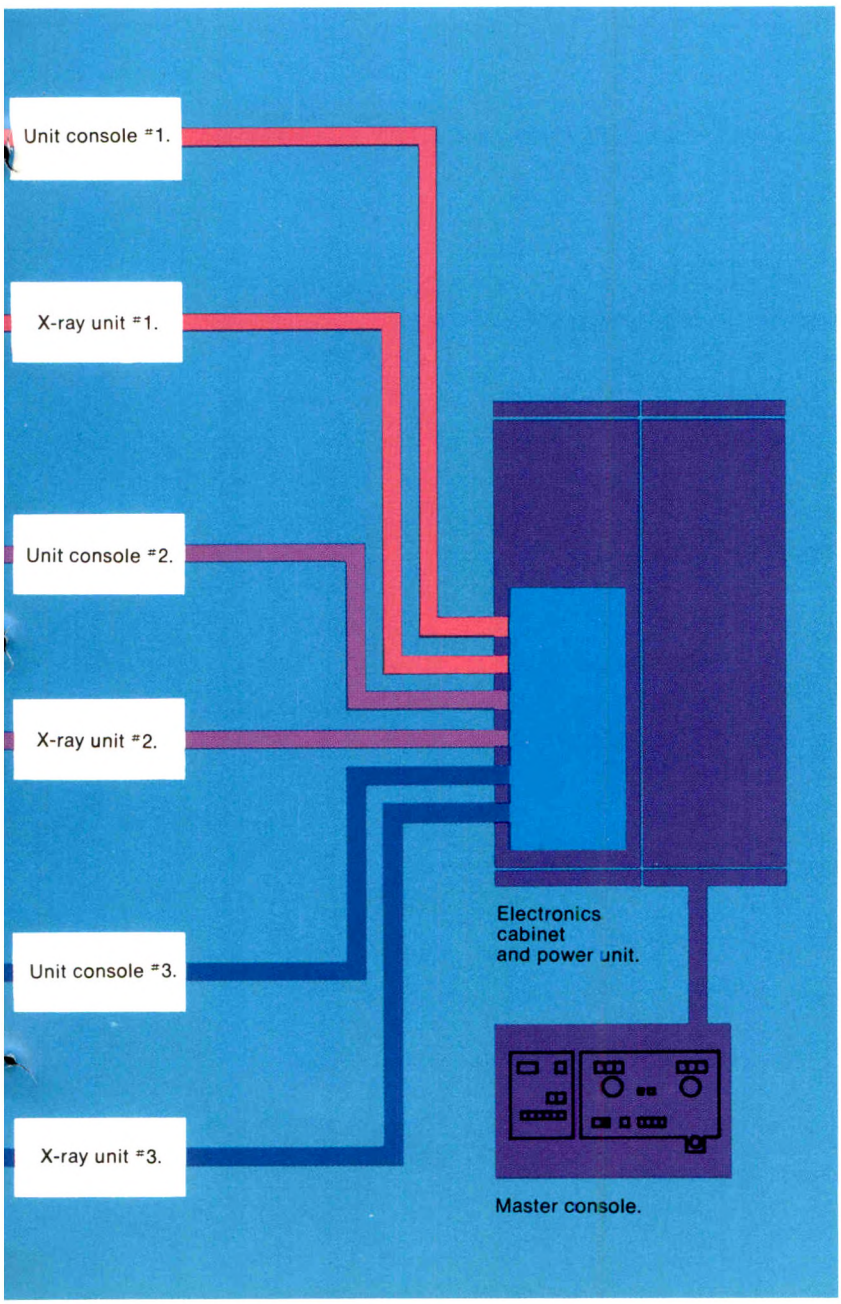
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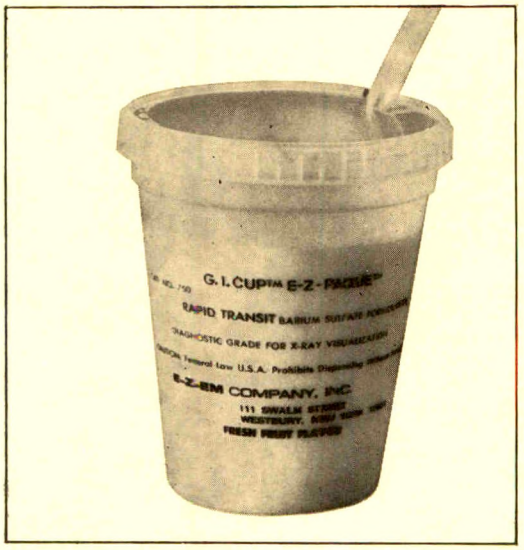
AGA Medical Thermovision ..... xxxviii, xxxix
Alderson Research Laboratories xlvi
Applied Radiation Corporation ..... vi, vii
Atomic Energy of Canada, Ltd. xxvii
Barnes-Hind Pharmaceuticals xxxiv
Eastman Kodak ..... xlviii, xlix
Elema-Schonander xvii
E-Z Em Company ..... Inside Back Cover
General Electric ..... xliv, xlv
Ilford X-Ray ..... xviii, xix
Low X-Ray xxviii
Machlett Laboratories ..... Inside Front Cover
Donald McElroy xxvi
Medi-Tech xxiii
Mosby, The C. V. Company ..... Back Cover
Ortho Pharmaceuticals xlvii
Philips Medical Systems (U.S.A.) ..... xl, xli
Picker Corporation xxxv
Procter & Gamble ..... xxiv, xxv, xxvi
Profexray, Inc. ..... xxxix, li
Purdue-Frederick Company v
Radx Corporation i
Schneeman Electronics, Inc. xliii
Shimadzu Seisakusho, Ltd. xvi
Siemens Corporation ..... xxxii, xxxiii, lii, liii
Society of Photo-Optical Instrumentation Engineers xv
E. R. Squibb & Sons, Inc. ..... xii, xiii, xiv, xx, xxi, xxii
S & S X-Ray l
Thomas, Charles C, Publisher ..... xxxi, xxxvi
3-M Company ..... viii, ix
Winthrop Laboratories ..... x, xi, xlii, xliii
Xonics ..... xxix, xxx

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We try to present an accurate index. Occasionally this may not be possible because of a last-minute change or an omission.

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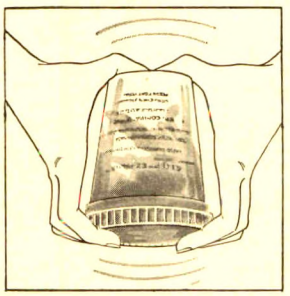
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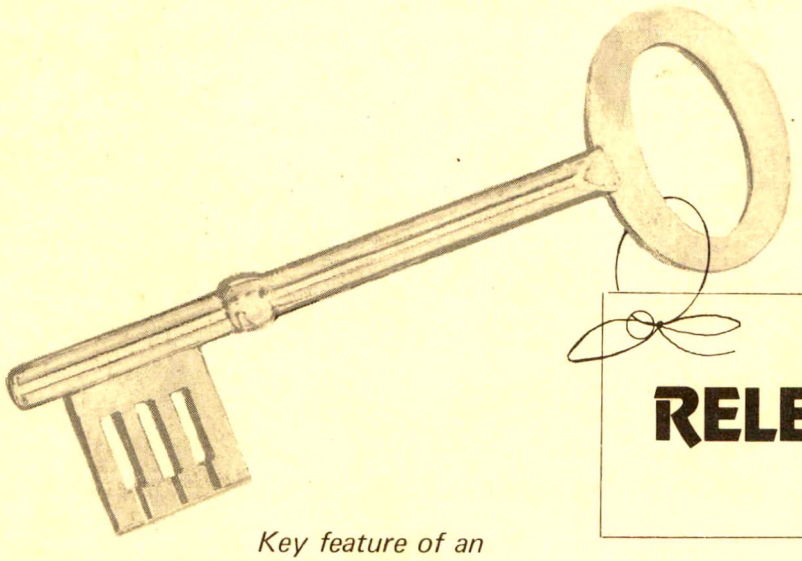
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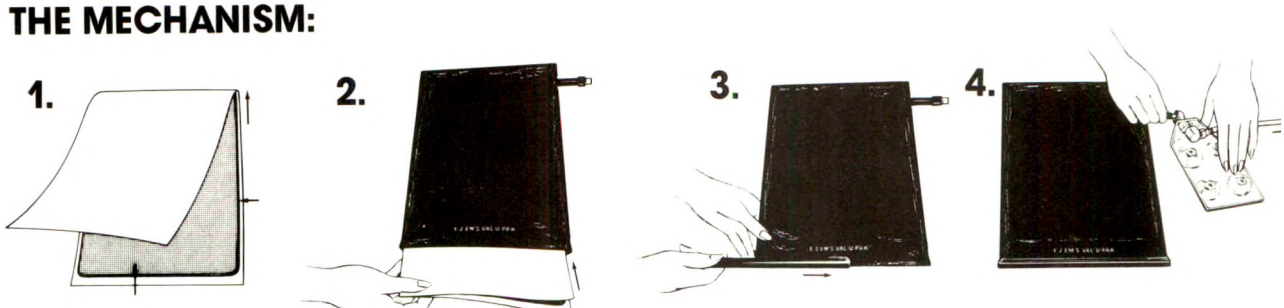
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VOL. 121 JULY, 1974 No. 3

TABLE OF CONTENTS

Radiotherapy of Tumors of the Urinary Tract: Hickey Lecture, 1974. J. A. DEL REGATO, M.D. 467

Megacalyces: Further Observations and Differentiation from Obstructive Renal Disease. LEE B. TALNER, M.D., AND RUBEN F. GITTES, M.D. . . . 473

Roentgenographic Findings in Urinary Tract Tuberculosis: A 10 Year Review. STEPHEN A. KOLLINS, M.D., GLEN W. HARTMAN, M.D., DAVID T. CARR, M.D., JOSEPH W. SEGURA, M.D., AND ROBERT R. HATTERY, M.D. 487

Xanthogranulomatous Pyelonephritis. MICHAEL C. BEACHLEY, M.D., KLAUS RANNIGER, M.D., AND FRANZ-JOSEPH ROTH, M.D. 500

Angiography in Unilateral Renal Bleeding with a Negative Urogram. HAROLD A. MITTY, M.D., AND HOWARD GOLDMAN, M.D. 508

Adrenal Tumors Simulating Intrarenal Lesions. GARY G. SHRAGO, MAJOR, MC, C. MALCOLM MCKINNON, MAJOR, MC, AND ROBERT CLARK, MAJOR, MC 518

Diagnosis of Perirenal and Subcapsular Masses: Anatomic-Radiologic Correlation. MORTON A. MEYERS, M.D., JOSEPH P. WHALEN, M.D., AND JOHN A. EVANS, M.D. 523

Angiographic Features of Alport's Syndrome: Hereditary Nephritis. VINCENT P. CHUANG, M.D., AND STEWART R. REUTER, M.D. 539

Acute Renal Failure after Angiography. FREDERICH K. PORT, M.D., RICHARD D. WAGONER, M.D., AND RICHARD E. FULTON, M.D. . . . 544

Arteriography and Phlebography of the Limbs in Planning Shunt and Fistula Operations in Patients with Chronic Renal Failure. M. LEA THOMAS, A. S. RAPPAPORT AND A. J. WING 551

Congenital Subclavian Steal. MICHAEL J. BORUSHOK, M.D., ROBERT I. WHITE, JR., M.D., KOOK SANG OH, M.D., AND JOHN P. DORST, M.D. . 559

Angulated Views in the Sagittal Plane for Improved Accuracy of Cinecoronary Angiography. JACQUES LESPÉRANCE, M.D., JACQUES SALTIEL, M.D., ROBERT PETITCLERC, M.D., AND MARTIAL G. BOURASSA, M.D. . . 565

New Lordotic Projection for Improved Visualization of the Left Coronary Artery and Its Branches. THOMAS A. SOS, M.D., JACK G. LEE, M.D., DAVID C. LEVIN, M.D., AND HAROLD A. BALTAXE, M.D. 575

(*Continued on page iv*)

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Coronary Arteriography in Left Main Coronary Artery Disease. Josef Rösch, M.D., Henry De Mots, M.D., Ruza Antonovic, M.D., Shahbudin H. Rahimtoola, M.B., F.R.C.P., Melvin P. Judkins, M.D., and Charles T. Dotter, M.D. 583

Angiographic Demonstration of the Absence of an Atrial Septal Defect in the Presence of Partial Anomalous Pulmonary Venous Connection. Thomas A. Sos, M.D., Daniel Tay, M.D., Aaron R. Levin, M.D., M.R.C.P., David C. Levin, M.D., and Harold A. Baltaxe, M.D. 591

Left Ventricular Angiographic Anatomy of Ostium Primum Defect in the Adult. Patricia A. Randall, M.D., Daniel J. Goodman, M.D., and John S. Schroeder, M.D. 597

The Importance of Selective Angiography of Systemic Arterial Supply to the Lungs in Planning Surgical Correction of Pseudotruncus Arteriosus. David C. Levin, M.D., Harold A. Baltaxe, M.D., Henry P. Goldberg, M.D., Mary Allen Engle, M.D., Paul A. Ebert, M.D., Thomas A. Sos, M.D., and Aaron R. Levin, M.D. 606

Bronchocutaneous Fistula As a Late Complication of Permanent Epicardial Pacing. Charles J. Tegtmeyer, M.D., James G. Hunter, Jr., M.D., and Theodore E. Keats, M.D. 614

Roentgenographic Evaluation of Dissecting Aneurysms of the Aorta. Michael C. Beachley, M.D., Klaus Ranniger, M.D., and Franz-Joseph Roth, M.D. 617

Continuous Ultrasound "B" Scanning of Abdominal Aortic Aneurysms. F. Winsberg, M.D., C. Cole-Beuglet, M.D., and D. S. Mulder, M.D. 626

Double Catheter Technique in Aortography. John E. Antoine, M.D., Philip J. Middleton, M.D., and Philip W. Carmody, M.D. 634

Radiation Reduction by Means of Low Pulse-Rate Fluoroscopy during Cardiac Catheterization and Coronary Arteriography. J. H. Grollman, Jr., M.D. 636

Hysterosalpingography in Infertility. Heun Y. Yune, M.D., Eugene C. Klatte, M.D., Robert E. Cleary, M.D., and Loren Petersen, M.D. 642

Officers 652

Editorial

Heuristic Gems from the American Radium Society. Traian Leucutia, M.D. 653

American Roentgen Ray Society, Section on Instruction 661

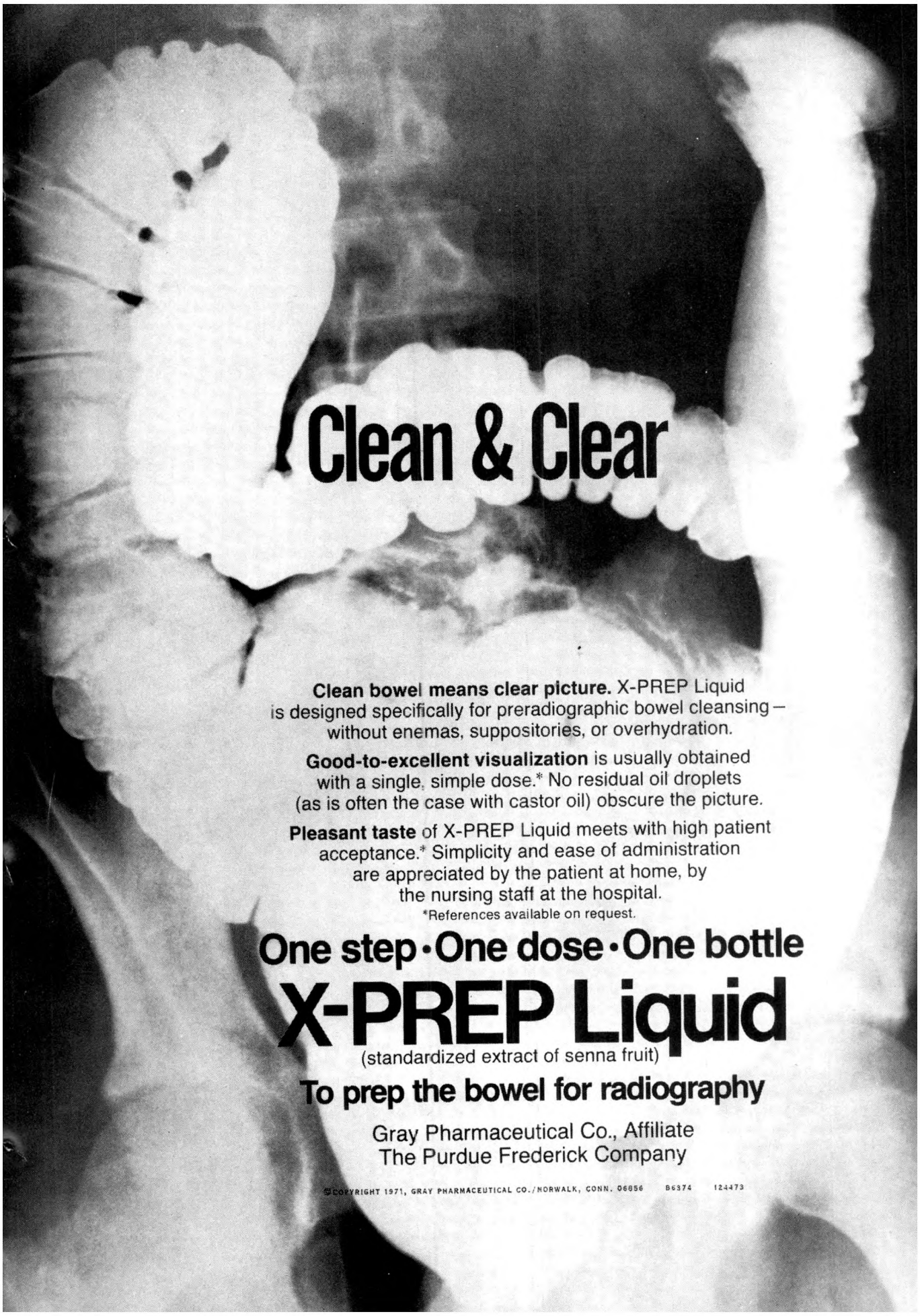
News Items 685

Book Reviews 687

Books Received 688

Society Proceedings 689

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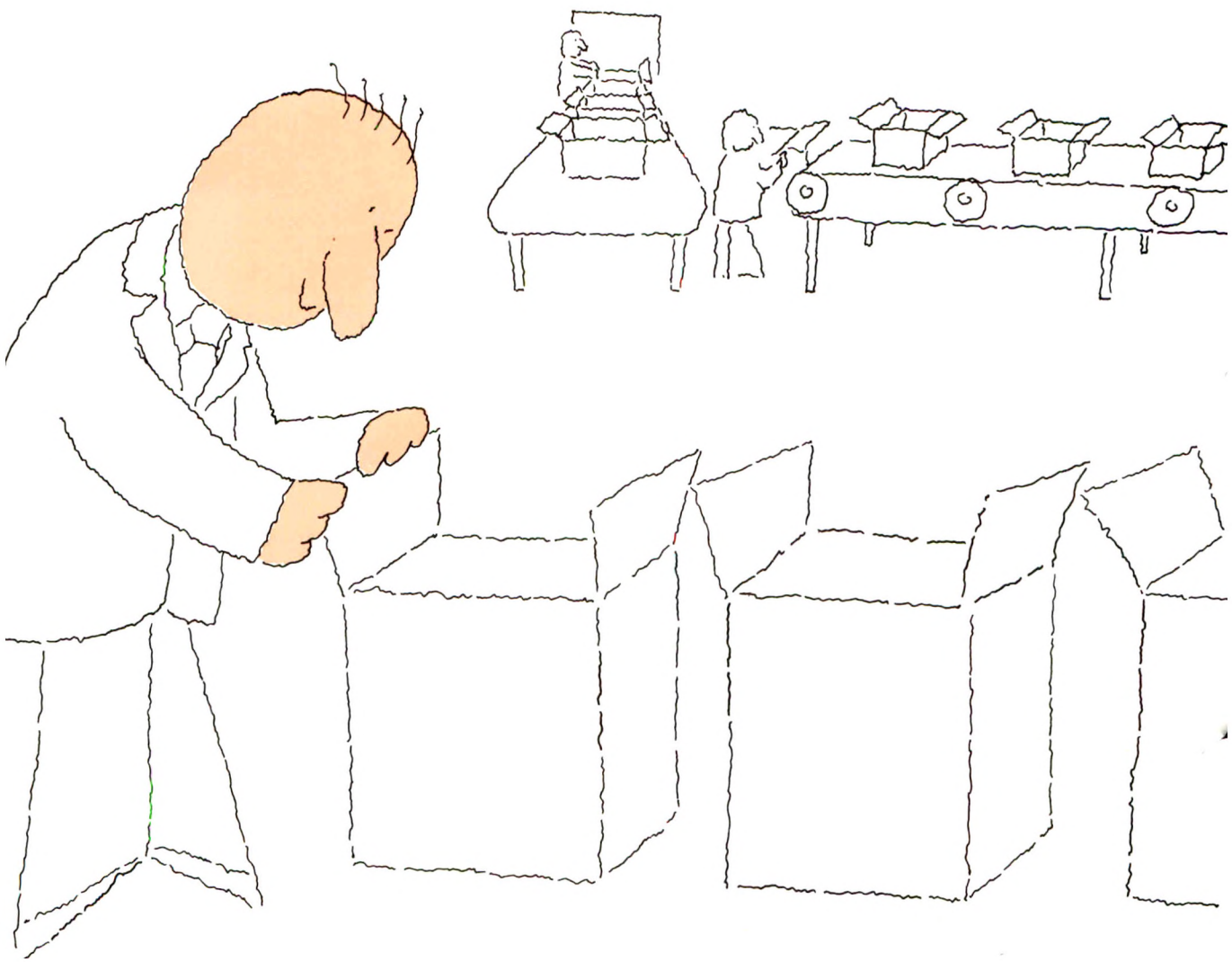
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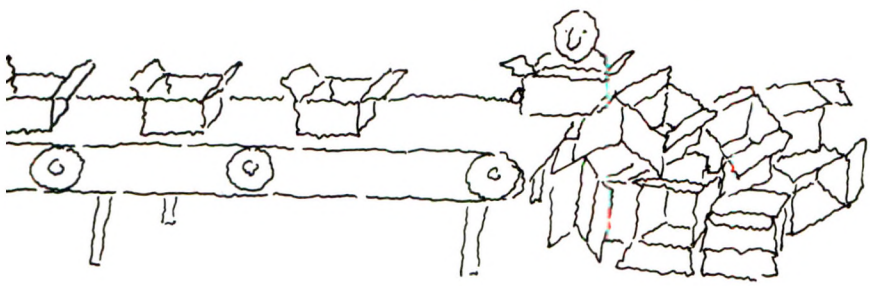
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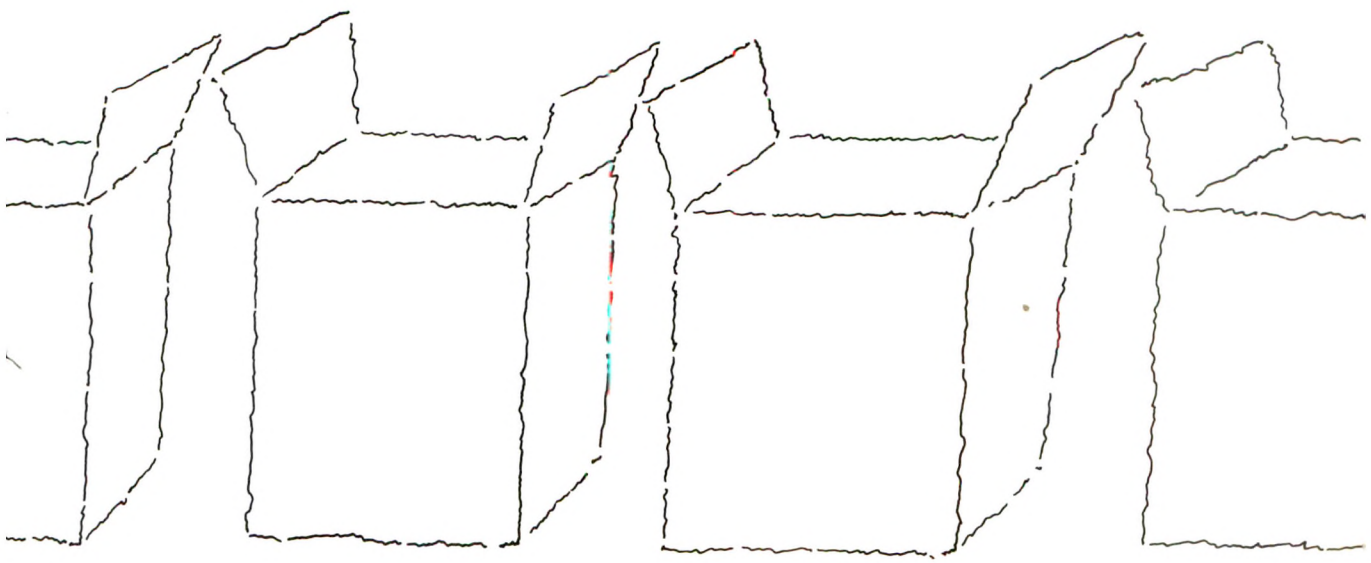
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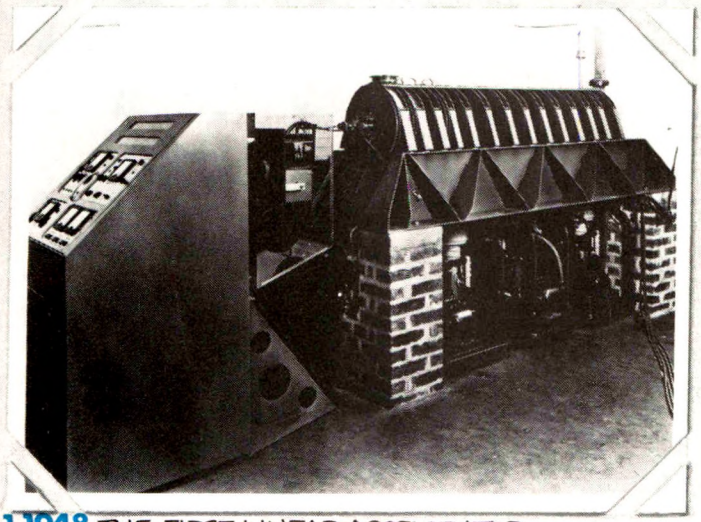
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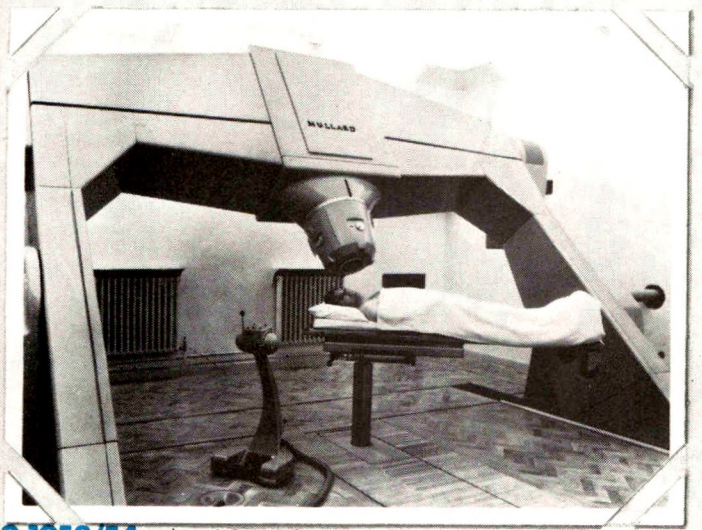
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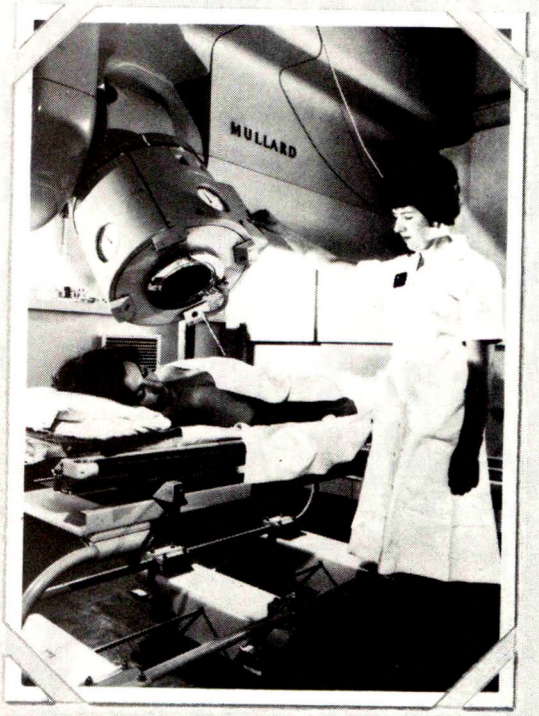
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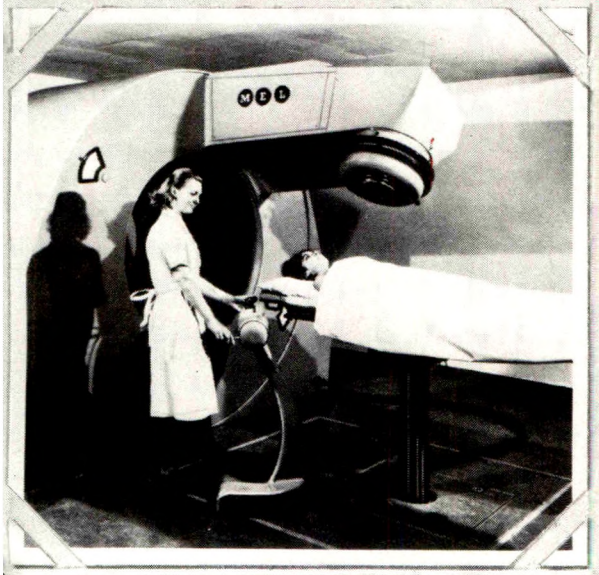


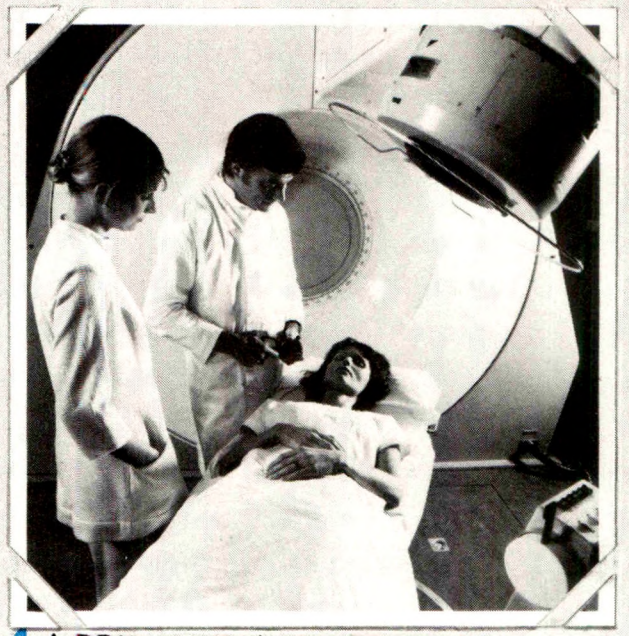


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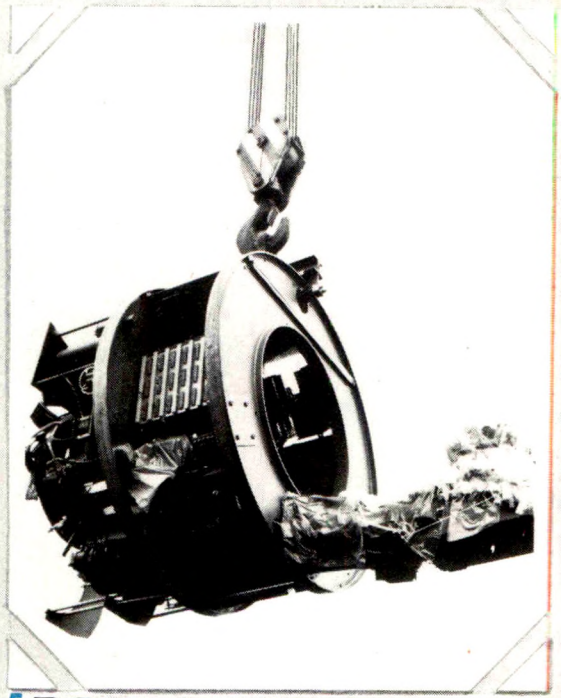
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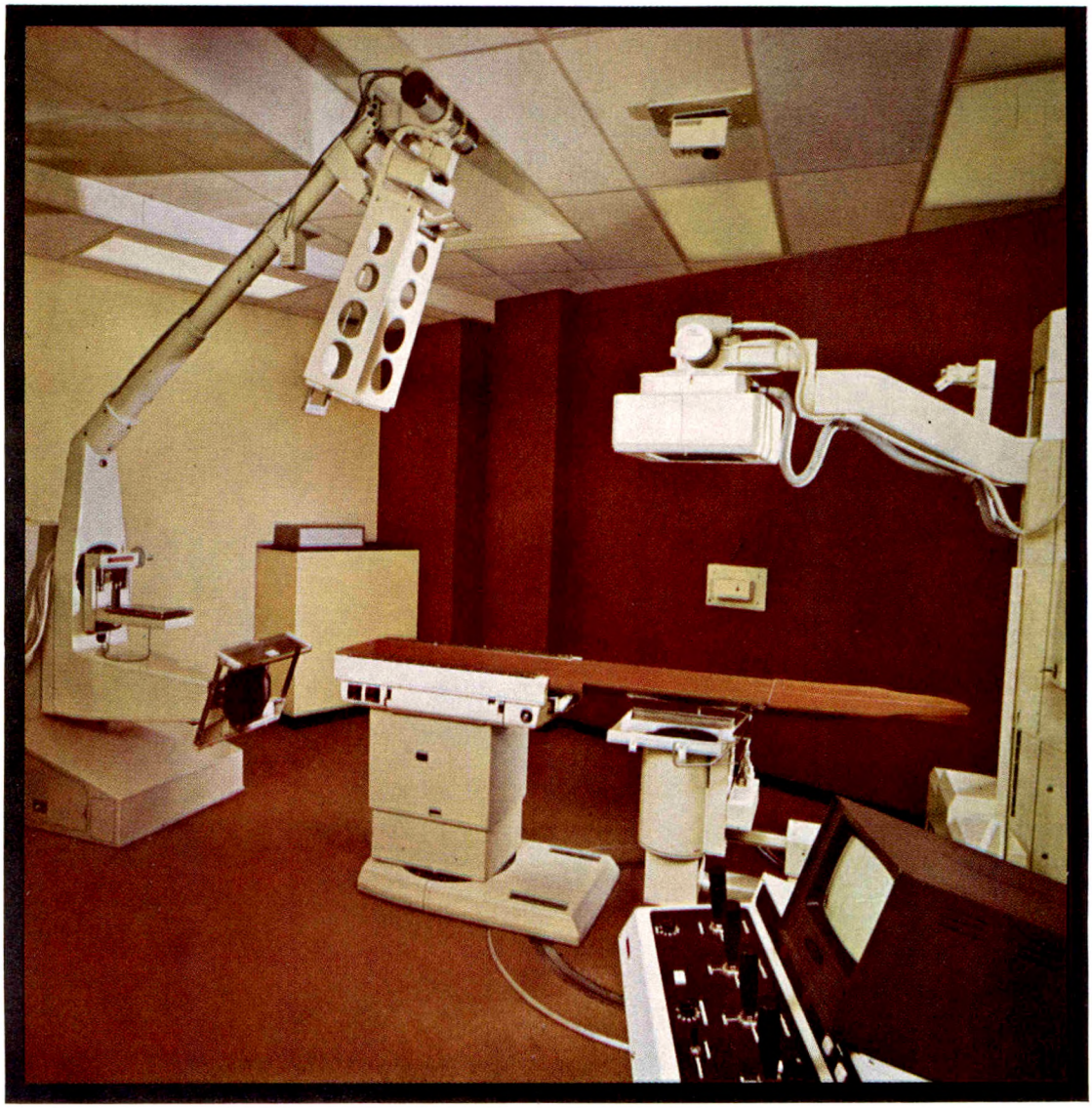
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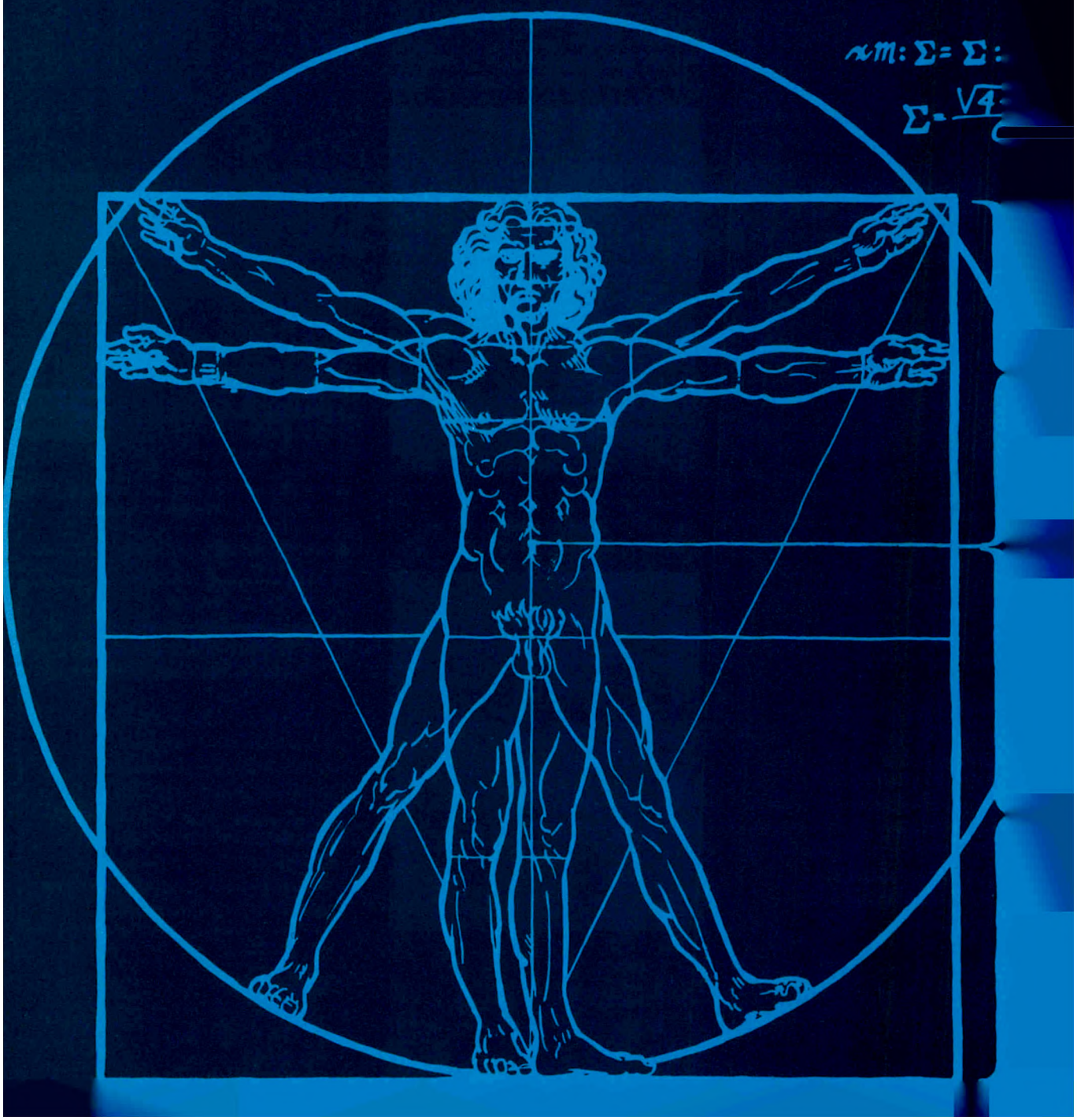
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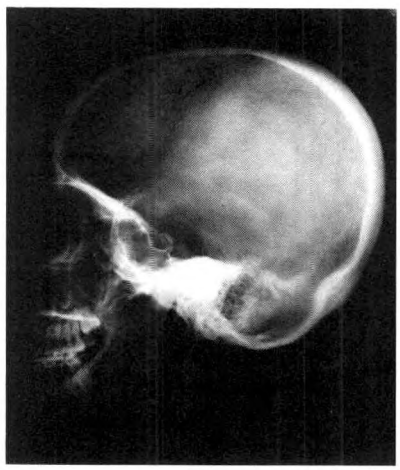
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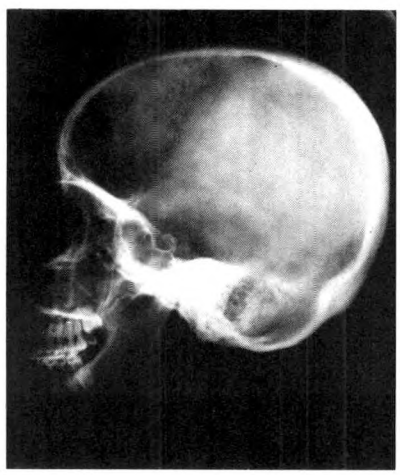
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*Radiology 105: 185-190, October, 1972. X-ray exposure reduction using Rare-Earth Oxysulfide Intensifying Screens; Robert A. Buchanan, PhD., Solon I. Finkelstein, M.D., and Kenneth A. Wickersheim, PhD.

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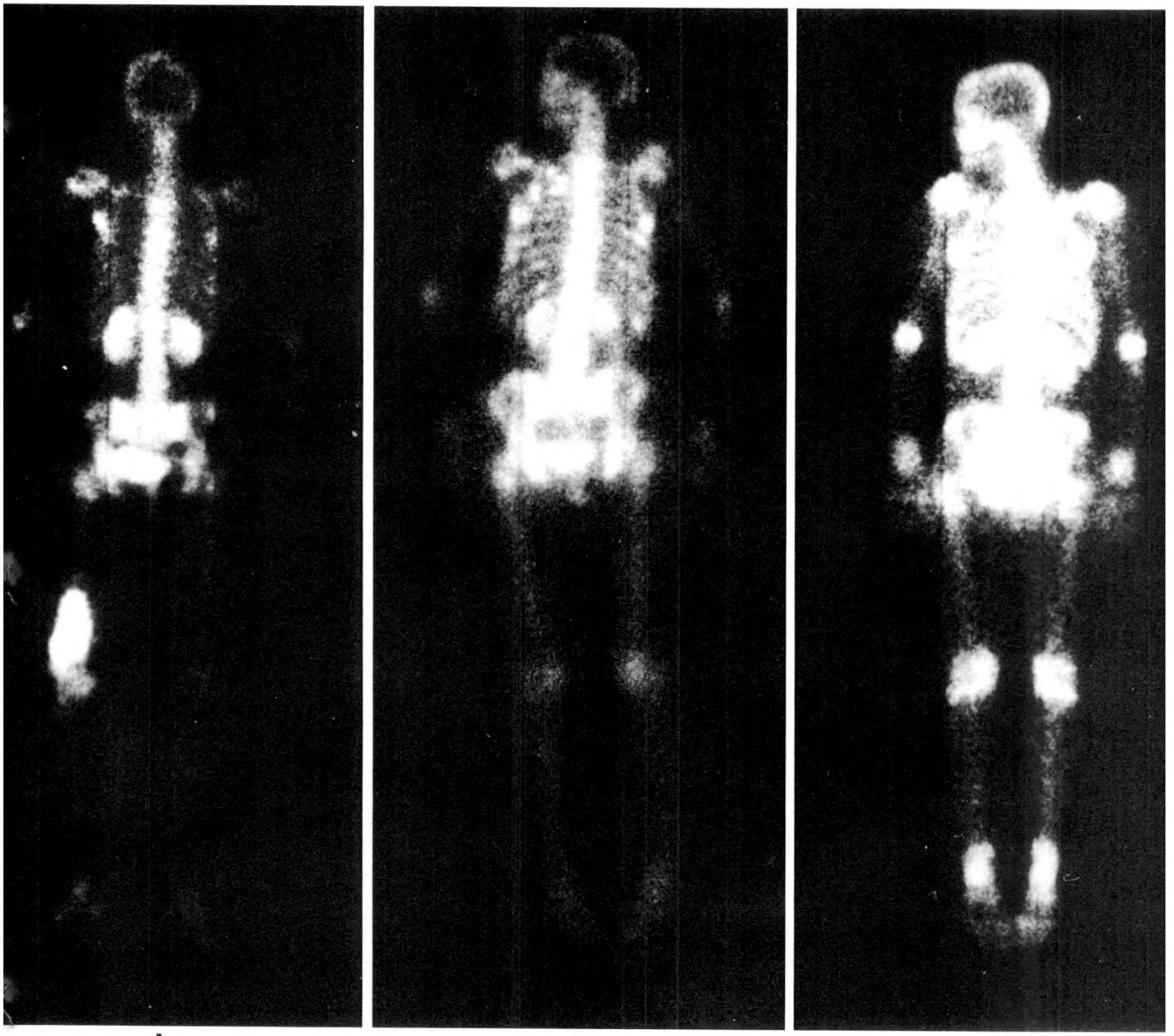
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SKELETAL IMAGING AGENT

See following page for brief summary of package insert.

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OSTEOSCAN

(5.9 MG DISODIUM ETIDRONATE
0.16 MG STANNOUS CHLORIDE)
SKELETAL IMAGING AGENT



Brief summary of Package Insert. Before using, please consult the full Package Insert included in each kit.

DESCRIPTION

Each vial of OSTEOSCAN contains 5.9 mg disodium etidronate and 0.16 mg stannous chloride as active ingredients. Upon addition of ADDITIVE-FREE ^{99m}Tc-pertechnetate, these ingredients combine with ^{99m}Tc to form a stable soluble complex.

ACTIONS (CLINICAL PHARMACOLOGY)

When injected intravenously, ^{99m}Tc-labeled OSTEOSCAN has a specific affinity for areas of altered osteogenesis. Areas of bone which are undergoing neoplastic invasion often have an unusually high turnover rate which may be imaged with ^{99m}Tc-labeled OSTEOSCAN.

Three hours after intravenous injection of 1 ml ^{99m}Tc-labeled OSTEOSCAN, an estimated 40-50% of the injected dose has been taken up by the skeleton. At this time approximately 50% has been excreted in the urine and 6% remains in the blood. A small amount is retained by the soft tissue. The level of ^{99m}Tc-labeled OSTEOSCAN excreted in the feces is below the level detectable by routine laboratory techniques.

INDICATIONS

OSTEOSCAN is a skeletal imaging agent used to demonstrate areas of altered osteogenesis.

CONTRAINDICATIONS

None.

WARNINGS

This radiopharmaceutical should not be administered to patients who are pregnant or lactating unless the information to be gained outweighs the potential hazards.

Ideally, examinations using radiopharmaceuticals, especially those elective in nature, of a woman of childbearing capability should be performed during the first few (approximately 10) days following the onset of menses.

Radiopharmaceuticals should be used only by physicians who are qualified by specific training in the safe use and handling of radionuclides produced by nuclear reactor or particle accelerator and whose experience and training have been approved by the appropriate government agency authorized to license the use of radionuclides.

The ^{99m}Tc-generator should be tested routinely for molybdenum breakthrough and aluminum. If either is detected, the eluate should not be used.

PRECAUTIONS

Both prior to and following ^{99m}Tc-labeled OSTEOSCAN administration, patients should be encouraged to drink fluids. Patients should void as often as possible after the ^{99m}Tc-labeled OSTEOSCAN injection to minimize background interference from accumulation in the bladder and unnecessary exposure to radiation.

As in the use of any other radioactive material, care should be taken to insure minimum radiation exposure to the patient, consistent with proper patient management, and to insure minimum radiation exposure to occupational workers.

ADVERSE REACTIONS

None.

DOSAGE AND ADMINISTRATION

The recommended adult dose of ^{99m}Tc-labeled OSTEOSCAN is 1 ml with a total activity range of 10-15 mCi. ^{99m}Tc-labeled OSTEOSCAN should be given intravenously by slow injection over a period of 30 seconds within three (3) hours after its preparation. Optimum scanning time is 3-4 hours postinjection.

The patient dose should be measured by a suitable radioactivity calibration system immediately prior to administration.

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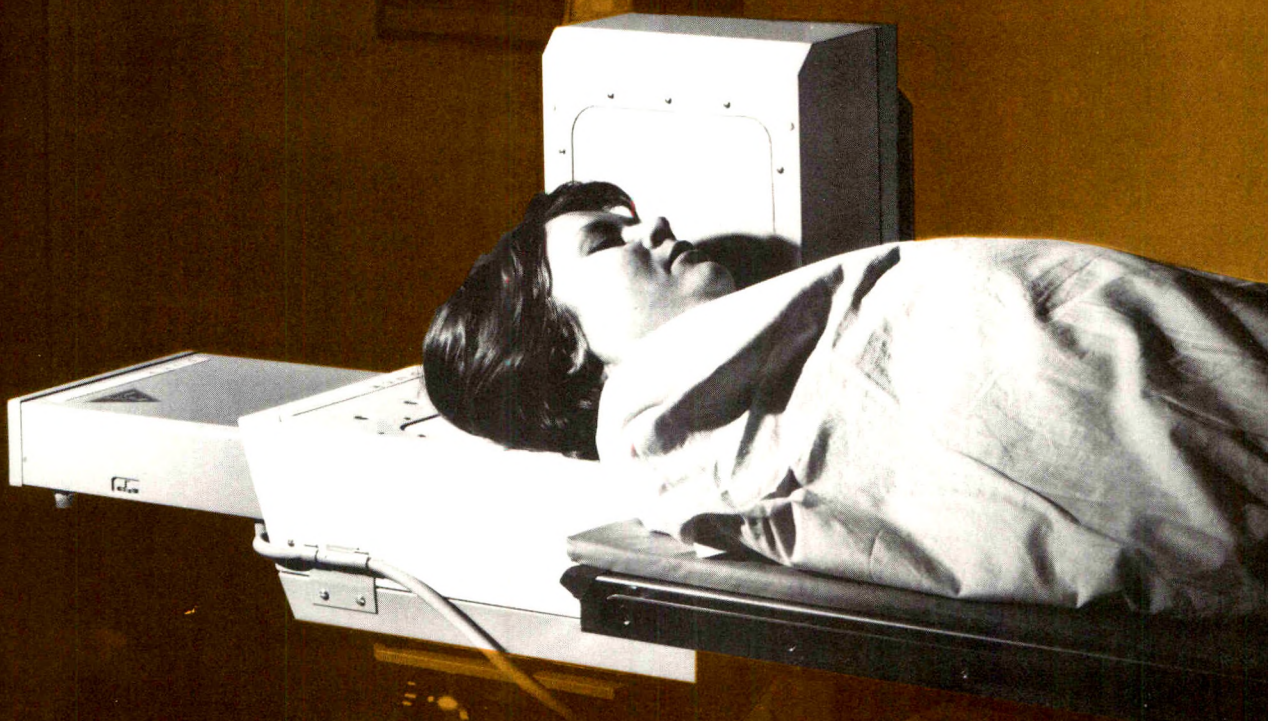
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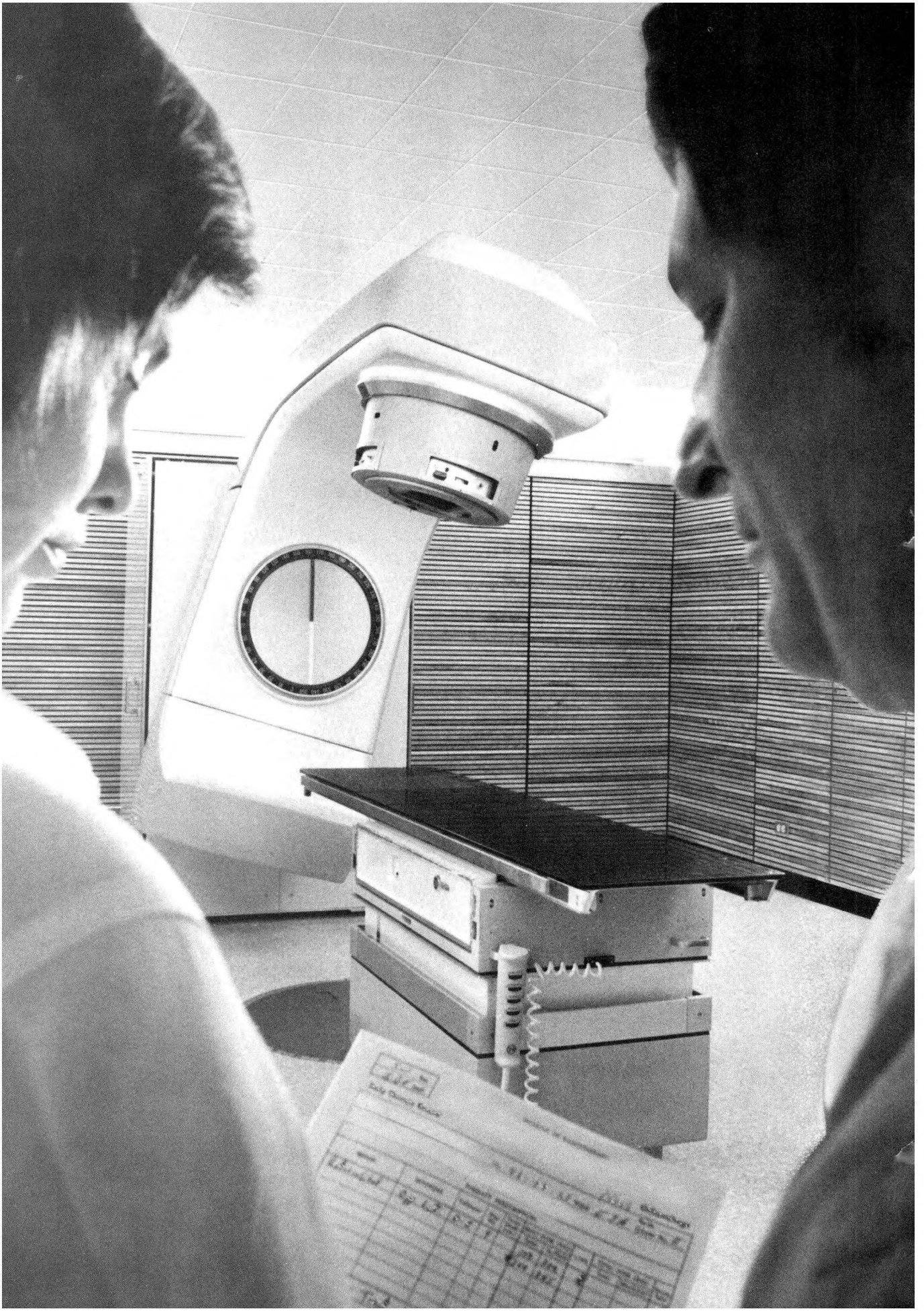
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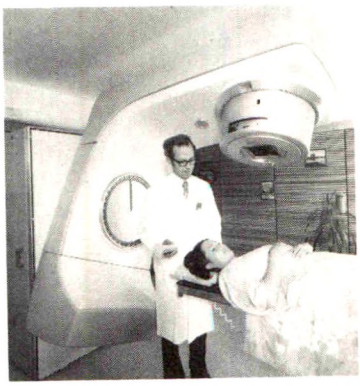
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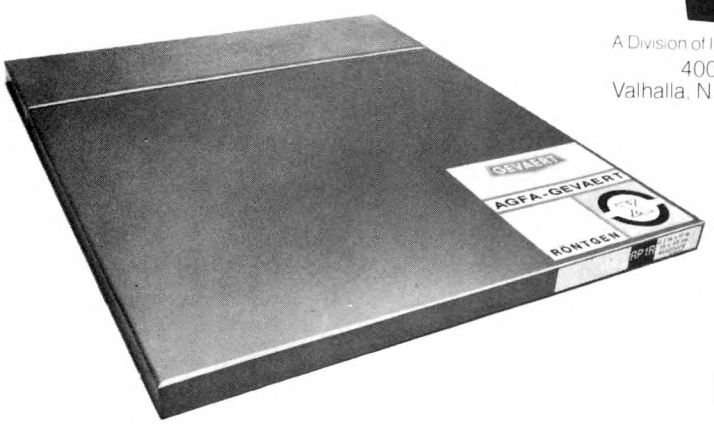
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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 121 JULY, 1974 No. 3

RADIOTHERAPY OF TUMORS OF THE URINARY TRACT*

HICKEY LECTURE, 1974

By J. A. DEL REGATO, M.D.†
TAMPA, FLORIDA

THE remarkably prompt utilization of radiations in the treatment of cancer led the pioneers of therapeutic radiology into early and fruitful interaction with dermatologists, gynecologists and otolaryngologists. Indeed, some of these specialists contributed, with their skill and dedication, to the development of techniques and basic concepts of radiotherapy which were to be widely adopted. By contrast, the interaction of radiotherapists and urologists has always been light and limited to the treatment of testicular tumors; in fact, the lack of a widely held point of view on this very subject attests to the need for a better understanding among the advocates of both specialties.

An impartial assessment of the results of surgery in the treatment of urinary tumors, the advent of sources of nobler quality radiations, as well as the development of new concepts and skills, have brought about, recently and rather suddenly, an uneasy interaction between urologists and radiotherapists in the treatment of three major forms of cancer.

THE KIDNEY

(*Fig. 1*)

For a long time therapeutic radiologists labored under a dogma of the "radioresistance" of adenocarcinomas; radioresistance implied radioincurability and as a consequence all such tumors were either treated by surgery or not at all. As it often happens, the obedient acquiescence of the dogma deprived everyone of an opportunity to test its veracity. Very slowly and painstakingly we have learned that the assumed radioresistance of adenocarcinomas was in reality the result of delayed response. What is involved can be expressed as a law of radiopathophysiology of tumors: *the regressive speed of irradiated tumors is proportional to their rate of development before irradiation*. Thus, although the rates may not be commensurate, a fast growing

* Presented at the Joint Meeting of the Wayne County Medical Society, the Wayne State University College of Medicine and the Michigan Radiological Society, Detroit, Michigan, March 7, 1974.

† Professor of Radiology, University of South Florida College of Medicine, Tampa, Florida.

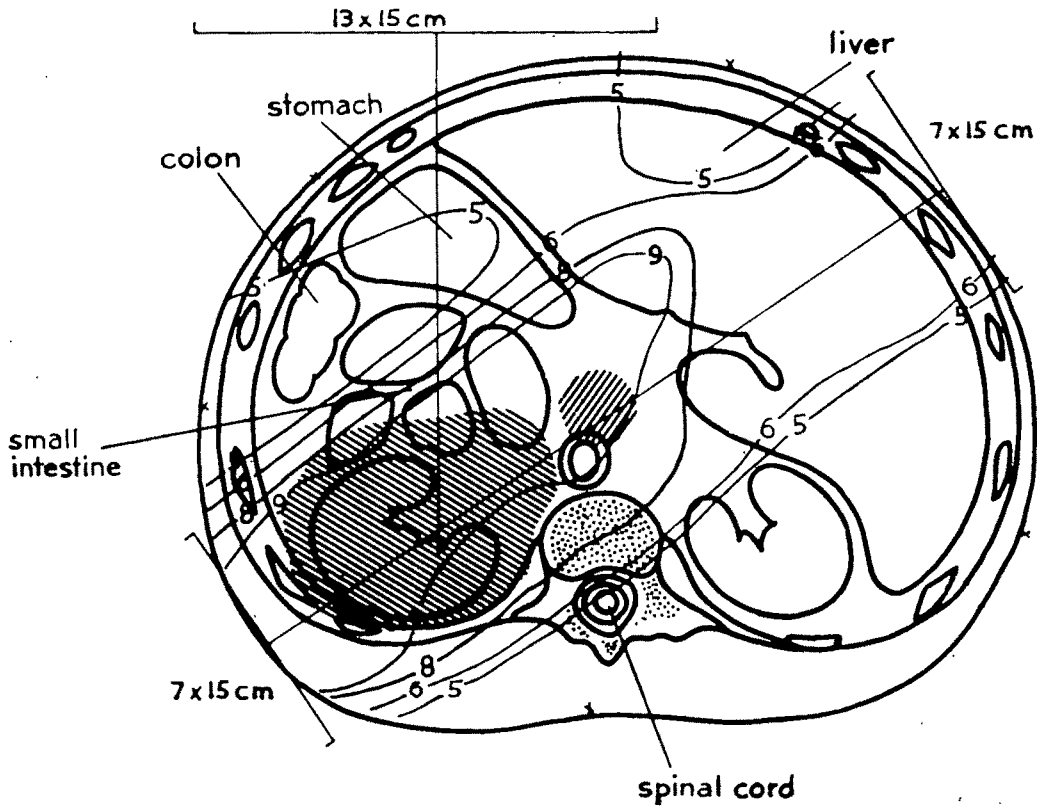


FIG. 1. Schematic plan for the preoperative irradiation of a renal tumor and its potential mid-line metastases, while avoiding the untoward irradiation of the spinal cord and the opposite kidney. Treatments should be fractionated over at least 5 weeks and an interval of no less than 4 weeks should be allowed before nephrectomy.

lymphosarcoma regresses rapidly under incomplete irradiation, whereas a slow developing adenocarcinoma may not have disappeared completely weeks and months after adequate irradiation.

Under favorable circumstances a nephrectomy is capable of curing a patient with renal carcinoma; however, the operation is often insufficient to the task because of the frequent spread of tumor to the perirenal bed, and because of vascular involvement and early development of paraaortic metastases. It was the genial Sir Eric Riches with the cooperation of one of the most capable clinical radiotherapists in England, Sir Brian Windeyer, who pointed out the usefulness of *postnephrectomy radiotherapy* in improving his results.[16]

It is a good rule of clinical radiotherapy that whenever postoperative radiotherapy proves useful, it is likely that preoperative radiotherapy may prove still more effective. *Preoperative irradiation* was tried with evident effectiveness some 40 years ago;[19] the subject has been taken up in recent years with the advantage of better quality radiations.[8,15] At the International Symposium on Renal Neoplasia, held in Brazil, in September, 1965, Kagan and I discussed some basic considerations in the irradiation of these tumors.[8] Supported by Sir Eric Riches and by myself, Dr. P. J. Cavanaugh, of Duke University, proposed a cooperative clinical trial of pre- and postoperative radiotherapy in renal carcinoma:[5] the initiative was officially approved by the select international assembly. This plan has been adopted by a group of young American urologists and radiotherapists, under the chairmanship of Dr. Clair E. Cox;[6] the

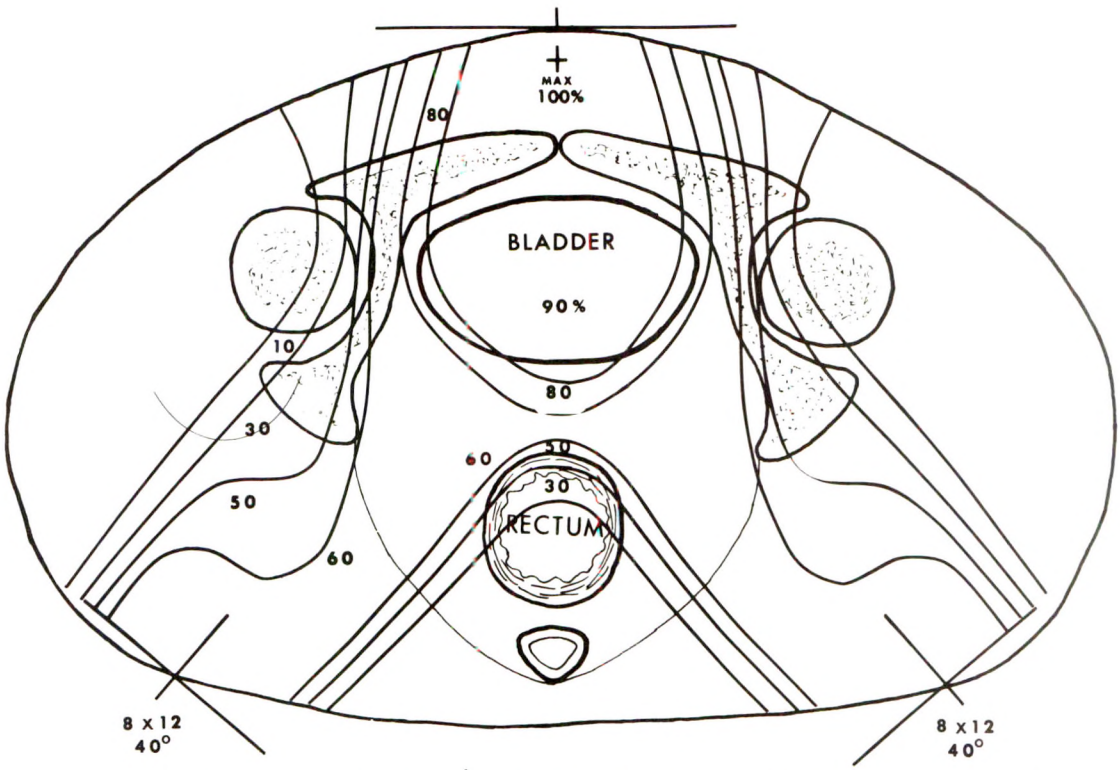


Fig. 2. Schematic plan for the irradiation of a tumor of the bladder and its pelvic extension through 1 anterior field and 2 posterolateral fields to produce a heavier irradiation of the anterior pelvis. Treatments should be fractionated over 7 to 8 weeks for best results.

study is now being conducted under a grant of the National Cancer Institute.

THE BLADDER

(*Fig. 2*)

It is generally known that some very well differentiated and pedunculated tumors of the bladder may be successfully destroyed through the cystoscope, but unfortunately a majority of these tumors either recur and become infiltrating or are ineligible from the beginning for this approach. Interstitial implantation of radon "seeds" through a suprapubic cystostomy became a popular treatment because of the excellent results reported by Barringer;[2] however, a reappraisal of these results[13] coincided with a rebirth of enthusiasm for the surgical treatment of cancer of the bladder. Unfortunately, these tumors are often situated on the trigone area and compromise the ureters, requiring a rather radical surgical approach; at the same time, they often occur in elderly patients whose pathologic talley makes them often ineligible for such procedures. Whether the surgical approach would be a wedge resection, a simple cystectomy, a radical cystectomy or a pelvic evisceration, either very few patients are eligible or the results are poor.

In 1961, a national cooperative study was proposed to test the value of radiotherapy and of chemotherapy as surgical adjuvants in the treatment of cancer of the bladder. With the assistance of Drs. M. D. Schulz, W. T. Moss, W. S. Maxfield, E. R. King and J. Stein, we developed a protocol for *preoperative radiotherapy*; we proposed a moderate but well fractionated total dose of radiations and insisted on an interval of several weeks before operation. The study, conducted under the chairmanship of Dr. George Prout, Jr.,[14] eventually revealed, to the surprise of the participating urol-

ogists that radiotherapy, unlike chemotherapy, did not interfere with the surgical procedure and that a whole third of the irradiated patients presented no residual tumor in the surgical specimen. The fractionation and presurgical interval were key elements not always appreciated by surgeons.

Although we cooperated with this surgical trial, our evaluation has long been drawn to the point of view that *integral external pelvic irradiation* is the treatment of choice of most patients with carcinoma of the bladder. Buschke and Cantril[4] were among the first to point out the advantage of supervoltage roentgen therapy in this area; their good results were perhaps unduly reduced by their excessive daily dose. Chahbazian and I reported results on a small series of inoperable patients who were treated with cobalt 60 teletherapy over periods of 8 weeks: this series yielded a 40 per cent 5 year survival and evidence that in 60 per cent of the cases the bladder tumor had been destroyed and healed.[7] The most surprising part of the results was the reacquired bladder elasticity and capacity. And again, the fractionation of these treatments failed to impress the surgeons as much as the dose.

THE PROSTATE

(Fig. 3)

At the turn of the Century, the efforts of urological surgeons such as Young and Albarán succeeded in the development of a radical perineal prostatectomy for the treatment of cancer of this structure. The well-conceived surgical procedure involves the removal of the entire gland, the seminal vesicles, the prostatic urethra *in toto* with the fascia of Denonvillier. The operation is

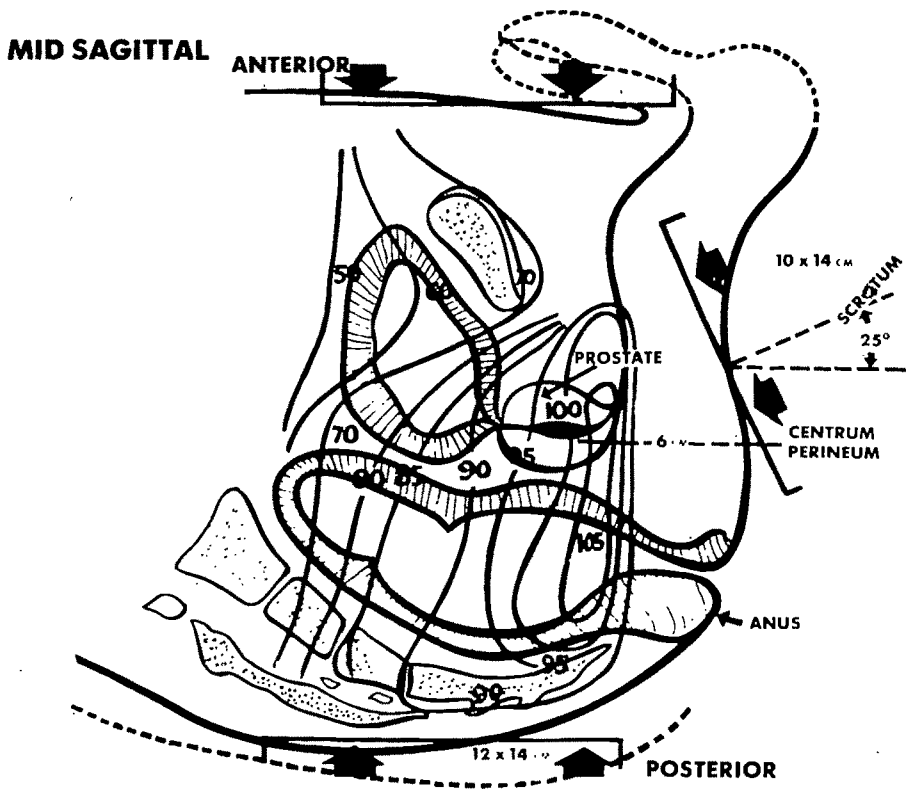


FIG. 3. Schematic plan for the irradiation of carcinoma of the prostate and its potential pelvic extension and metastases through 1 anterior, 1 posterior and 2 perineal fields. This rather homogeneous irradiation, when carried out daily for 7 to 8 weeks, allows for effective irradiation to a rather high dose without intensive reactions.

only successful if the tumor is confined within the capsule; it is invariably followed by impotence and frequently by incontinence. Because of extent of the lesion, age, or surgical risk, only about 5 per cent of all patients diagnosed are eligible for this approach. There were only 196 such operations performed at the Massachusetts General Hospital during a period of 25 years.[18] Yet, for a long time this was the only curative approach.

The work of Huggins[11] revealed the frequent hormone dependence of these tumors and the spectacular palliative usefulness of orchiectomy and/or estrogen therapy. Because so many patients are ineligible for curative surgery, urologists everywhere adopted the practice of these androgen-controlled procedures even before any palliation was called for. The assumption was that orchiectomy and/or estrogens would delay the growth, postpone the development of metastases and the appearance of symptoms, preserve the weight and general condition of the patient and prolong life. In a well-planned randomized study of the Urological Research Group of the Veterans Administration none of these assumptions was found to be true.[17]

Flocks and his associates[12] proposed to destroy carcinoma in the prostate and to pursue it to its metastases by means of the injection of radioactive gold; although these hopes proved often illusory, their work brought to the attention of urologists the serious possibilities of radiotherapy in this field.

In 1967, we reported on 4 inoperable patients with cancer of the prostate who were living and well 5 to 8 years after radical radiotherapy given with an aim to cure.[9] Contemporaneously, Bagshaw[1] accumulated a respectable number of patients with the same results. In February, 1965, we proposed to the Committee for Radiation Therapy Studies the organization of a cooperative study for a larger clinical trial of our experience. A protocol committee of urologists, radiotherapists, a physicist, an epidemiologist, a radiodiagnostician, and a pathologist was formed under our chairmanship. A grant of the National Cancer Institute permitted the necessary financial support for the conduct of the study; it implies verification of histopathology and survey of the dosimetric compliance of participants.[10] This study has now accumulated over 300 well documented cases; however, any present analysis of the gathered data would be premature. It is evident that we have come into possession of a curative approach to inoperable carcinomas of the prostate which remain confined within the pelvis. Once again, surgeons as well as others are impressed by the dose of radiations administered rather than the fractionation and field approach which make it possible to administer a rather large dose of radiations without untoward effects or important complications.

CONCLUSION

Fate has brought radiotherapists, rather precipitously, to play an important role in the treatment of patients with tumors of the urinary tract. Not only must they become acquainted with the protean character and clinical behavior of these tumors, they must also develop a working relationship and cooperative ability with the urologists; the task at hand requires learning as well as didactic ability, and patient understanding.

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REFERENCES

1. Bagshaw, M. A. Definitive radiotherapy in carcinoma of prostate. *J.A.M.A.*, 1969, *210*, 326–327.
2. Barringer, B. S. Twenty-five years of radon treatment of cancer of bladder. *J.A.M.A.*, 1947, *135*, 616–618.
3. Bratherton, D. G. Place of radiotherapy in treatment of hypernephroma. *Brit. J. Radiol.*, 1964, *37*, 141–146.
4. Buschke, F., and Cantril, S. T. Roentgentherapy of carcinoma of urinary bladder: analysis of 52 patients treated with 800 K.V. roentgentherapy. *J. Urol.*, 1942, *48*, 368–383.
5. Cavanaugh, P. J. Adjunctive radiotherapy in

renal carcinomas of adults. In: Renal Neoplasia. Edited by J. S. King, Jr. Little, Brown & Company, Boston, 1967, 559–569.

6. COX, C. E. Personal communication.
7. DEL REGATO, J. A., and CHAHBAZIAN, C. M. Radiotherapy for transitional-cell carcinoma of bladder with cobalt-60. *Radiology*, 1966, *87*, 1053–1057.
8. DEL REGATO, J. A., and KAGAN, A. R. Basic considerations in radiotherapy of renal carcinomas. In: Renal Neoplasia. Edited by J. S. King, Jr. Little, Brown & Company, Boston, 1967, 547–557.
9. DEL REGATO, J. A. Radiotherapy in conservative treatment of operable and locally inoperable carcinoma of prostate. *Radiology*, 1967, *88*, 761–766.
10. DEL REGATO, J. A. (Chairman.) Protocol and experimental design for a cooperative study of radiotherapy for carcinoma of the prostate Stage C. Grant No. 1-ROI-CA-10184-Rad, under the auspices of the National Institute, September, 1967.
11. HUGGINS, C. Prostatic cancer treated by orchiectomy: five-year result. *J.A.M.A.*, 1946, *131*, 576–581.
12. KERR, H. D., FLOCKS, R. H., ELKINS, H. B., and CULP, D. A. Follow-up study of one hundred cases of carcinoma of prostate treated with radioactive gold. *Radiology*, 1955, *64*, 637–641.
13. MARSHALL, V. F. Results of radiation therapy of bladder cancer. *New York J. Med.*, 1948, *48*, 875–876.
14. PROUT, G. R., JR., SLACK, N. H., and BROSS, I. D. Preoperative irradiation and cystectomy. IV. Results in a selected population. Seventh National Cancer Conference Proceedings, 1973, American Cancer Society.
15. REBOUL, J., VALLANGER, F., DELORME, G., TAVERNIER, J., and GEINDRE, M. Association de radiothérapie pré-operatoire et chirurgie dans le traitement du cancer primitif du rein de l'adulte. *Ann. de radiol.*, 1962, *5*, 283–288.
16. RICHES, E. W. Malignant disease of urinary tract. The Lettsomian Lectures. *Tr. Roy. Soc. Med.*, 1957–1958, *74*, 77–139.
17. VETERANS ADMINISTRATION COOPERATIVE UROLOGICAL RESEARCH GROUP. Treatment and survival of patients with cancer of prostate. *Surg., Gynec. & Obst.*, 1967, *124*, 1011–1017.
18. VICKERY, A. L., and KERR, W. S., JR. Carcinoma of prostate treated by radical prostatectomy: clinico-pathological survey of 187 cases followed for 5 years, and 148 cases followed for 10 years. *Cancer*, 1963, *16*, 1598–1608.
19. WATERS, C. A. Pre-operative irradiation of cortical renal tumors. AM. J. ROENTGENOL. & RAD. THERAPY, 1935, *33*, 149–164.
20. YOUNG, H. C. Cure of cancer of prostate by radical perineal prostatectomy (prostatoseminal vesiculectomy): history, literature and statistics of Young's operation. *J. Urol.*, 1945, *53*, 188–256.

Vol. 121, No. 3

MEGACALYCES: FURTHER OBSERVATIONS AND DIFFERENTIATION FROM OBSTRUCTIVE RENAL DISEASE*

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WE have previously published[5,12] the clinical, urographic, and angiographic findings in a small series of patients with megacalyces—a condition first described by Puigvert in 1963.[8–10] Within the past 18 months, we have studied 6 more patients with megacalyces, bringing our case number to 15.

We shall present our most recent material and discuss the role of radioisotope renal scanning in substantiating the diagnosis.

REPORT OF CASES

Case 1. P.V., an 8 year old boy, had renal angiography and renal scanning in September 1972, because of an abnormal excretory urogram. In 1970 he had left pyelonephritis manifested by fever, hematuria, and pyuria. He responded promptly to treatment with antibiotics and became asymptomatic. In 1971 he again had a urinary tract infection and responded promptly to treatment. Since that time the urine has been sterile. The 1972 laboratory values included blood urea nitrogen (BUN) 18 mg. per cent, creatinine 0.6 mg. per cent, and a normal urinalysis. A voiding cystourethrogram excluded vesicoureteral reflux. The excretory urogram (Fig. 1, *A* and *B*) showed dilated calyces on the left with no obstruction. There was no change in the general appearance since the 1971 urogram (Fig. 1*C*), but there had been equal growth in renal length bilaterally. Aortography and selective renal arteriography were performed in an attempt to assess the amount of cortical tissue in both kidneys (Fig. 1, *D–G*). On the basis of the arteriographic nephrogram, it was estimated that the amount of cortical tissue was the same on both sides. This was confirmed on renal scanning with Tc^{99m}-penicillamine complex (Tc^{99m}-Pen) (Fig. 1, *H* and *I*). A radioisotope renogram was also done with I^{131}-labeled hippuran (Fig. 1, *J* and *K*).

Comment. The dilated calyces in the left kidney predisposed to stasis and urinary infection. Although the urogram showed diminished thickness of the renal substance, the angiogram showed that the thinning was confined to the medulla, with the cortex thickness remaining normal. This qualitative estimate from the angiogram was confirmed by the renal scans.

Case 11. J.R., an 18 year old man, had a 1 year history of intermittent back pain and urinary infection. Urinalysis showed many red blood cells and 2 to 5 white blood cells per high power field, no bacteria, and no casts. Urine protein was 3 plus; specific gravity 1.025; urine culture was sterile. The BUN was 13 mg. per cent; creatinine 1.0 mg. per cent. Excretory urogram showed dilated right calyces and bilateral primary megaureter, more pronounced on the right (Fig. 2, *A* and *B*). Cystogram showed no reflux and a normal bladder. A retrograde pyelogram ruled out obstruction. Fluoroscopic examination of excretory urography showed normal peristalsis in the right upper urinary tract, but a "yo-yo" peristalsis related to the adynamic segment in the distal right ureter. Renal scanning with Tc^{99m}-Pen demonstrated approximately equal amounts of radioactivity in both kidneys at 1 hour (Fig. 2, *C* and *D*). The patient underwent resection of the distal portion of the right ureter with a ureteroneocystostomy. Postoperatively there was no change in the appearance of the upper urinary tract. Histologic examination of the ureter showed hypertrophy of muscle fibers and a great increase in the amount of fibrous tissue.

Comment. At first inspection it would be attractive to diagnose obstructive atrophy of the right kidney related to the primary megaureter. Renal scanning showed similar uptake of radio-

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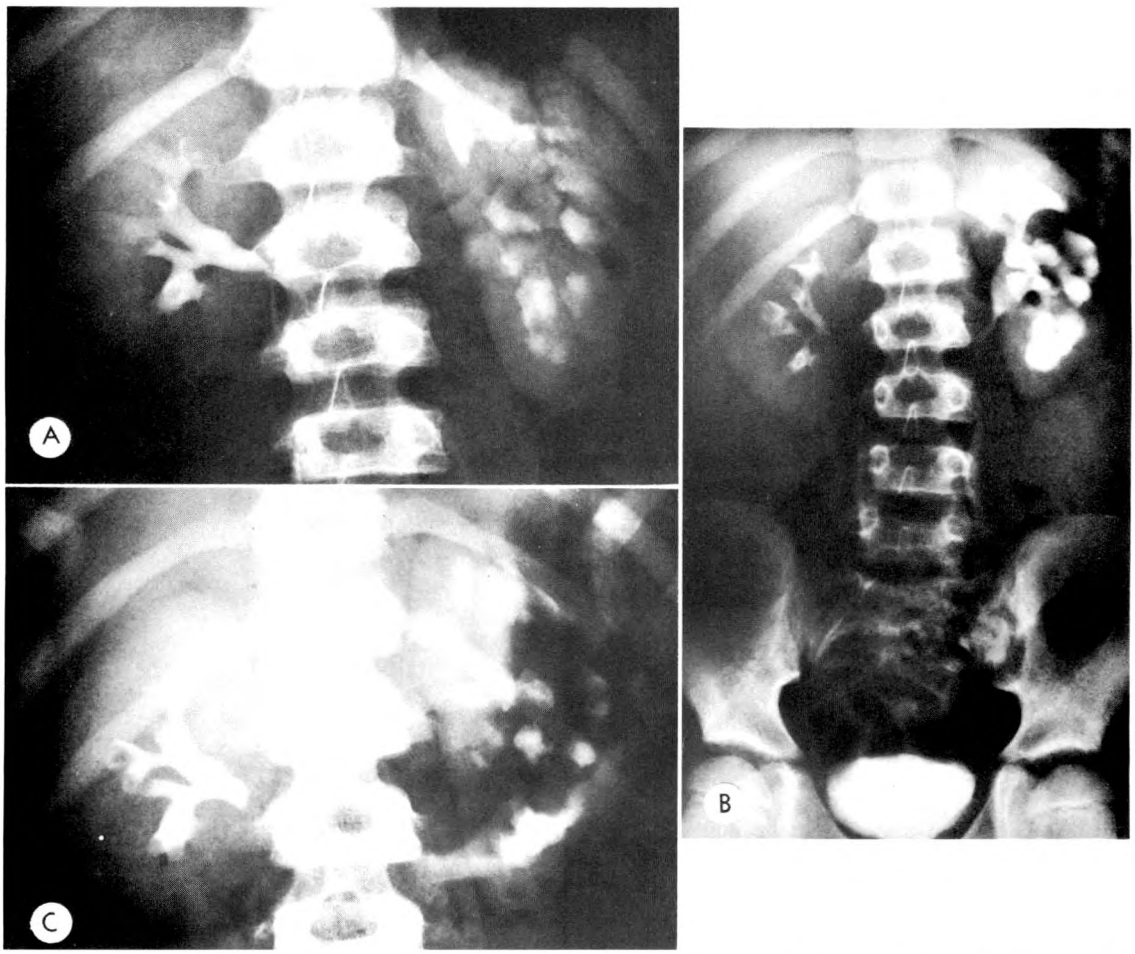


FIG. 1. Case 1. (*A*) Three minute urogram, September 1972. R = 8.6 cm., L = 9.7 cm. The right kidney and collecting system are normal. Only partial filling of the dilated left collecting system is present. (*B*) Ten minute urogram, September 1972. All left calyces are dilated with reduced parenchymal thickness. There is focal scar in lower pole, probably atrophic pyelonephritis. The pelvis and ureter are normal. Retrograde pyelogram and voiding cystourethrogram ruled out obstruction or reflux. (*C*) Six minute urogram, April 1971. R = 7.9 cm., L = 9.0 cm. Morphology of collecting systems and pelves are the same as in 1972. There is a similar delay in complete filling of the left side. Equal growth of the kidneys has occurred between 1971 and 1972.

activity in the 2 kidneys, indicating that there had been no significant reduction in the amount of functioning cortex on the right side compared to the left. It appears that the calyces are dilated on a developmental basis rather than secondary to obstruction. This association of megacalyces with primary megaureter has been noted previously.[14]

CASE III. G.J., a 52 year old man, was found to be hypertensive (160/110 mm. Hg) during an examination for an inguinal hernia. There was no history of urinary tract disease. Urinalysis was normal. BUN was 26 mg. per cent; creatinine was 1.0 mg. per cent. Excretory urography done as part of his work-up for hypertension showed megacalyces on the right side (Fig. 3, *A* and *B*). Renal scan with Tc^{99m}-Pen showed approximately equal amounts of functioning cortical tissue in both kidneys (Fig. 3, *C* and *D*). I^{131}-hippuran renogram showed a delayed excretory phase on the right side. The patient remains asymptomatic with respect to his urinary tract.

CASE IV. P.J., a 46 year old woman, has had chronic ulcerative colitis for approximately 10

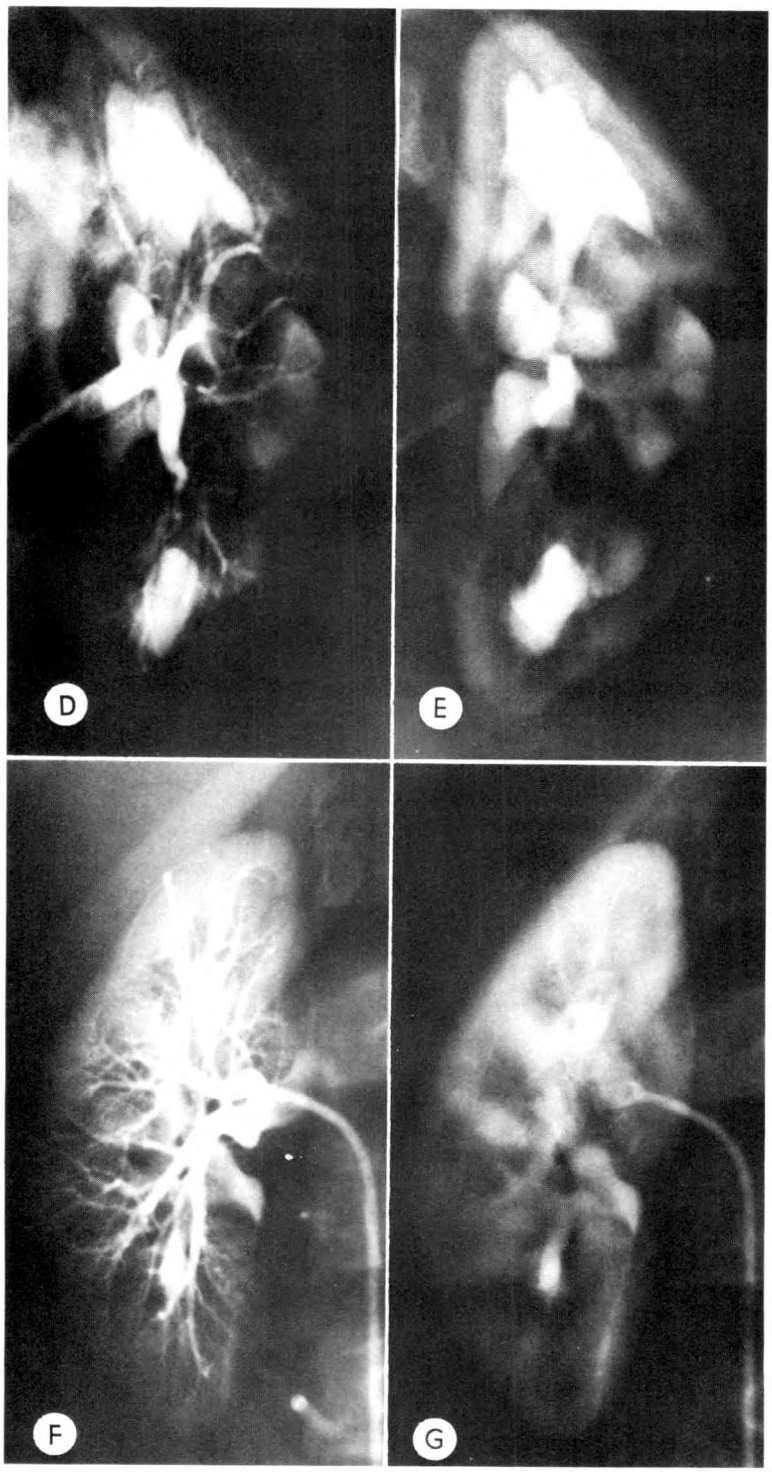
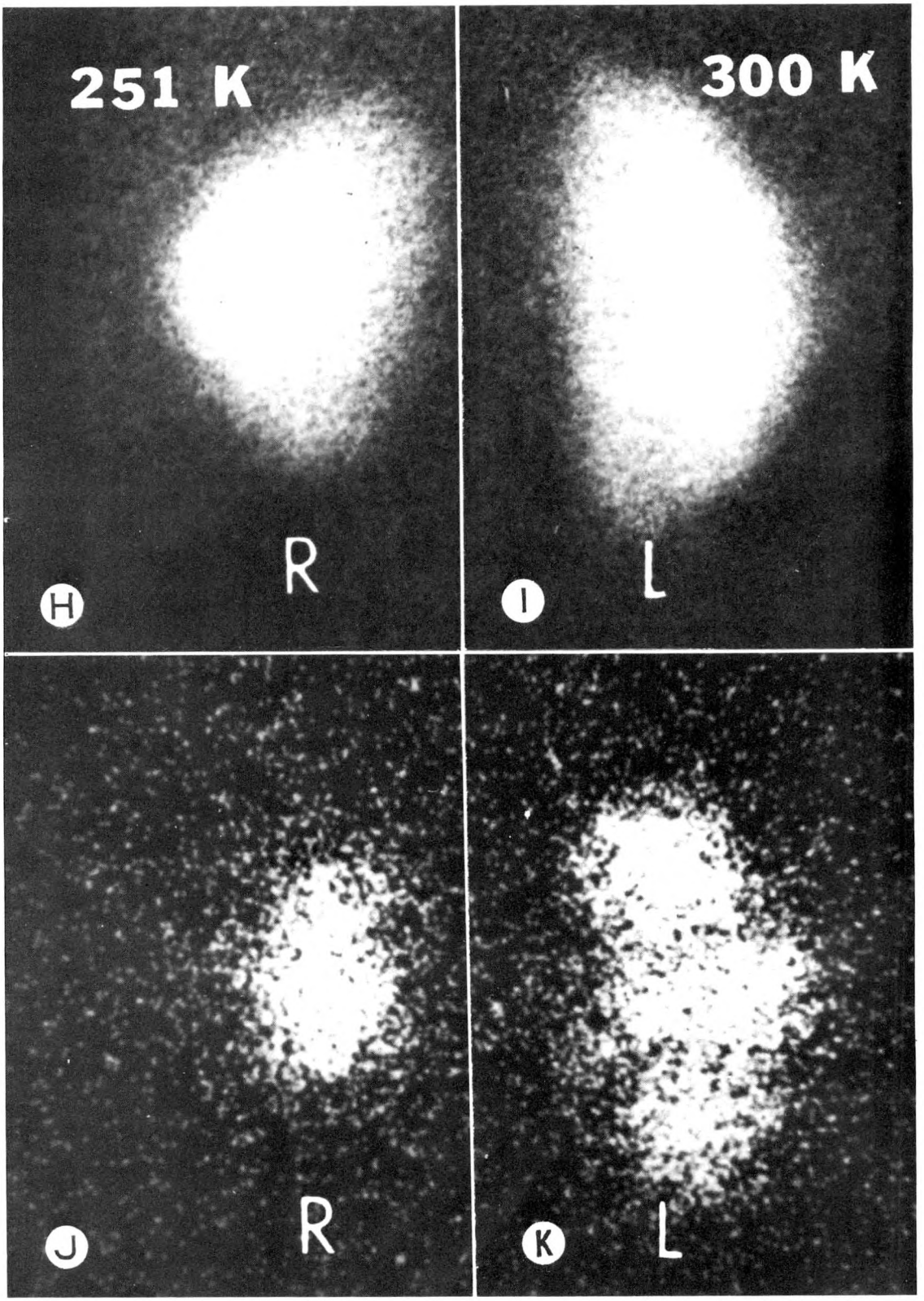


Fig. 1. (*D* and *E*) Left renal angiograms (LPO), early and late phase. There is mild draping of the intrarenal arteries around the enlarged calyces. Excellent demarcation between cortex and medulla is present. Except for a lower pole scar, the cortex measures normal in width. The medulla is thin. (*F* and *G*) Right renal angiograms (RPO), early and late phase. Normal renal angiograms. The thickness of the peripheral cortex and the interior columns of Bertin are easily measured. Although the left and right renal cortex measure approximately equal, the medullary regions on the left are substantially thinner than those on the right.


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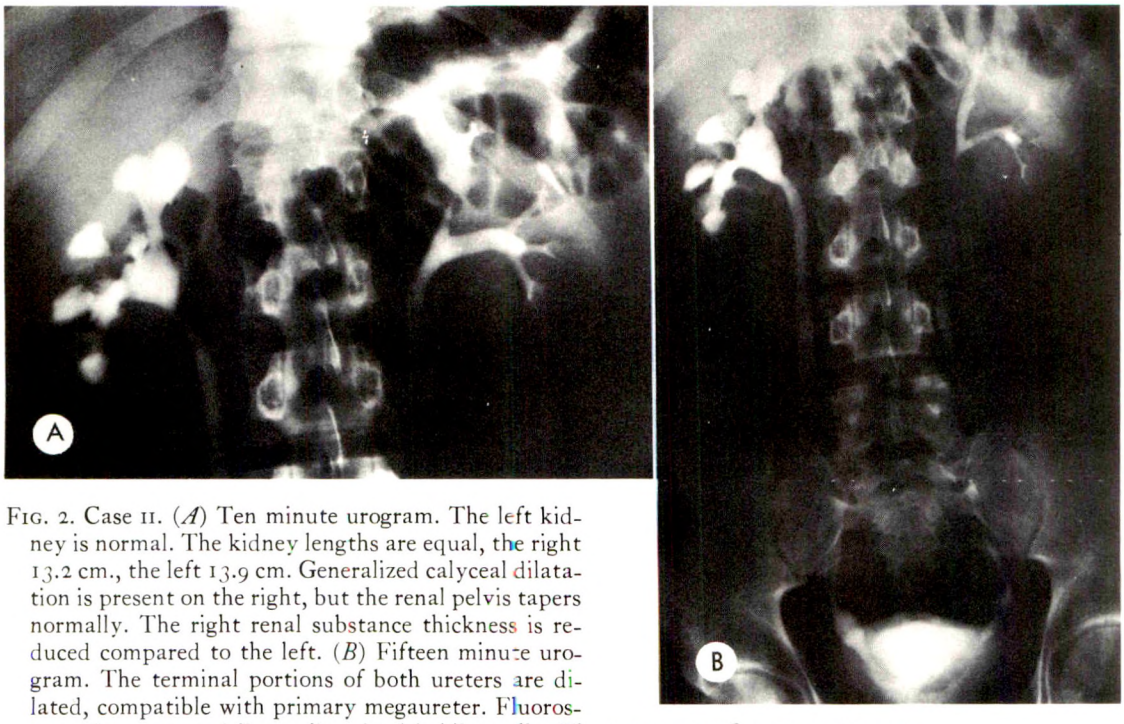


Fig. 2. Case 11. (*A*) Ten minute urogram. The left kidney is normal. The kidney lengths are equal, the right 13.2 cm., the left 13.9 cm. Generalized calyceal dilatation is present on the right, but the renal pelvis tapers normally. The right renal substance thickness is reduced compared to the left. (*B*) Fifteen minute urogram. The terminal portions of both ureters are dilated, compatible with primary megaureter. Fluoroscopy demonstrated "yo-yo" peristalsis bilaterally. There was no reflux on cystogram.

years. In 1967 she underwent a subtotal colectomy with ileostomy and shortly thereafter was found to have asymptomatic bilateral renal calculi (Fig. 4*A*). An excretory urogram (Fig. 4*B*) showed left megacalyces without evidence of obstruction. Over the ensuing 5 years the patient had recurrent urinary infections with *E. coli* and *Klebsiella*, and one episode of transient renal colic followed by the passage of a small stone. Her colitis remained quiescent. The bilateral calculi enlarged gradually, progressing to staghorn calculi (Fig. 4*C*), but the left collecting system did not change (Fig. 4*D*). Renal function remained normal with BUN 13 mg. per cent; creatinine from 0.8 to 1.2 mg. per cent. Bilateral, staged pyelolithotomies were performed. Biopsy of the right lower pole showed pyelonephritis with acute and chronic changes. The left upper pole wedge biopsy demonstrated a normal cortex without inflammation (Fig. 6*C*). There was a reduced width of medulla with a minimal chronic inflammatory reaction just beneath the calyx.

Comment. It is well known that patients with ulcerative colitis who have undergone a colectomy and ileostomy have a predisposition to stone formation due to chronic, mild acidosis with high urinary calcium excretion. The situation is compounded by a tendency on the part of the patient to avoid fluids because of the effects on ileostomy drainage. In view of the biopsy findings in the left kidney, it appears that the patient had congenital megacalyces on the left with subsequent development of stones due to her several metabolic problems. Without the benefit of the 1967 roentgenograms, it would have been reasonable to diagnose left postobstructive atrophy secondary to staghorn

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Fig. 1. (*H* and *I*) Renal scintiphotos 1 hour after Tc^{99m}-Pen injection. The numbers indicate the total counts from each kidney over 1 minute. Fifty-five per cent of the functioning renal tubules are on the *left* side. (*J* and *K*) I^{131}-hippuran renograms, 20 minutes after injection. The dilated left collecting system acts as a reservoir and accumulates more radioisotope than the right system. The early portions of the renogram curves were equal bilaterally, but the excretory phase was prolonged markedly on the left side.

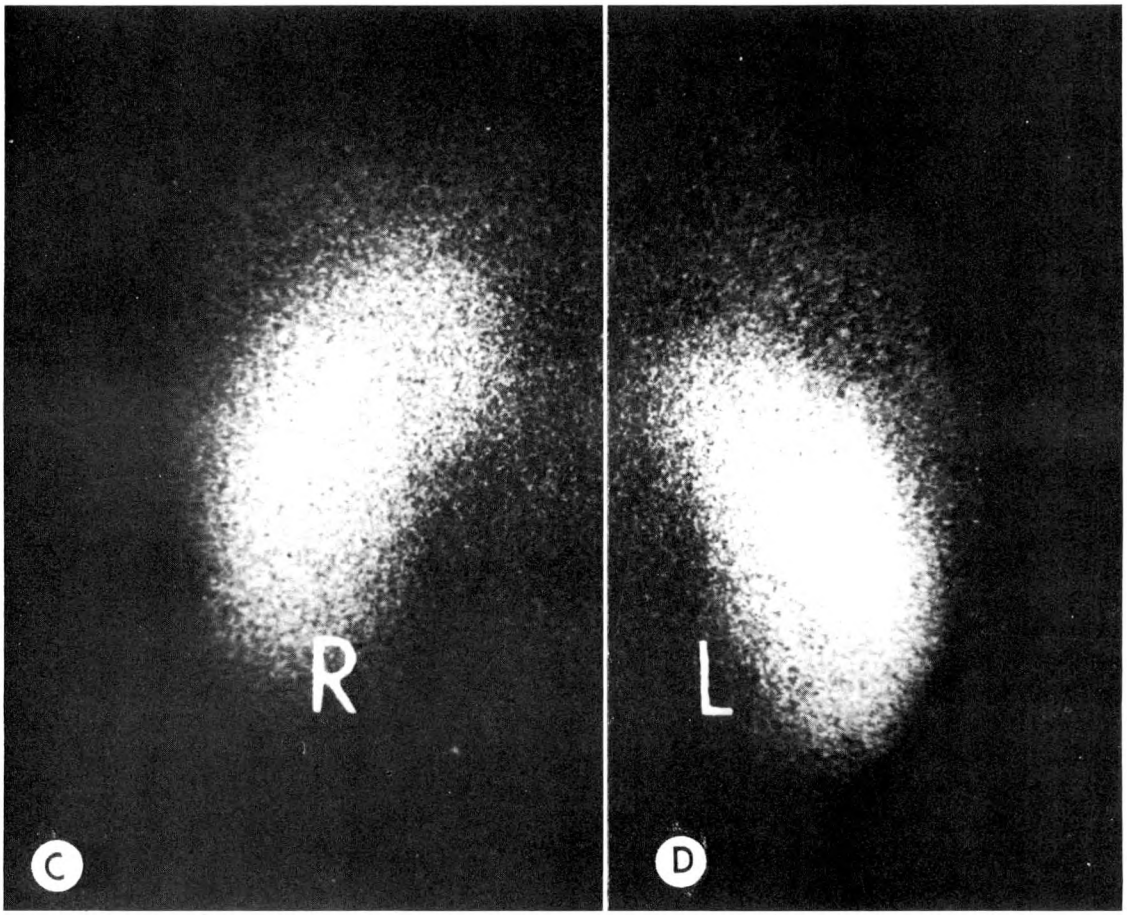


FIG. 2. (*C* and *D*) Renal scan taken 25 minutes after injection of Tc^{99m}-Pen. Counting with a divided crystal showed 53 per cent of the radioactivity in the right kidney, 47 per cent in the left. These measurements suggest equal amounts of functioning renal tissue on the 2 sides. The I^{131}-hippuran renogram (not shown) revealed normal and equal early portions of the curves, but a delayed excretory phase on the right, due to the reservoir effect of the large collecting system.

calculus, but the early roentgenograms showed that the calyceal enlargement *preceded* the development of the staghorn calculus.

CASE V. M.C. was first evaluated at the age of 17 years in 1970 because of hematuria. His case history, laboratory and radiographic findings have been published previously.[12] In brief, he was found to have normal creatinine clearance and a maximum concentrating ability of 943 mosm per kg. (normal for this age should be >1000 mosm per kg.). The kidneys were normal in size and contour, but there were bilateral megacalyces. Angiography showed normal intrarenal arteries and a normally thick cortex (Fig. 5). Renal venography demonstrated displacement of interlobar veins by the large calyces. No bleeding source was discovered.

He was treated conservatively for the next 2 years, but because of persistent hematuria he underwent a right renal exploration in 1972. Intraoperative nephroscopy showed bleeding from the right lower pole, and the lower pole of the kidney was resected. A small capillary vascular malformation was discovered adjacent to one of the megacalyces. Detailed examination of the resected kidney tissue showed perfectly normal cortex and thin medulla. There was no inflammatory reaction and no evidence of previous infection (Fig. 6*A*). The patient has had no further hematuria since surgery.

Comment. This patient with bilateral megacalyces afforded us an excellent opportunity to

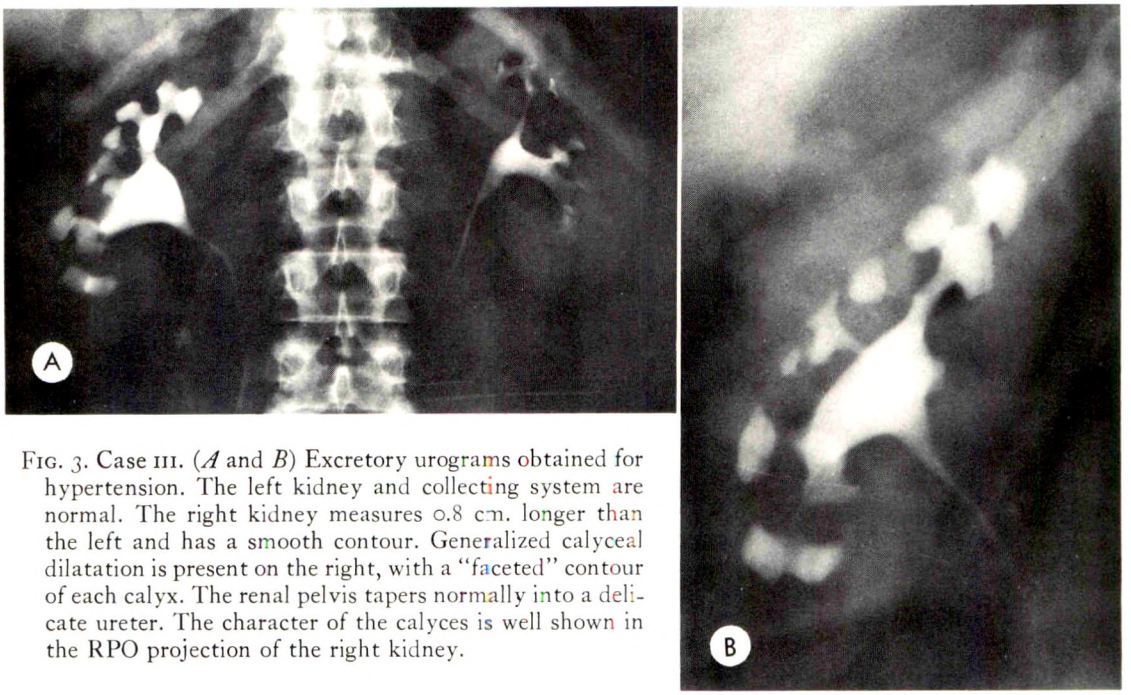


Fig. 3. Case III. (*A* and *B*) Excretory urograms obtained for hypertension. The left kidney and collecting system are normal. The right kidney measures 0.8 cm. longer than the left and has a smooth contour. Generalized calyceal dilatation is present on the right, with a "faceted" contour of each calyx. The renal pelvis tapers normally into a delicate ureter. The character of the calyces is well shown in the RPO projection of the right kidney.

examine the effects of this malformation, in its uncomplicated form, upon renal function. His creatinine clearance was normal, but there was mild interference with the renal concentrating mechanism. What appeared to be normal cortex on angiography in 1970 was confirmed by the histologic examination in 1972.

DISCUSSION

It has become apparent that megacalyces is not rare since, in a little over 3 years, we have come upon 15 patients in whom the diagnosis is reasonably certain. About half of these patients were seen at our hospital and had extensive investigations. The other patients had their records and roentgenograms referred to us because of our known interest in this condition. An abbreviated summary of the clinical data on all the patients is given in Table 1.

UROGRAPHY

Although Anselmo and Cosciani-Cunico[2] found megacalyces bilaterally in 80 per cent of their 30 patients, our experience and that of Puigvert[10] has been more commonly unilateral. Only 2 of our 15 patients had bilateral megacalyces. The findings on excretory urography are consistent from patient to patient. The appearance time of contrast material in the calyces of both kidneys is equal, whether there is unilateral or bilateral involvement. Complete and maximum opacification of the collecting system and pelvis is delayed to 15 or 30 minutes because of the large volume of urine which must be displaced. The renal pelvis is normal in configuration. The number of calyces may be normal or may be increased to as many as 20 or 25. In several unilateral cases the renal length on the involved side has been increased. Renal parenchymal thickness is reduced when compared to normal kidneys, due to the increased calyceal volume and thinned medulla. The calyceal pattern has been variously described as "mosaic" or "faceted"[10] because of the polygonal contour of each calyx. The tips of the papillae either are not visualized at all or are extremely shallow if present. Fluoroscopy during urographic examination has shown very little, if any, change in size of the calyces or their infundibula. Renal pelvic contractions appear normal, but are infrequent (1–3 per minute). The ureters are normal in course and caliber except when there is an associated

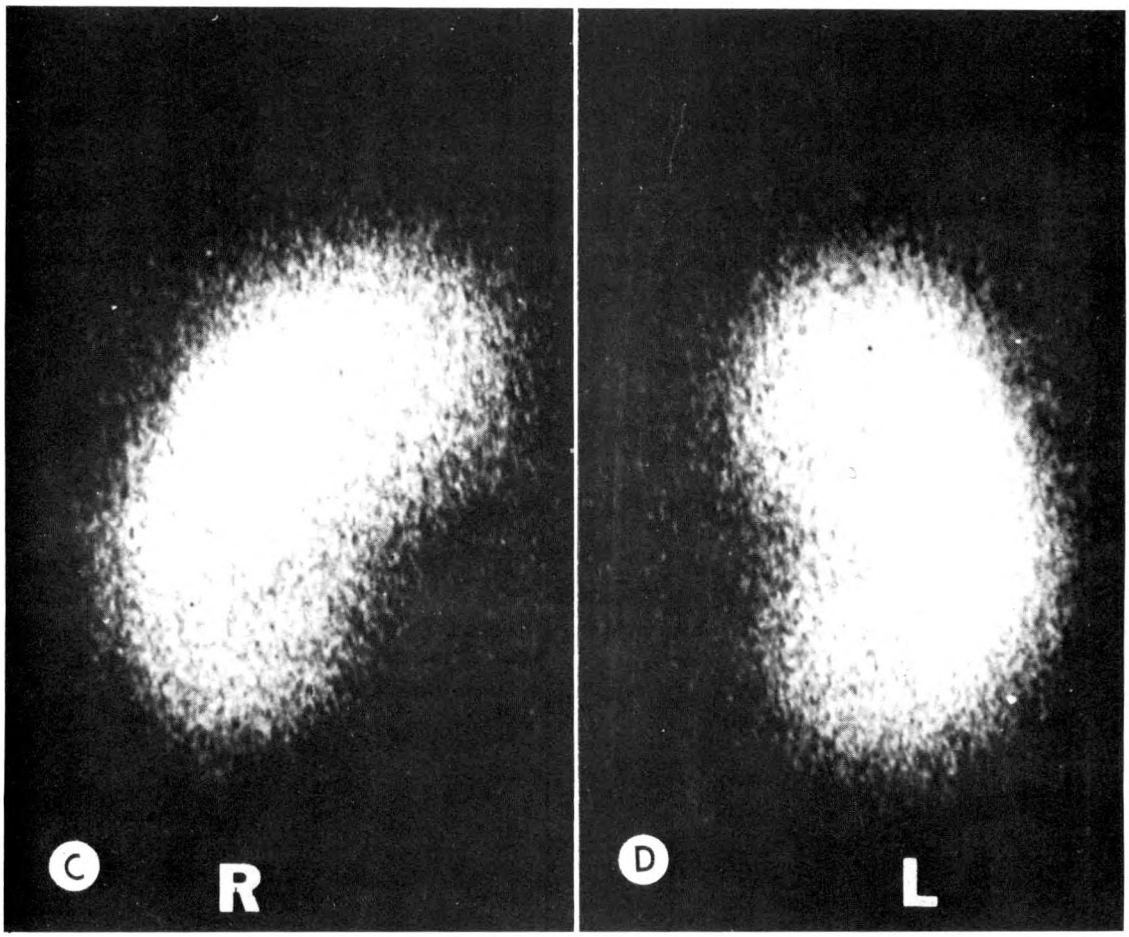


FIG. 3. (*C* and *D*) Renal scintiphotos (supine) 2 hours after Tc^{99m}-Pen injection. Even at this time, the amount of radioisotope in the collecting systems is negligible compared with that in the parenchyma. Divided crystal counting showed 52 per cent of the activity in the left kidney, 48 per cent in the right. I^{131}-hippuran renogram (not shown) as in Case II, revealed normal and equal early portions of the curves with a delayed and prolonged excretory phase on the right side.

primary megaureter involving the distal segment.[14]

The enlarged, floppy calyces produce stasis of urine, predisposing to stone formation and/or infection. Once renal calculi develop, the urographic findings may alter, especially if there is a superimposed episode of acute ureteral obstruction due to the lodgement of a calculus.

The differentiation between megacalyces and acquired calyceal dilatation is not difficult as a rule. Intrarenal hydronephrosis, sometimes termed hydrocalycosis, may result from diseases which scar the infundibula. Tuberculosis or staghorn calculus with infection are examples. The infundibula in kidneys with megacalyces are wide open and therefore should not lead to confusion with such cicatricial diseases. Atrophic pyelonephritis may cause clubbing of most or all of the calyces, but the presence of coarse and focal renal scarring in that disease distinguishes it from megacalyces where the renal outlines are smooth. Widespread papillary necrosis may lead to grossly blunted calyces, but they are never as symmetrically enlarged as is seen in megacalyces. Postobstructive atrophy or back pressure atrophy due to reflux are the 2 entities which deserve serious consideration when one is faced with the urographic findings indicated above. While the latter

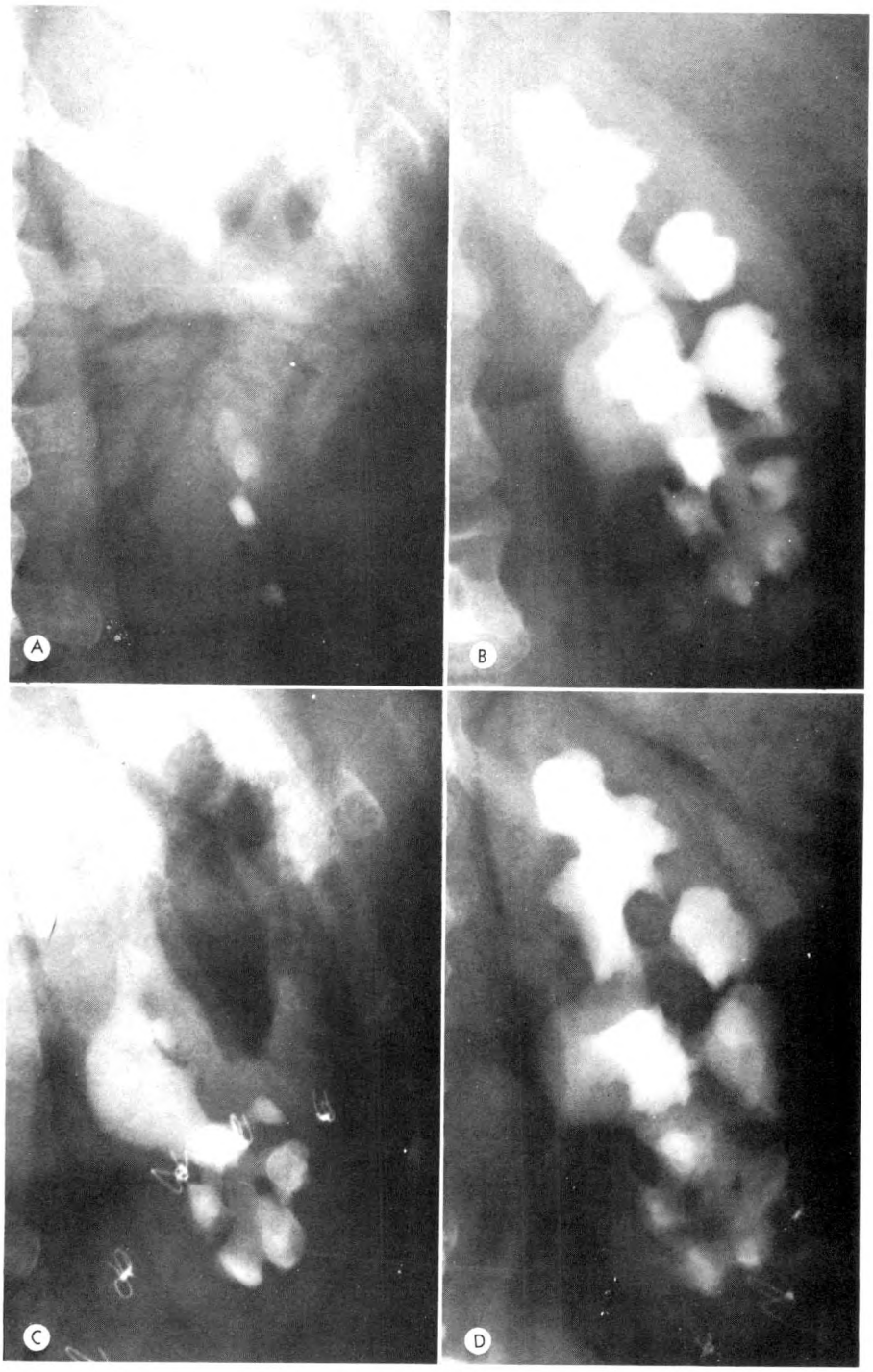


FIG. 4. Case IV. (*A*) Plain film roentgenogram of the left kidney in 1967 shows several small calculi in the lower pole. (*B*) Excretory urogram in 1967 demonstrates generalized calyceal enlargement on the left. The renal pelvis is normal and there is no ureteral obstruction. Renal size is normal and no roentgenographic scarring is evident. (*C*) Plain film roentgenogram of the left kidney in 1972 shows a staghorn calculus involving almost the entire collecting system and pelvis. (*D*) Excretory urogram in 1972 shows no change in the caliber of the collecting system over the 5 year period. The renal contour remains smooth. Biopsy was taken from the left upper pole (Fig. 6*C*).



FIG. 5. Case v. Nephrogram phase of selective left ventral renal arteriography, LPO projection. The cortex measures over 1 cm. thick and the columns of Bertin are well demonstrated. The thin medulla is easily visible between the contrast-filled calyces and the opacified cortex. Histologically the kidney tissue was completely normal (Fig. 6*A*).

can be excluded easily with a cystogram, the former can be excluded only by using one or more of the examinations to be subsequently discussed.

ANGIOGRAPHY

Since there is no way to determine from the urogram whether the reduced renal substance thickness is a manifestation of thinning of the cortex, medulla, or a combination of the two, the angiographic nephrogram was explored in an effort to sort out this problem. Angiography has been performed in 4 patients with megacalyces and in each instance there has been extremely sharp demarcation between the cortex and medulla, permitting an assessment of cortical thickness. In all cases the cortex measured well within or over the normal range (0.5–0.8 mm.).[1] Therefore the reduced parenchymal thickness occurs solely at the expense of the medulla (Fig. 7, *A–C*). Normal cortical thickness in these patients correlates well with their normal measurements of creatinine clearance. The main renal artery, the interlobar, and arcuate arteries are normal except for minimal draping about some of the dilated calyces. The interlobular arteries are strikingly normal in contrast to the attenuation of these arteries seen in kidneys with postobstructive atrophy.[11]

RADIOISOTOPE STUDIES

The only way to be certain one is dealing with megacalyces rather than a postobstructive or otherwise damaged kidney is to measure individual renal function. Postobstructive atrophy is characterized by permanent loss of nephrons and impairment of maximum renal concentrating ability.[7] As indicated above, renal angiography can provide a semiquantitative estimate of the amount of renal cortex on the 2 sides. Ureteral catheterization for split renal function measurements is tedious and may be hazardous in these patients with urinary stasis, and it cannot be easily justified for diagnostic purposes. We have recently found that renal scanning with a new agent, technetium 99m-penicillamine complex, is useful for estimating the amount of functioning tissue on either side. This agent, initially developed by Halpern *et al.*[6] as Tc^{99m}-penicillamine-acetazolamide complex (Tc^{99m}-Pac), localizes in the renal cortex, presumably being bound to proximal tubule cells. Less than 10 per cent of the injected radiopharmaceutical is excreted in the urine within the first 3 hours.[13] Because of the short half-life of Tc^{99m}, relatively large amounts of the agent can be

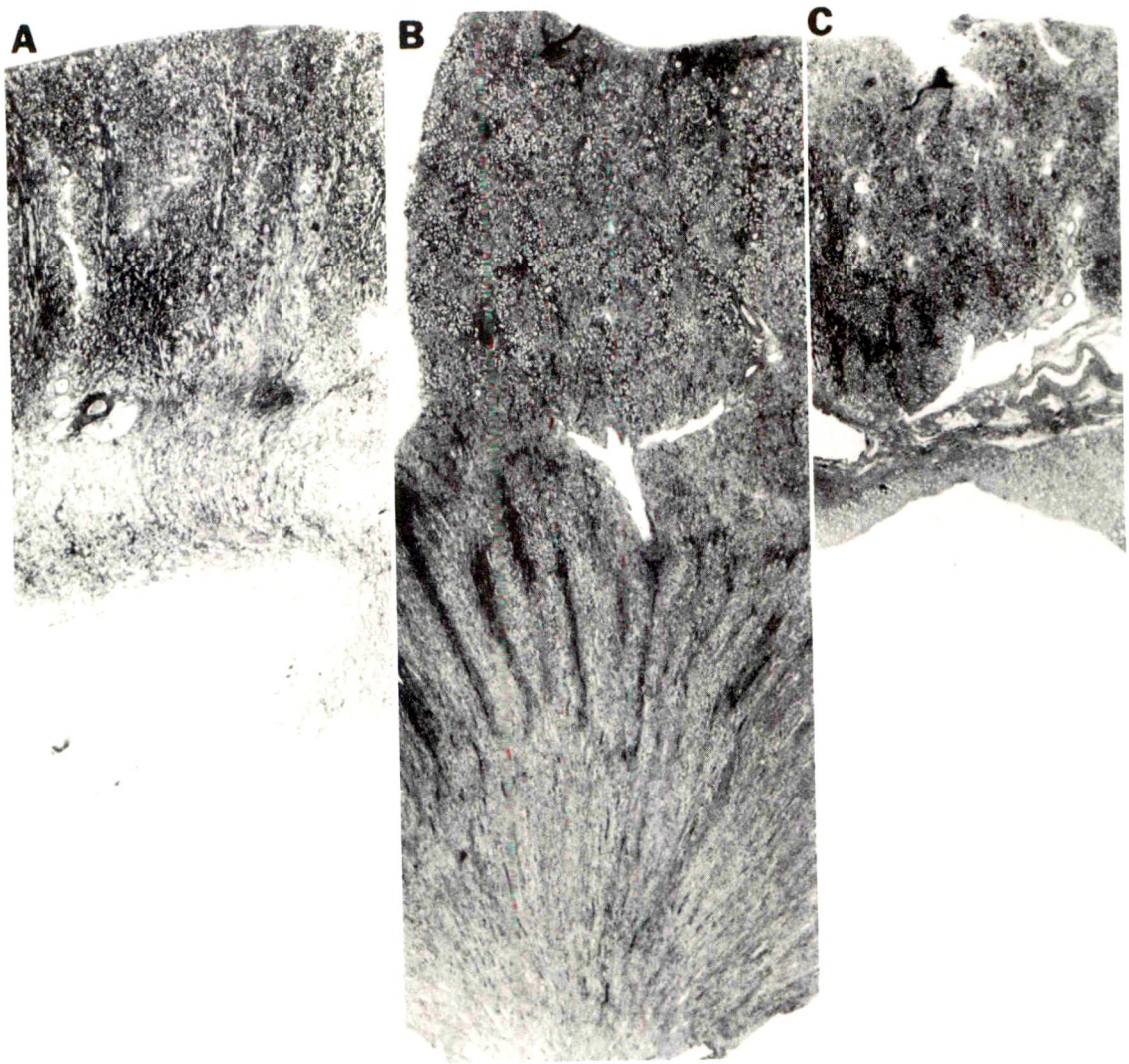


Fig. 6. Kidney tissue from (*A*) Case v and (*C*) Case iv, compared with (*B*) normal renal tissue. All are shown at the same low power magnification. Each specimen has a normal cortex. Note the extreme thinning of the medulla in *A* and *C*, compared to the normal medulla in *B*.

administered without significant radiation hazard. Renal scintiphotos (supine, posterior) obtained 1–3 hours after the injection are of excellent quality, and the number of counts from each kidney can be recorded with split crystal counting. Characteristically, no visualization of the collecting system, pelvis, or ureter is present on these scintiphotos. We have obtained Tc^{99m}-Pen renal scans in 4 patients with unilateral megacalyces, and in each instance have found the amount of radioactivity to be very nearly equal on the 2 sides. Kidneys with postobstructive atrophy or other forms of renal damage leading to nephron loss, are expected to show diminished accumulation of radioisotope compared to the normal side.[6]

The normal Tc^{99m}-Pen scans stand in contrast to the I^{131}-hippuran renograms on the same patients. In the latter, there is a marked prolongation of the excretory phase on the affected side, simulating ureteral obstruction. This appearance, of course, results from the gradual mixing of radioisotope with the large volume of urine in the

L BRARY

TABLE I

CLINICAL DATA ON PATIENTS WITH MEGACALYCES

Patient	Age	Sex	Presentation	Side	Angiogram	Scan	Duration of Roentgenographic Follow-up
L.W.	45	M	Hypertension	Left; also primary left megaureter	−	−	<1 yr.
G.J.	52	M	Hypertension	Right	−	+	<1 yr.
J.R.	18	M	Urinary infection	Right; also primary right megaureter	−	+	2 yr.
T.B.	27	M	Calculi (left)	Left; polycalyces	−	−	3 yr.
R.S.	33	M	Calculi (bilateral)	Bilateral; also left upper pole medullary sponge	+	−	12 yr.
M.C.	17	M	Hematuria; mild hypertension	Bilateral	+	−	3 yr.
R.H.	53	M	Infection	Right (had previous left nephrectomy for "hydronephrosis")	−	−	18 yr.
J.E.	48	M	Hypertension	Left	+	−	2 yr.
R.B.	38	M	Calculi (right); mild hypertension	Right	−	−	3 yr.
J.H.	20	M	Calculus (left)	Left	−	−	1 yr.
R.S.	33	M	Hypertension, calculi hypercalcemia	Left	+	−	2 yr.
L.S.	19	M	Fever unknown origin	Left	−	−	<1 yr.
P.V.	8	M	Urinary infection	Left	+	+	2 yr.
R.C.	22	M	Calculi (bilateral) with acute obstruction on right	Left; ?right	−	−	1 yr.
P.J.	46	F	Calculi (bilateral), ulcerative colitis, and urinary infection	Left	−	+	6 yr.

expanded collecting system, and the slow drainage which follows.

RENAL FUNCTION

Serum creatinine has been normal in all our patients and creatinine clearance has been normal in those few in whom this test has been performed. Maximum renal concentrating ability, measured by urine osmolality after fluid deprivation and vasopressin administration, was mildly reduced in our Case V (943 mosm per kg.) with bilateral megacalyces. In 2 other patients in whom concentrating ability was measured there was a moderate concentrating defect, but both of these patients had calculus formation and urinary infection. Renal infection *per se*, as well as postobstructive atrophy can diminish the ability to concentrate the urine.

PATHOGENESIS

It is likely that megacalyces is a congenital defect, but this remains unproven. Puigvert[9,10] has suggested that there is fault in embryology which occurs during the early divisions of the ureteral bud when it is joining with the metanephric blastema. Other possibilities are a temporary intrauterine obstruction or a maldevelopment of the pericalyceal muscles. Galian *et al.*[4] found a decreased number of juxtamedullary glomeruli in a kidney with megacalyces and theorized that the condition is due to a primary hypoplasia of these glomeruli, which would explain the mild

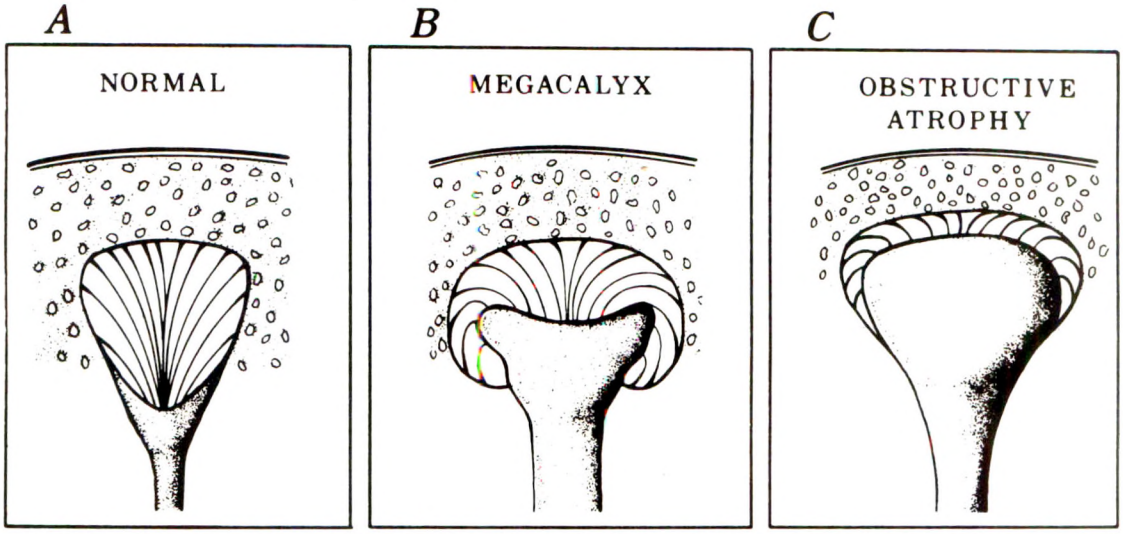


Fig. 7. Diagram of calyx and adjacent renal parenchyma in: (*A*) normal kidney, (*B*) kidney with megacalyx, and (*C*) kidney with obstructive atrophy. In the normal kidney the ratio between cortical thickness and medullary thickness (C:M) measured to the tip of the papilla is approximately 1:2 (range 1:1.5 to 1:2.5). In the kidney with megacalyces the medulla is thin but the cortex remains normal, resulting in a change of the C:M ratio to 1:1 or greater. In the kidney with postobstructive atrophy there is thinning of both cortex and medulla, the ratio between the 2 varying with the extent of atrophy. The parenchymal thickness (distance from calyx to edge of kidney) is reduced both in postobstructive atrophy and in megacalyces. In the former, parenchyma is reduced at the expense of both cortex and medulla, while in the latter only the medulla is thin.

concentrating defect exhibited by these patients.

We have continued to find radiologists and urologists who, while recognizing the appearance of nonobstructed kidneys with large calyces, prefer to think of them as being the result of some previous episode of obstruction or medullary necrosis, perhaps in infancy. While this can never be excluded with certainty, the preservation of renal cortex in these patients appears to contradict this theory. Furthermore, early episodes of medullary necrosis as recently described by Chrispin[3] would be expected to lead to coarse renal scarring.

CLINICAL OBSERVATIONS

The complications of urinary stasis, *i.e.*, stone formation and infection, have usually brought the patient with megacalyces to medical attention and subsequent urography. Six of our 15 patients presented with renal calculi and 3 presented with urinary tract infection. Five patients had moderate hypertension and 1 presented with hematuria. Our youngest patient was 8 years of age, the oldest 52. Fourteen of our patients were male. Unilateral megacalyces was found as an incidental finding in 5 patients, 4 of whom were being evaluated for mild hypertension and the fifth for a fever of unknown origin. Although the urographic findings should suggest the diagnosis, it should be evident that other studies must be done before the diagnosis can be claimed with confidence. Vesicoureteral reflux must be excluded with a voiding cystourethrography. Serum creatinine and creatinine clearance should be normal. A semiquantitative or quantitative estimate of unilateral functioning renal mass should be obtained. Our present policy is to perform a renal scan with Tc^{99m}-Pen for this purpose. Only when there is no evidence of nephron loss on the side with large calyces are we satisfied with the diagnosis. Follow-up examinations are useful in that they demonstrate no change in the urographic appearance of the

adult kidney with megacalyces. Children with megacalyces should manifest normal renal growth based on our experience in 1 patient.

The management of the patient with megacalyces is conservative, and surgical therapy enters the picture only when there is calculus disease or uncontrollable hematuria. Treatment should be directed at controlling urinary tract infections with appropriate antibiotics and attempting to minimize calculus formation with large fluid intake.

SUMMARY

Megacalyces is a nonobstructive enlargement of the calyces due to a malformation of the renal papillae. The renal cortex is normal but the medulla is hypoplastic, leading to a variable concentrating defect.

Calyceal urinary stasis predisposes to stone formation and/or infection.

The dilated calyces and reduced parenchymal thickness may resemble obstructive hydronephrosis or postobstructive atrophy, but the urographic and angiographic features of megacalyces are sufficiently distinctive for the correct diagnosis to be suggested in most cases.

Renal function tests and radioisotope studies are important in substantiating the diagnosis.

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REFERENCES

1. ABRAMS, H. L. Quantitative derivates of renal radiologic studies. *Invest. Radiol.*, 1972, *7*, 240–279.
2. ANSELMO, G., and COSCIANI-CUNICO, S. Le displasie megacalicali. *Urologia (Treviso)*, 1965, *32*, 376–386.
3. CHRISPIN, A. R. Medullary necrosis in infancy. *Brit. M. Bull.*, 1972, *28*, 233–236.
4. GALIAN, P., FOREST, M., and ABOULKER, P. La megacalicose. *Presse méd.*, 1970, *78*, 1663–1667.
5. GITTES, R. F., and TALNER, L. B. Congenital megacalices versus obstructive hydronephrosis. *J. Urol.*, 1972, *108*, 833–836.
6. HALPERN, S., TUBIS, M., GOLDEN, M., KUNSA, J., ENDOW, J., and WALSH, C. ^{99m}T-Pac, new renal scanning agent. II. Evaluation in humans. *J. Nuclear Med.*, 1972, *13*, 723–728.
7. HODSON, C. J. Post-obstructive renal atrophy (nephropathy). *Brit. M. Bull.*, 1972, *28*, 237–240.
8. PUIGVERT, A. Megacalicosis: diagnostico diferencial con la hidrocaliectasia. *Med. Clin.*, 1963, *41*, 294–302.
9. PUIGVERT, A. Interpretación etiopatogenica de las dilataciones calicilares. *Clin. Lab.*, 1964, *77*, 81–84.
10. PUIGVERT, A. Malformaciones de la Pirámide renal de Malpighi. Editorial Eco. S.A., Barcelona, 1965.
11. SIEGELMAN, S. S., and BOSNIAK, M. A. Renal arteriography in hydronephrosis: its value in diagnosis and management. *Radiology*, 1965, *85*, 609–616.
12. TALNER, L. B., and GITTES, R. F. Megacalyces. *Clin. Radiol.*, 1972, *23*, 355–361.
13. TALNER, L. B., and SOKOLOFF, J. Unpublished data.
14. VON NIEDERHÄUSERN, W., and TUCHSCHMID, D. Une association mal connue: le megauretère congénital primaire et le rein à mega-calices. *Ann. Urol.*, 1971, *5*, 225–231.

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ROENTGENOGRAPHIC FINDINGS IN URINARY TRACT TUBERCULOSIS*

A 10 YEAR REVIEW

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THE morbidity and mortality associated with urinary tract tuberculosis have been reduced significantly in recent years,[6,13,14] but the disease remains of concern to both practicing clinicians and public health officials. Although better controlled and less common today than formerly, renal tuberculosis continues to present a diagnostic problem, with the key role often played by the radiologist. In all cases, urography is essential to assess the extent and severity of involvement, to monitor the effects of treatment, and to discover the complications.[22]

This study was undertaken to review the clinical and roentgenographic findings in patients with urinary tract tuberculosis seen at the Mayo Clinic during the past decade and to evaluate the usefulness of ureteral compression and routine tomography in the demonstration of minimal renal changes.

MATERIAL AND METHOD

The histories and excretory urograms of 160 patients with urinary tract tuberculosis who were seen at the Mayo Clinic from 1962 to 1973 were reviewed. In 27 cases, the diagnosis had been made elsewhere. These patients had undergone nephrectomy, and when seen at our clinic, all had negative urine cultures and normal-appearing solitary kidneys. These 27 patients and 35 patients whose urograms had been discarded were excluded from the study group. Clinical histories and excretory urograms of the remaining 98 patients were studied.

Our current routine technique of excretory urography employs a 50 ml. bolus injection of urographic contrast medium. When indicated, larger doses are administered by infusion. For the past 3 years, linear tomography has been regularly used during excretory urography. Prior to that time, tomography was done only when it was indicated by the supervising radiologist. Ureteral compression has been employed routinely during the decade under study.

CLINICAL HISTORY

Urinary tract tuberculosis is primarily a disease of adults, affecting males more frequently than females. Of 98 patients included in our study group, 61 were males and 37 were females. Although 80 per cent of the 700 patients with urinary tract tuberculosis whose histories were reviewed in 1956 were between 16 and 40 years old,[3] today the disease occurs more frequently in an older age group.[4,20] The ages at diagnosis of tuberculosis involving any part of the body and the ages at onset of genitourinary involvement in these 98 patients varied (Table 1). When first examined at our institution, almost 80 per cent of the patients were 60 years old or older, but their medical histories revealed that the clinical onset of their genitourinary disease generally occurred at an earlier age. In 60 per cent of the cases, however, this was after the age of 40 years.

Genitourinary involvement was preceded in 27 cases by an earlier history of clinically apparent tuberculosis involving another part of the body. The most common site in our series was the skeletal system (17 patients). Although more than half the pa-

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From the Mayo Clinic and Mayo Foundation.

TABLE I

AGE OF PATIENTS WITH URINARY TRACT TUBERCULOSIS AT DIAGNOSIS OF TUBERCULOSIS

Age at Diagnosis (yr.)	Tuberculosis	
	Any Form	Genitourinary
0–9	4	0
10–19	9	4
20–29	20	15
30–39	15	19
40–49	22	30
50–59	12	12
60–69	14	16
70–79	2	2
Total	98*	98*

* Males, 61; females, 37.

tients had roentgenographic evidence of prior or current pulmonary tuberculosis, only 13 patients had corresponding clinical symptoms. Five patients gave histories of both pulmonary and skeletal tuberculosis.

Other related clinical findings included nonspecific systemic symptoms such as fever, anorexia, fatigue, or weakness (41 patients). Six patients were diabetic, and the diabetes was exacerbated by the tuberculous infection in 2. One patient with Addison's disease developed renal tuberculosis after 20 years of steroid treatment.

Of the 98 patients, 35 were either foreign-born or had spent a significant period of their life in other countries (Table II); 11 patients had been captives in concentration camps during World War II. Seventeen patients had a proved family history of tuberculosis.

Ninety-five of the 98 patients had histories of urinary tract symptoms; in some, the symptoms were very mild, but in others, they were severely disabling (Table III). The most common urinary tract symptoms were dysuria, frequency, and nocturia. Most patients had multiple symptoms. Many had received several unsuccessful courses of treatment with antibiotics for nontuberculous infections before the tuberculosis was diagnosed. Only 3 patients did not have urinary tract symptoms. The urograms of all 3 patients revealed unsuspected, calcified, nonfunctioning kidneys—examples of the so-called silent autonephrectomy.

CLINICAL AND LABORATORY FINDINGS

Urine cultures from 89 patients were positive for *Mycobacterium tuberculosis*, confirming the clinical diagnosis. Surgical specimens obtained in the other 9 cases all showed pathologic evidence of tuberculosis, and in 6 of these, cultures of the specimens were positive. Prior to being seen at the Mayo Clinic, 31 patients had been treated medically for tuberculosis, and 19 were currently taking antituberculous medication. Cultures were positive in 14 of the 31 patients who had previously received anti-

TABLE II

COUNTRIES OF BIRTH OR OF PROLONGED FOREIGN TRAVEL OF PATIENTS WITH URINARY TRACT TUBERCULOSIS

	No. of Patients
Europe	
Germany	5
Italy	4
Greece	2
Spain	2
England	1
Poland	1
Latvia	2
Hungary	1
Czechoslovakia	1
Romania	1
Austria	1
Yugoslavia	1
Asia and Far East	
Russia	2
China	1
South Pacific (WW II)	1
South America	
Venezuela	2
Colombia	1
Peru	1
North America	
Canada	3
Mexico	2
Total	35

tuberculous treatment and in 8 of the 19 patients currently under such treatment. Routine blood cell counts, sedimentation rates, and other laboratory studies were of little diagnostic help. Although not specific for tuberculosis, urinary findings were positive in almost all cases (Table IV).

Chest roentgenograms revealed positive findings in only 52 of the 98 patients. Forty-five patients with roentgenographic evidence of pulmonary tuberculosis had apical fibrocalcific disease or calcified granulomata, but evidence of an infiltrate or cavity disease was seen in only 7 patients. Only 31 of the 52 patients with positive chest roentgenograms had sputum or gastric washings cultured for tubercle bacilli, and of these, 4 had positive findings. Seven patients with normal chest roentgenograms had similar cultures taken, and all 7 had normal findings. The tuberculin reaction was positive in all 34 patients who were so tested.

Cystoscopy was performed in 46 patients, and 39 had positive findings—most had multiple findings (Table V). The most common findings were those of mucosal inflammation or nonspecific areal cystitis (27 patients). Mucosal ulcerations were seen in 12 patients, and delta-shaped areas of redness were seen in 8 patients and were associated with other findings in all but 2 of the 8. Nine patients had granulomatous mucosal changes. Twelve patients had contracted bladders of limited capacity. Eight patients had scarring about the ureteric orifice, resulting in a constricted orifice in 4 and a dilated "golf-hole" ureteric orifice in the other 4.

TABLE III

CLINICAL FINDINGS IN 95 PATIENTS WITH GENITOURINARY SYMPTOMS

Symptoms	No. of Patients*
Urinary tract	
Dysuria	53
Frequency	41
Nocturia	36
Gross hematuria	25
Cloudy urine (pyuria, tissue fragments)	25
Urgency	20
Suprapubic pain	5
Total incontinence	2
Epididymitis	12
Draining scrotal fistula	1

* Most had multiple symptoms.

TABLE IV

LABORATORY FINDINGS IN 98 PATIENTS WITH URINARY TRACT TUBERCULOSIS

	Range	Mean
Blood		
Sedimentation rate (mm. in 1 hr.)	1–119	25
Leukocytes (/mm.^{3})	4,000–24,300	8,000
Urea (mg./dl.)	14–42	37
Creatinine (mg./dl.)	0.7–9.9	1.50
	Patients with positive findings (per cent)	
Urine		
Pyuria	89	
Proteinuria	80	
Microhematuria	77	
Casts	27	

TABLE V

CYSTOSCOPIC FINDINGS IN 46 PATIENTS WITH URINARY TRACT TUBERCULOSIS

Finding	No. of Patients*
Cystitis (not specified other than areal)	26
Mucosal ulcerations	12
Mucosal granulomatous change	9
Mucosal injection or congestion	12
Delta area of redness below ureteric orifices	8
Contracted bladder	12
Reflux or "golf-hole" ureteric orifice	4
Pinpoint or scarred orifice	4
Bullous edema over trigone	2
Cystitis cystica	1
Posterior urethritis or prostatic duct abscess	1
Ureteral obstruction above ureteral vesicular junction	1

* Most patients had multiple findings.

ROENTGENOGRAPHIC FINDINGS

Plain film roentgenographic examination, a necessary preliminary for excretory urography, provided valuable information. The demonstration of tuberculous disease of the skeletal system, paraspinous or psoas abscesses, and calcification in the liver, spleen, adrenal glands, or lymph nodes, or in the base of the lung should alert the radiologist to the likelihood of a tuberculous process (Fig. 1, *A*, *B*, and *D*). Such findings were present in 31 patients of our series (Table VI). Excretory urography revealed renal abnormalities in 90 patients (Table VII): bilateral in 27 patients and unilateral in 63 patients.

Renal calcification was present in 50 patients (Table VIII). Eleven patients had extensive parenchymal calcification, with a "putty-like" appearance (Fig. 1*A*). In all 11, the kidneys were nonfunctioning. All 11 kidneys had rather uniform calcification, but none had diffuse peripheral calcification of the type seen in renal cortical necrosis, oxalosis, and chronic glomerulonephritis. Twenty-nine patients had localized areas of parenchymal calcification (Fig. 1, *B–D*). In 11 of the 29, the focal calcification was well circumscribed, amorphous, irregular, and often faint and was considered to be associated with a granulomatous mass (Fig. 1, *B* and *C*). In the remaining 18 kidneys, no evidence of such a mass was apparent. Renal calculi were seen in 18 patients, 8 with both parenchymal calcification and renal calculi.

Parenchymal scars were also seen in 50 patients. These scars varied from small and localized ones to scars that involved the entire kidney. Usually, scars were associated with parenchymal calcification in areas of tissue destruction or were located in areas of the kidney involved with an obstructive process. Each case had some abnormality of the underlying collecting system, and none of the scarring was considered to represent early or minimal change.

Medullary cavitation with communication to the collecting system was seen in 35 patients. In 22 patients, a small cavity was seen in only one renal papilla (Fig. 2*A*), while an additional 13 patients had more advanced involvement with multiple papillary cavities (Fig. 2*B*). In 6 patients, a small single cavity in a renal papilla was seen as the sole roentgenographic abnormality. In 29 patients, the cavitation was found in only 1 kidney, while in 6, the cavitation was bilateral. In 2 of these 6, all the renal papillae were involved. Cavitation either involved the tip of the papillae as just described or progressed to destroy the parenchyma. In some, the cavitation was extensive and irregular (Fig. 2*C*). When the cavities communicated freely with the collecting system, their nature could be readily determined. There was, at times, a striking similarity of early papillary cavitation to renal papillary necrosis resulting from other causes.

Irregularity of the papillary tips, best described as a "moth-eaten" appearance, was seen in 8 patients (Fig. 2*D*). In 6, the irregularity involved only 1 renal papilla, but in all 8 of these patients, there were other findings in the kidney. Calyceal diverticula were seen in 2 patients and were distinguishable from the small papillary cavity.

Thirty-five patients had narrowing or complete stenosis of one or more infundibula of the minor calyces (Fig. 2*D*; and 3). In 1 patient, an infundibular stricture with mild dilatation of that single calyx was the solitary finding on the urogram. These infundibular strictures, when complete, result in the exclusion of a previously dilated calyx from the remainder of the collecting system. When such a parenchymal cavity was excluded from the collecting system, it was often impossible to determine if it was the result of a localized intrarenal obstruction or a parenchymal abscess cavity due to a caseous granuloma with necrosis. Tomography was often helpful in recognizing the nature of these intrarenal obstructions and, in several cases, demonstrated an excluded cavity that had been seen to communicate with the renal pelvis (Fig. 3, *C* and *D*).

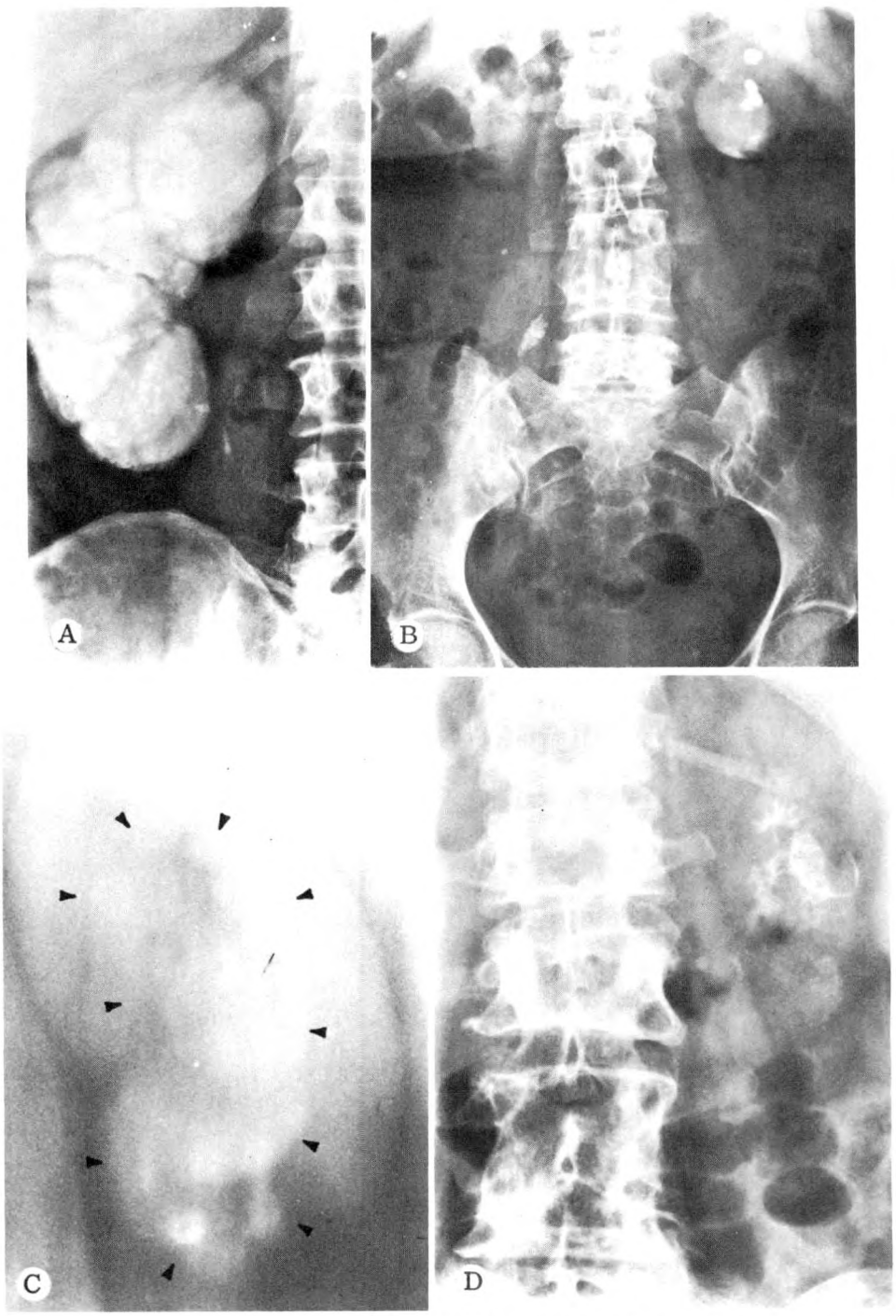


Fig. 1. Renal calcification. (*A*) Extensive amorphous calcification in nonfunctioning kidney—autonephrectomy pattern. Ureteral calculus in upper ureter. (*B*) Amorphous calcification in left lower pole and multiple left renal calculi. Tuberculous spondylitis, with fusion of the bodies of L-4 and L-5 and right psoas abscess. Calcified mesenteric lymph nodes. (*C*) Lobulated amorphous calcification of lower two-thirds of this kidney demonstrated only on tomography. (*D*) Focal discrete calcification scattered throughout parenchyma. Tuberculous spondylitis, with fusion of L-3 and L-4.

TABLE VI

ACCESSORY ROENTGENOGRAPHIC FINDINGS IN 31 PATIENTS WITH URINARY TRACT TUBERCULOSIS

Finding		No. of Patients
Pott's disease		9
With paraspinous abscess	2	
Calcified abdominal and retroperitoneal lymph nodes		11
Calcified adrenal gland		1
Tuberculous arthritis		9
Hip	2	
Sacroiliac	3	
Knee	2	
Ankle	1	
Wrist and hand	1	
Intracranial calcification		1

Blunting or clubbing was seen in 34 patients. In 20 patients, an extrarenal site of obstruction was present and generalized hydronephrosis resulted. In the other 14 patients, a localized intrarenal obstruction resulted in similar but more localized changes.

Advanced cicatricial changes involving the calyces or renal pelvis or both were seen in 16 patients and appeared to be the result of healing of a parenchymal lesion (Fig. 4). These changes were commonly associated with an infundibular stricture in either pole and often resulted in traction on and subsequent obstruction of the remaining segments, a phenomenon well described by Barrie *et al.*[2]

Eight patients had normal-appearing kidneys, and all 8 had positive urine cultures. One patient had had a nephrectomy and had presented with residual tuberculous cystitis. A second patient with tuberculous cystitis had had pulmonary tuberculosis more than 25 years previously and had never received antituberculous medication; a left ureteral urine specimen obtained from this patient was positive for *M. tuberculosis*. The remaining 6 patients had evidence of either current or past tuberculous epididymitis.

In our series, 2 patients had intramural ureteral calcification, but neither had bladder calcification. Ureteral stones were found in 5 patients and, although unconfirmed, were likely in an additional 2 patients. No bladder calculi were found roentgenographically, although 1 patient had a small stone seen at cystoscopy. Prostatic calcification indistinguishable from other types of prostatic concretions was seen in 14 patients but was not a reliable diagnostic sign. The presence of prostatic calcification was not related to either the symptoms or the severity of the renal tuberculosis.

Calcification involved the seminal vesicle in 2 patients and the vas deferens in 3 patients. The calcification in the vas deferens of 2 patients was intramural and indis-

TABLE VII

RENAL CHANGES IN 90 PATIENTS WITH URINARY TRACT TUBERCULOSIS

Changes	No. of Patients*
Parenchymal scarring	50
Parenchymal calcification	40
Papillary cavitation	35
Infundibular stenosis or stricture	35
Blunting or clubbing of calyces	34
Granulomatous mass	24
Renal calculi	18
Cicatricial changes in collecting system	16
Caliectasis	10
Minimal calyceal irregularity	8

* Most patients had multiple changes.

TABLE VIII

RENAL CALCIFICATION ON UROGRAPHY OF 50 PATIENTS WITH URINARY TRACT TUBERCULOSIS

Calcification		No. of Patients*
Parenchymal		40
Diffuse (autonephrectomy pattern)		11
Focal		29
In cortex	1	
In medulla	6	
Of indeterminate location	11	
Within granulomatous mass	11	
Renal calculi		18

* Eight patients had both parenchymal calcification and renal calculi.

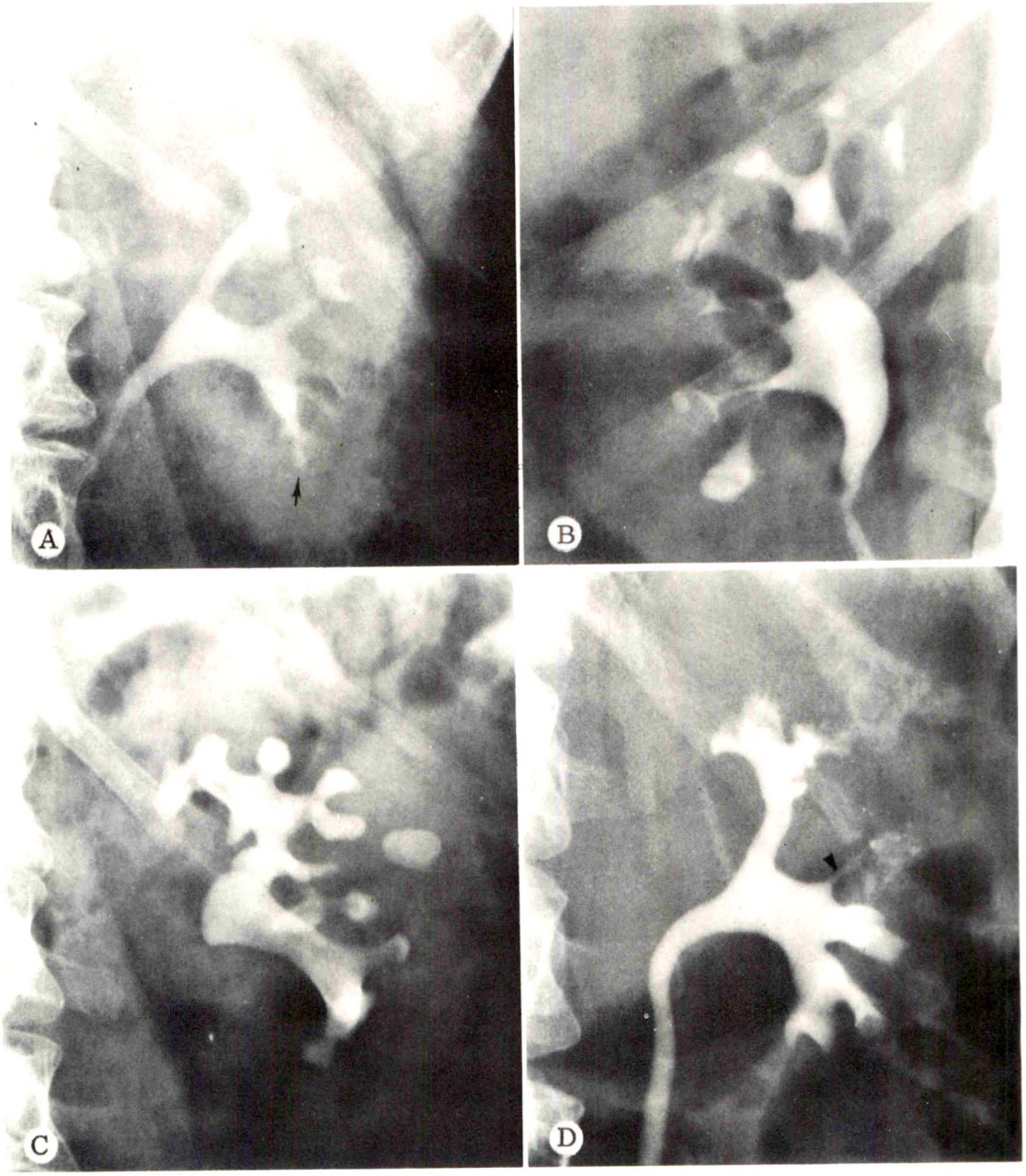


Fig. 2. Medullary cavitation. (*A*) Tiny 2 mm. cavity in papillary tip of lower pole calyx (arrow), representing only roentgenographic finding. (*B*) Extensive papillary cavitation involving almost all of renal papillae. (*C*) More extensive changes of papillary destruction. (*D*) Moth-eaten appearance in composite upper calyx due to irregular ulceration of papillary surface. Infundibular stricture in midportion of kidney (arrowhead).

tinguishable from that seen with normal aging or diabetes; however, neither patient was old (34 and 46 years old) and neither had diabetes mellitus. An intraluminal concretion seen in the vas deferens of the third patient is reportedly more characteristic of tuberculosis (Fig. 5).[12]

Inflammatory changes involving the renal pelvis and ureter were seen in 8 patients. Four of these patients had dilatation

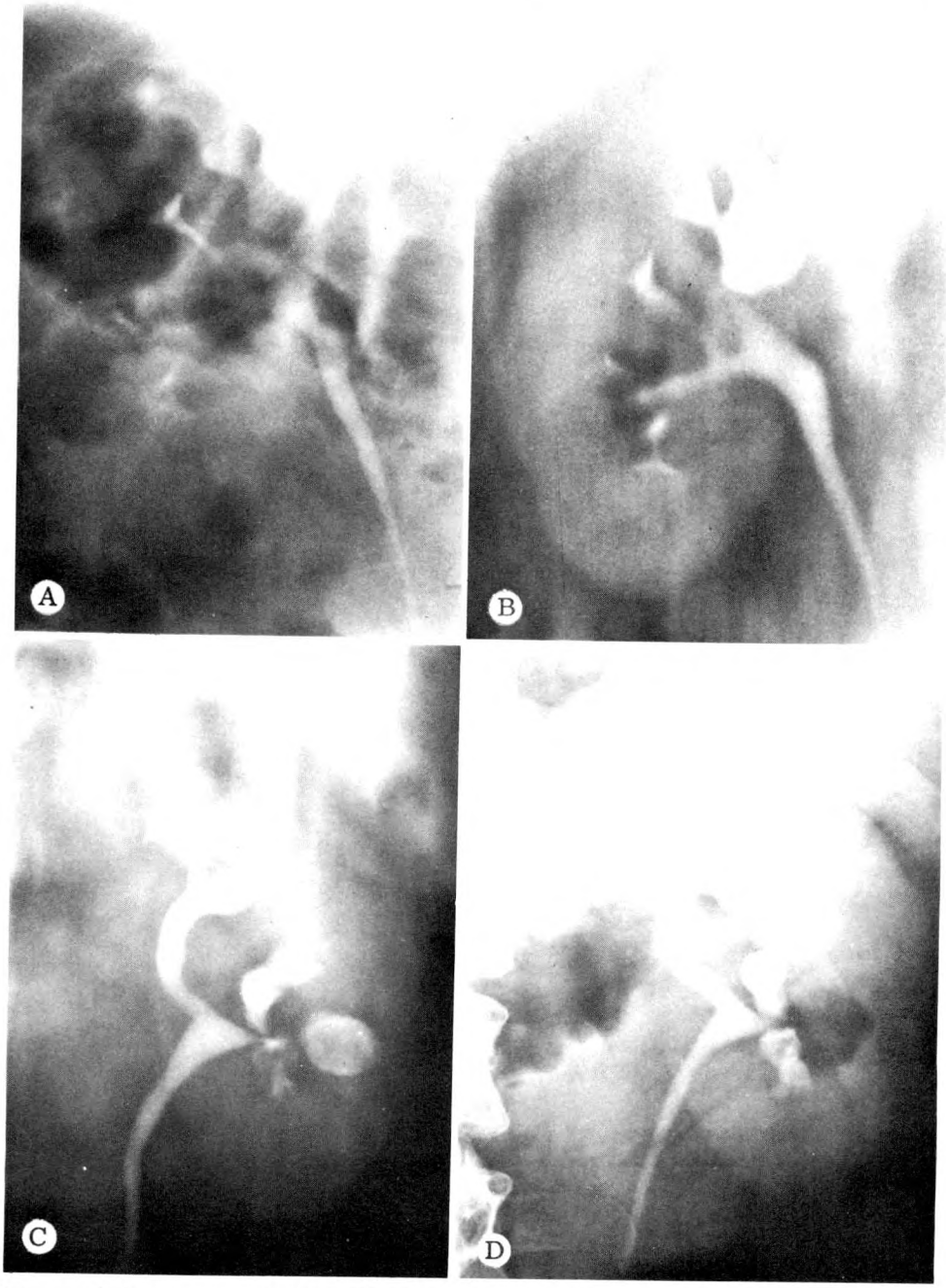


Fig. 3. Intrarenal obstruction. (*A*) Dilatation of upper calyces noted, but renal area is obscured by overlying bowel contents. (*B*) Tomogram demonstrates upper pole infundibular stricture as well as remainder of collecting system. (*C*) Lower pole infundibular stricture, with associated distortion of adjacent collecting system. (*D*) One year later, stricture has completely excluded previously dilated calyx from remainder of collecting system.

of the ureter without evidence of stricture. Two patients had mucosal striations in the renal pelvis and in a ureter, and 2 patients had frank mucosal ulceration involving one ureter.

An isolated single ureteral obstruction was seen in 14 patients, while multiple strictures were demonstrated in 7. These

single strictures identified were found with equal frequency on either side. Ureteropelvic junction obstruction was seen in 9 patients. Obstruction at the ureterovesical junction was demonstrated unilaterally in 7 patients and bilaterally in 1.

Four patients had advanced fibrosis of the ureter, which is characteristic of tuberculous ureteritis.[7,9,10,19] Two of these patients had irregularly dilated ureters that were best described as "beaded" or nodular (Fig. 6*A*). In the third patient, a rigid, straight "pipestem" ureter was demonstrated by a retrograde pyelogram after a catheter was passed beyond a high-grade ureterovesical junction obstruction (Fig. 6*B*). The fourth patient had a corkscrew configuration of a fibrotic ureter (Fig. 6*C*).

The bladder was urographically abnormal in 27 of the 98 patients. In 7 patients, irregular scarring of the bladder wall was present, and in 5 patients, the bladder was smooth and contracted. Generalized thickening of the bladder wall was seen in many patients but was severe in only 2. A filling defect was seen in the bladder of 1 patient and corresponded to an area of granulomatous tissue seen at cystoscopy. The remaining patients with abnormal-appearing bladders had either prostatic enlargement or bladder diverticula. All patients whose bladders were abnormal at urography and who were examined by cystoscopy had positive cystoscopic examinations.

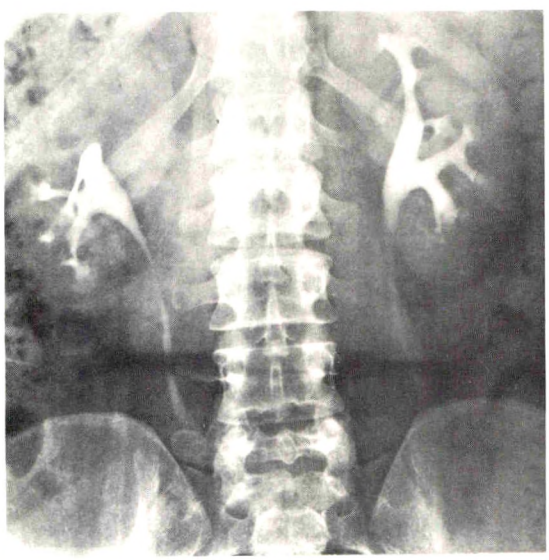


Fig. 4. Advanced cicatricial changes with traction on collecting system, associated with healing of upper pole lesion.

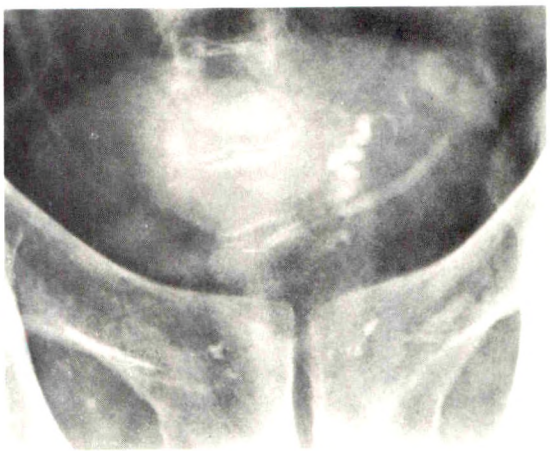


Fig. 5. Calcification of vas deferens, seminal vesicle, and prostate.

DISCUSSION

There is uniform acceptance that renal tuberculosis is a bilateral process resulting from hematogeneous spread of tubercle bacilli from a distant focus, usually a pulmonary lesion. Because the initial lesion often heals, few patients who have renal tuberculosis present with positive sputum cultures indicating the source of the dissemination.[5] Although the disease is always bilateral, the presenting clinical symptoms do not usually indicate this.[17]

Ninety-five of the 98 patients in our study had symptoms related to either their urinary tract or genital organs some time during their illness. Fourteen of the 61 men had histories of prior tuberculous epididymitis that often antedated the clinically apparent renal involvement by several decades. Several patients had histories of a prior tuberculous epididymitis that was treated surgically many years earlier, although it was not immediately associated with the presenting urinary tract symptoms. The significance of this history was usually appreciated when the routine cultures were "sterile," whereas the urinalysis

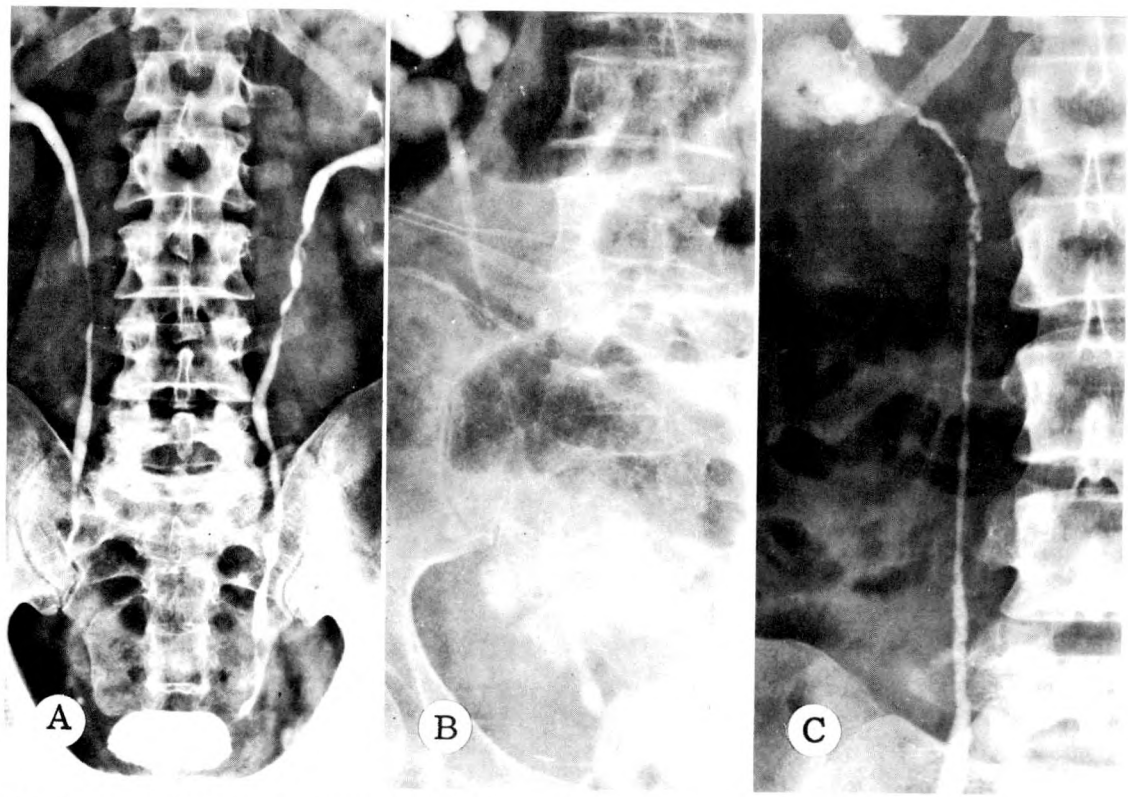


FIG. 6. Advanced tuberculous ureteritis. (*A*) Alternating areas of ureteral fibrosis and dilatation, producing beaded or nodular appearance. (*B*) Uniform thickening of wall of ureter and bladder, resulting in rigid pipestem appearance of ureter and smoothly contracted bladder. (*C*) Irregular scarring of this ureter, producing corkscrew appearance associated with extensive scarring in nonfunctioning kidney.

indicated a probable infectious process. There were no symptoms referable to the reproductive organs among the women studied.

The correlation is high between the anatomic and pathologic changes and the roentgenographic findings.[23] However, a normal excretory urogram does not exclude the possibility of renal tuberculosis, and the urogram often is normal when there is early disease. The early lesions involve the renal cortex or corticomedullary junction in almost 90 per cent of the cases; the other 10 per cent involve the renal medulla.[15–17] The most common locations are within a capillary tuft of a glomerulus or within capillaries between the convoluted tubules of the cortex or the collecting ducts of the renal pyramid. The early lesions are not detectable on excretory urography (Fig. 7).

The second stage of early renal tuberculosis involves the luminal space of the collecting ducts or loop of Henle and results from the intraluminal discharge of tubercle bacilli from the initial vascular lesion.[15–17] This second type leads to ulceration at the renal papillary tip and produces irregularity or a small communicating cavity—the earliest definitely discernible roentgenographic finding. Although some of these earlier papillary lesions heal, progression and further destruction of the renal tissue occur frequently. The asymmetric progression of tuberculosis is a characteristic feature, and only the association with other findings allows the early papillary cavity to be differentiated roentgenographically from other causes of renal papillary necrosis (Fig. 2*B*).

In our series, renal calcification was a more frequent finding in the kidneys than was cavitation and generally occurred with

more severe disease. The size of the calcification did not indicate the extent of the disease inasmuch as small foci of parenchymal calcification were seen in kidneys largely destroyed by caseous necrosis. Parenchymal calcification has been noted by others in from 7.1 to 24.4 per cent of patients with renal tuberculosis,[1,8,11] but we noted such calcification in approximately half of our patients. At times, the calcification required optimal technique for its demonstration (Fig. 1*C*). Renal calculi were seen in 18.4 per cent of our patients and have been noted by others in from 1.4 to 24 per cent[1,18,21] of patients with renal tuberculosis. Although renal calculi have been associated with increased incidence of secondary infection,[1] we did not observe this.

We noted that many patients with clinical or cystoscopic evidence of bladder involvement had normal-appearing bladders at urography. Much of this disparity is due to the early mucosal involvement that is difficult or impossible to recognize on a urogram. Voiding cinecystourethrograms were not obtained, and only 2 patients with far-advanced bladder involvement underwent retrograde cystography; severe bladder involvement was confirmed in both.

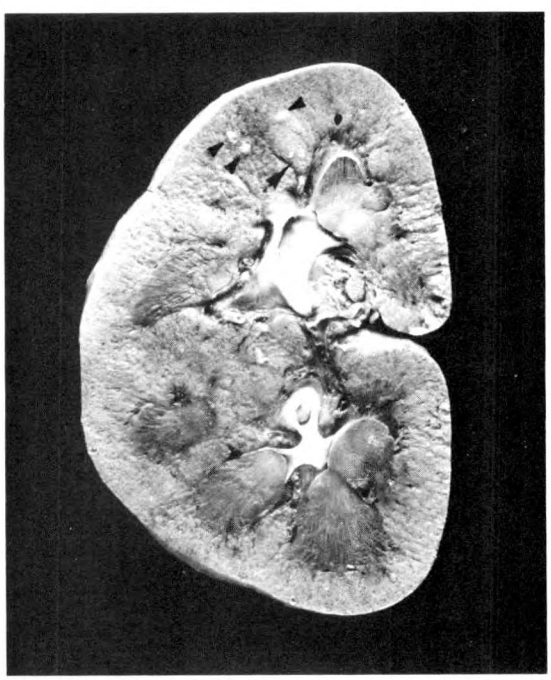


Fig. 7. Early pathologic findings. Multiple small cortical granulomata (arrowheads) and small necrotic cavities at papillary tip (arrow).

Excretory urography is the basic method of evaluation of patients with renal tuberculosis. Regularly employed ureteral compression often helps fill the collecting system well enough for minimal calyceal irregularity and small papillary cavities to be seen. Linear tomography is a helpful adjunct, particularly in evaluating the extent of the disease process and the residual functioning tissue in kidneys largely destroyed by tuberculous involvement (Fig. 8, *A* and *B*). Tomography was often helpful in demonstrating subtle amorphous calcification and small papillary cavities. In many patients, the changes in the renal outline and collecting system were obscured on conventional roentgenograms by overlying bowel contents but were well seen on the tomograms. In 5 patients, tomography demonstrated generalized hydronephrosis in the nonfunctioning kidneys. In 9 patients, retrograde pyelography was utilized in investigating nonfunctioning or poorly functioning kidneys. In these few patients, this procedure helped immeasurably to define the extent of calyceal changes but did not aid in the evaluation of the parenchymal involvement. The improved quality of the excretory studies with ureteral compression and tomography has greatly diminished the need for retrograde studies in these patients.

CONCLUSION

Renal tuberculosis continues to be a significant clinical problem, and the radiologist should be capable of suggesting the diagnosis in most instances. The most common roentgenographic findings are renal parenchymal calcification, parenchymal scarring, papillary cavities similar to those seen in papillary necrosis, and strictures of the collecting system. Intrarenal granulomatous masses, ranging up to 6 cm. in diameter, are also common, and the masses are usually calcified. Any combina-

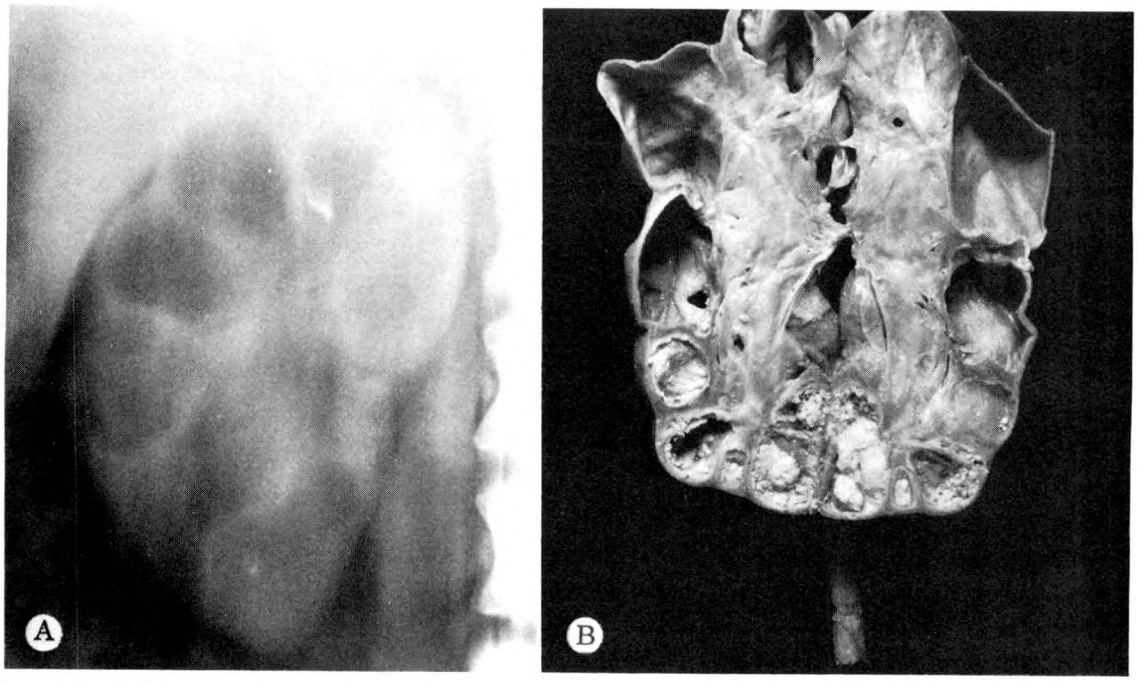


FIG. 8. (*A*) Extensive destruction of renal parenchyma with multiple radiolucent masses, suggestive of hydronephrosis. (*B*) Pathologic examination of kidney reveals hydronephrotic calyces and multiple granulomatous masses, both of which could produce changes present on tomogram.

tion of these findings increases the likelihood of renal tuberculosis.

Clinical findings can be helpful in indicating the diagnosis. Symptoms of chronic urinary tract infection associated with negative routine cultures are a common presentation. Such a history in a patient who is foreign-born or who has spent a significant period in a foreign country greatly increases the probability of urinary tract tuberculosis. A history or roentgenographic evidence of tuberculous infection of another part of the body may also alert the radiologist to the possibility of this condition. A positive chest roentgenogram is often helpful, but the absence of positive findings may be of little significance when establishing a diagnosis of renal tuberculosis (the chest roentgenogram was normal in 46 of 98 patients).

A positive urine culture for tuberculosis is necessary to establish the diagnosis definitely. One must be persistent when the urographic changes suggest the possibility of renal tuberculosis, because, in 1 case in our series, as many as 8 urine cultures were necessary before a positive culture was obtained.

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REFERENCES

1. APPERSON, J. W., WECHSLER, H., and LATTIMER, J. K. Frequent occurrence of both renal calculi and renal calcifications in tuberculous kidneys. *J. Urol.*, 1962, *87*, 643–646.
2. BARRIE, H. J., KERR, W. K., and GALE, G. L. Incidence and pathogenesis of tuberculous strictures of the renal pyelus. *J. Urol.*, 1967, *98*, 584–589.
3. BORTHWICK, W. M. Genito-urinary tuberculosis. *Tubercle*, 1956, *37*, 120–136.
4. BORTHWICK, W. M. Present position of urinary tuberculosis. *Brit. J. Urol.*, 1970, *42*, 642–646.
5. BRUCE, L. G. Incidence of genito-urinary tuberculosis in the western region of Scotland. *Brit. J. Urol.*, 1970, *42*, 637–641.
6. CARR, D. T. The treatment of renal tuberculosis. *Med. Clin. North America*, 1966, *50*, 1137–1139.

7. Claridge, M. Ureteric obstruction in tuberculosis. *Brit. J. Urol.*, 1970, *42*, 688–692.
8. Crenshaw, J. L. Renal tuberculosis with calcification. *J. Urol.*, 1930, *23*, 515–533.
9. Friedenberg, R. M. Tuberculosis of genitourinary system. *Seminars Roentgenol.*, 1971, *6*, 310–322.
10. Friedenberg, R. M., Ney, C., and Stachenfeld, R. A. Roentgenographic manifestations of tuberculosis of ureter. *J. Urol.*, 1968, *99*, 25–29.
11. Gow, J. G. Renal calcification in genito-urinary tuberculosis. *Brit. J. Surg.*, 1965, *52*, 283–288.
12. King, J. C., Jr., and Rosenbaum, H. D. Calcification of the vasa deferentia in nondiabetics. *Radiology*, 1971, *100*, 603–606.
13. Lattimer, J. K., Reilly, R. J., Segawa, A., Wechsler, H., Siegel, J., Girgis, A., and Gleason, D. Injections are no longer necessary in the treatment of renal tuberculosis. *J. Urol.*, 1965, *93*, 735–738.
14. Lowell, A. M. Tuberculosis (Vital and Health Statistics Monographs). Harvard University Press, Cambridge, MA, 1969, pp. 40–83.
15. Medlar, E. M. Cases of renal infection in pulmonary tuberculosis: evidence of healed tuberculous lesions. *Am. J. Pathol.*, 1926, *2*, 401–414.
16. Medlar, E. M., and Sasano, K. T. Experimental renal tuberculosis: with special reference to excretory bacilluria. *Am. Rev. Tuberc.*, 1924, *10*, 370–391.
17. Medlar, E. M., Spain, D. M., and Holliday, R. W. Post-mortem compared with clinical diagnosis of genito-urinary tuberculosis in adult males. *J. Urol.*, 1949, *61*, 1078–1088.
18. Murphy, G. P., and Fishbein, R. H. Urolithiasis and renal tuberculosis. *J. Urol.*, 1961, *85*, 879–881.
19. Rees, R. W. M., and Hollands, F. G. The ureter in renal tuberculosis. *Brit. J. Urol.*, 1970, *42*, 693–696.
20. Riddle, P. R. Urinary tuberculosis. *Postgrad. Med. J.*, 1971, *47*, 718–722.
21. Ross, J. C. Calcification in genito-urinary tuberculosis. *Brit. J. Urol.*, 1970, *42*, 656–660.
22. Roylance, J., Penry, B., Davies, E. R., and Roberts, M. Radiology in the management of urinary tract tuberculosis. *Brit. J. Urol.*, 1970, *42*, 679–687.
23. Theros, E. G. The value of radiologic-pathologic correlation in the education of the radiologist. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 235–257.

XANTHOGRANULOMATOUS PYELONEPHRITIS*

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XANTHOGRANULOMATOUS pyelonephritis (XGP) is a rare form of chronic pyelonephritis, characterized by yellow masses macroscopically and foamy, lipid-laden macrophages microscopically. It constitutes between 1 and 8 per cent of inflammatory conditions of the kidney requiring nephrectomy, with an age range between 10 months and 82 years. XGP may involve the kidney diffusely, or may be more focal as in the tumefactive form, which presents as a renal mass lesion. The disease is virtually always unilateral and the treatment of choice is nephrectomy.

During the past 7 years, we have had the opportunity to examine 15 patients with this diagnosis, 10 of whom had studies available for review. Nine patients had intravenous pyelograms, 1 had a retrograde pyelogram only, and 5 of the 10 patients had renal arteriograms. The arteriographic findings in 2 of these patients have been previously reported.[16]

The roentgenographic findings in this series are presented, indicating that this diagnosis can frequently be suggested preoperatively.

ROENTGENOGRAPHIC OBSERVATION IN PRESENT SERIES

Our group of patients included 4 male patients, and 6 females. The average age was 42 years, with an age range of 24 to 61 years. In all cases, the disease was unilateral, involving the right kidney in 4 patients and the left in 6 patients. However, contralateral renal calculi were observed in 2 patients.

The roentgenographic findings in this group are summarized in Table 1. Noteworthy is the high incidence of renal enlargement on the involved side, being present in 80 per cent of the patients. The average renal size in the 9 cases with enlarged kidneys was 18.8 cm. Renal and ureteral calculi were seen in 70 per cent and 80 per cent of cases, respectively. Of particular interest was the size of the ureteral calculi. Large obstructing ureteral calculi were seen in 5 patients (50 per cent), with the smallest measuring 1.0×2.2 cm., and the largest measuring 2.8×3.0 cm. (Fig. 1*A*). In the 3 other patients with ureteral calculi, 2 represented extensions of staghorn calculi into an obstructing position at the ureteropelvic junction (Fig. 2*A*), and 1 was a lucent calculus lodged at the pelvic brim, associated with ipsilateral renal calculi (Fig. 3, *A* and *B*).

In 6 of the 9 patients (67 per cent) who had intravenous pyelograms, there was nonvisualization of the collecting system. In the other 3 patients, 2 had only faint visualization (Fig. 2*B*), and the third had the tumefactive form of XGP, and showed the pyelographic findings of a renal mass (Fig. 4*A*). Four patients showed a total

TABLE I

ROENTGENOGRAPHIC FINDINGS IN XANTHOGRANULOMATOUS PYELONEPHRITIS (XGP)

Calculi		
Renal	7/10	
(Staghorn)		(2/7)
Contralateral renal calculi	2/10	
Ureteral	8/10	
(Ureteropelvic junction)		(6/8)
Renal enlargement		
Diffusely enlarged	8/10	
(Enlarged with local mass)		(2/8)
Tumefactive form in normal or small kidney	2/10	
Intravenous pyelography		
Nonfunction	6/9	
(Total absence of nephrogram)		(4/6)
Faint opacification	2/9	
Normal function	1/9	

* From the Department of Radiology, Virginia Commonwealth University-Medical College of Virginia, Richmond, Virginia.

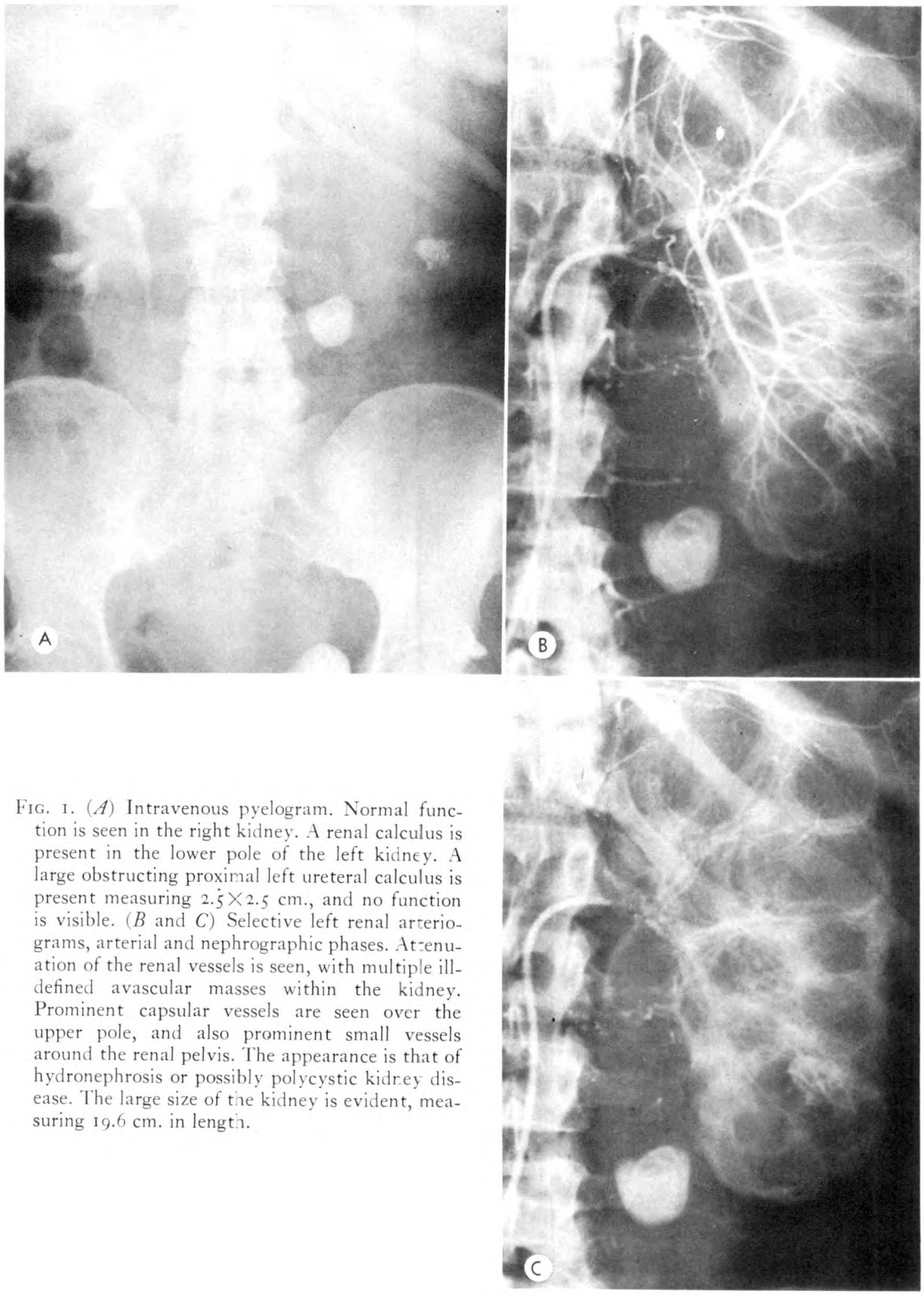


Fig. 1. (*A*) Intravenous pyelogram. Normal function is seen in the right kidney. A renal calculus is present in the lower pole of the left kidney. A large obstructing proximal left ureteral calculus is present measuring 2.5×2.5 cm., and no function is visible. (*B* and *C*) Selective left renal arteriograms, arterial and nephrographic phases. Attenuation of the renal vessels is seen, with multiple ill-defined avascular masses within the kidney. Prominent capsular vessels are seen over the upper pole, and also prominent small vessels around the renal pelvis. The appearance is that of hydronephrosis or possibly polycystic kidney disease. The large size of the kidney is evident, measuring 19.6 cm. in length.

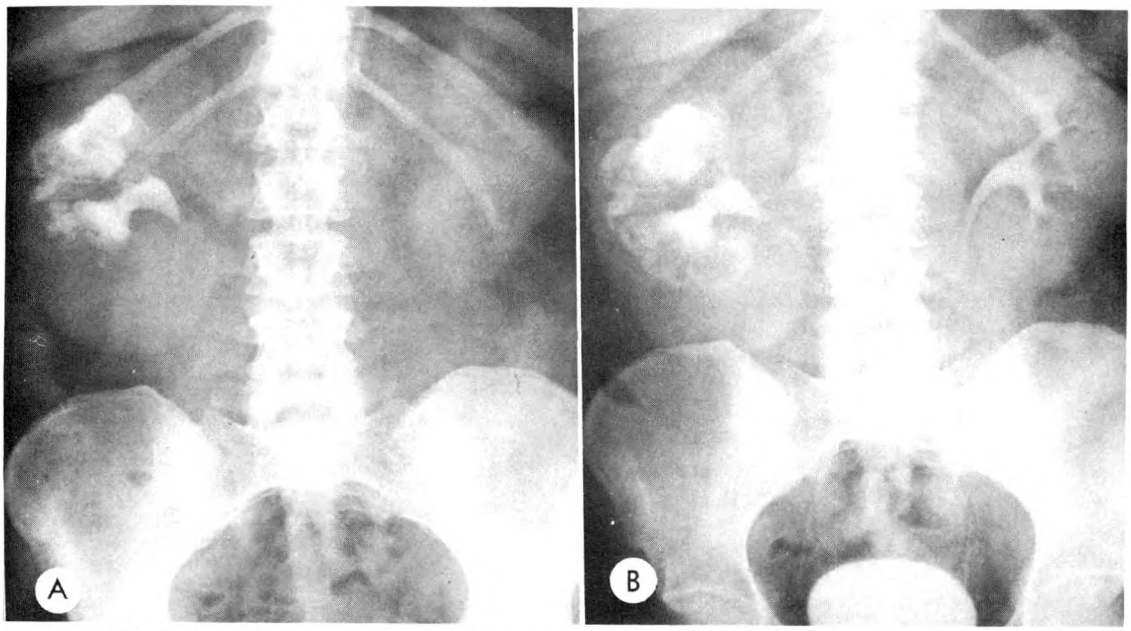


FIG. 2. (*A*) Plain film roentgenogram of the abdomen. A large staghorn calculus is present in the right kidney, extending into and partially obstructing the ureteral pelvic junction. The kidney measured 17.3 cm. in length. (*B*) Intravenous pyelogram. The left kidney functions normally. A very faint pyelogram is visible around the periphery of dilated calyces on the right side.

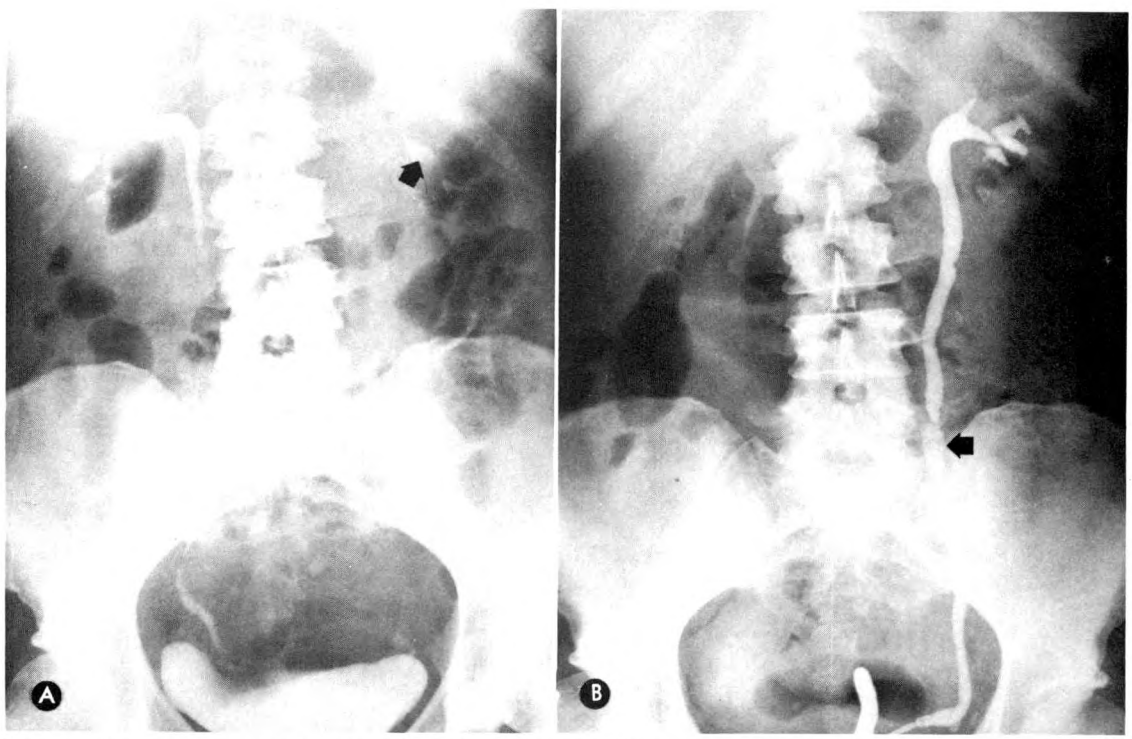


FIG. 3. (*A*) Intravenous pyelogram. The right kidney functions normally. A renal calculus is visible on the left (arrow), and the kidney is small in size, measuring 10.5 cm. in length. (*B*) Left retrograde pyelogram. A nonopaque calculus is present in the left ureter at the level of the pelvic brim (arrow). The major calyx to the upper pole is amputated by an upper pole mass, representing tumefactive xanthogranulomatous pyelonephritis.

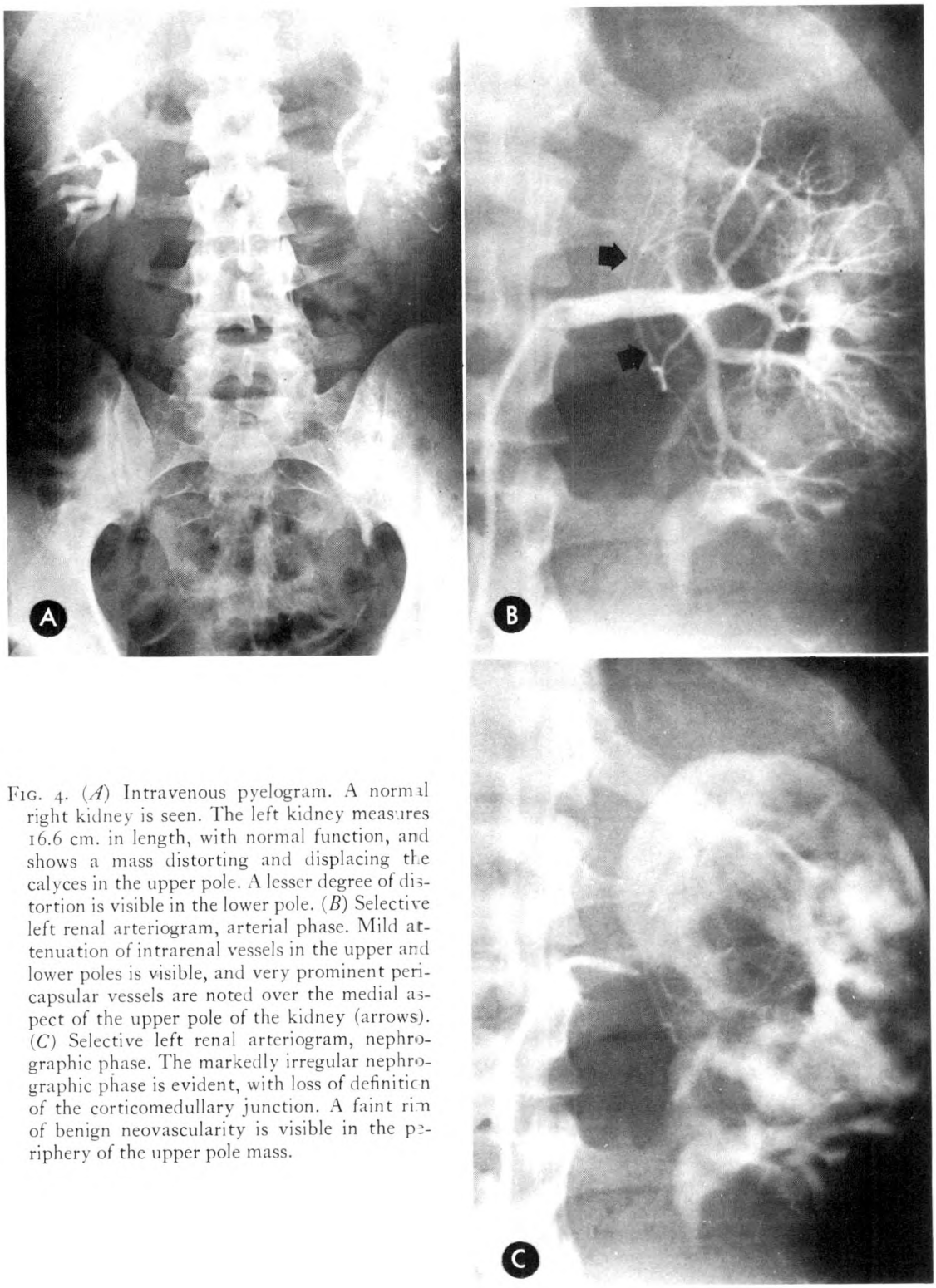


Fig. 4. (*A*) Intravenous pyelogram. A normal right kidney is seen. The left kidney measures 16.6 cm. in length, with normal function, and shows a mass distorting and displacing the calyces in the upper pole. A lesser degree of distortion is visible in the lower pole. (*B*) Selective left renal arteriogram, arterial phase. Mild attenuation of intrarenal vessels in the upper and lower poles is visible, and very prominent pericapsular vessels are noted over the medial aspect of the upper pole of the kidney (arrows). (*C*) Selective left renal arteriogram, nephrographic phase. The markedly irregular nephrographic phase is evident, with loss of definition of the corticomedullary junction. A faint rim of benign neovascularity is visible in the periphery of the upper pole mass.

TABLE II

ANGIOGRAPHIC FINDINGS IN XGP

Angiogram showing:	
Small main renal artery	2/5
Attenuation of vessels	5/5
Avascular mass (diffuse or local)	4/5
Benign neovascularity at margins	4/5
Irregular nephrogram	5/5
Absent corticomedullary junction	4/5
Increased capsular vessels	4/5
Angiogram simulating:	
Polycystic disease	1/5
Hydronephrosis	3/5

absence of a nephrogram phase—a point which has been claimed to be helpful in differentiating XGP from simple hydronephrosis.[5]

The findings in 5 patients who had renal angiography are summarized in Table II. The most common findings were attenuation of intrarenal vessels (Fig. 1*B*; and 5*A*), and a spotty, irregular nephrographic phase. The irregular nephrogram obliterated the corticomedullary junction (Fig. 1*C*; and 4*C*). The masses were usually avascular centrally, with inflammatory hypervascularity or a blush around the periphery. We also noticed a high incidence of prominent capsular and periureteral vessels (Fig. 1*B*; 4*B*; and 5*B*). In none of our cases could the vascularity be confused with that of a hypernephroma, but several cases closely simulated hydronephrosis or polycystic disease of the kidney (Fig. 1*C*; and 5*A*).

ETIOLOGY

The organism responsible for the infection is not specific, but most frequently is a gram negative organism, particularly *Proteus*.[1] The involved kidney frequently has calculi and is obstructed, but these are

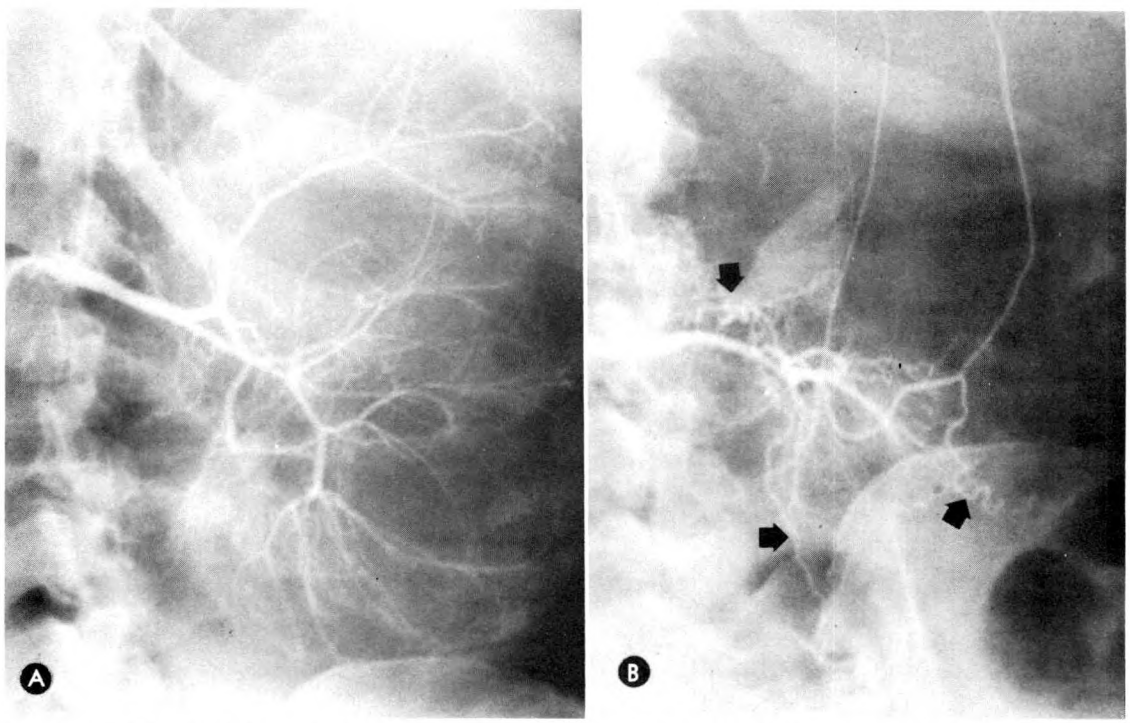


FIG. 5. (*A*) Selective left renal arteriogram of the upper of 2 left renal arteries. A staghorn calculus was obstructing the ureteropelvic junction. There is marked attenuation and stretching of the intrarenal vessels around multiple large avascular masses, reflecting the hydronephrosis present. The kidney is large, measuring 22.0 cm. in length. (*B*) Selective renal arteriogram of the lower of 2 left renal arteries. The lower pole of the kidney is supplied by this vessel, but very prominent pericapsular, peripelvic, and periureteral vessels are seen (arrows).

not prerequisites for this disease. The necessary conditions for the xanthogranulomatous type reaction seems to be the chronicity of the infection, and relatively low virulence of the organism.[5]

The clinical history of urinary tract infection is frequently of long duration, and it has been suggested that the slow development of XGP in an obstructed and infected kidney is due to intermittent antibiotic therapy preventing abscess formation or acute pyonephrosis.[4] It is an unanswered question why patients with XGP show this type of chronic pyelonephritis in the presence of obstruction, and others have a much more acute suppurative response to the combination of infection and obstruction.

PATHOLOGY

Most kidneys involved with XGP are either focally or diffusely enlarged, with multiple adhesions to the capsule.[4,15] Gross inspection reveals purulent fluid in the calyces, and multiple yellow nodules throughout the kidney with multiple abscesses, and thickening of the pelvic epithelium. Microscopically, acute and chronic pyelonephritis is seen, with polymorphonuclear leukocytes in variable quantities. The yellowish nodules consist of lymphocytes, fibroblasts, and plasma cells, but the hallmark is the lipid-laden histiocyte with foamy cytoplasm. These foamy macrophages stain positively for lipids. This differentiates them from hypernephromas, which may have a similar appearance on routine staining techniques. Several cases have been reported in which a false diagnosis of a hypernephroma was made initially, and only subsequent fat stains led to the diagnosis of XGP.[10,14] In addition, the histiocytes have nuclei which are uniform in size, shape and stainability, and there is no papillary or acinar arrangement of the cells, also serving to distinguish XGP from a hypernephroma.[13]

DISCUSSION

The roentgenographic evaluation is important for several reasons. The first, of course, is to identify the abnormality on the plain film roentgenograms and intravenous pyelogram. Retrograde pyelography is frequently the next procedure, to exclude hydronephrosis. Obstruction often is present in XGP, however, so that this does not help in the differential diagnosis. Arteriography is necessary to exclude an obstructing hypernephroma. In the tumefactive form of XGP, arteriography may make the distinction between a hypernephroma, cyst, or inflammatory process of the kidney. The correct preoperative diagnosis is helpful in planning appropriate surgery, particularly since the surgical findings and occasionally pathologic findings can also resemble a hypernephroma, and its exclusion prevents an unnecessary radical resection.[5,14]

Friedenberg and Spjut[4] outlined the typical findings in their group of patients with XGP, which was a triad including renal enlargement, obstruction, and nonfunction on the intravenous pyelogram, frequently with calculi. This triad was also recognized in our group of patients, but is also seen in obstructive hydronephrosis without XGP. A history of a chronic urinary tract infection is helpful in making the diagnosis. Furthermore, total absence of a nephrogram is uncommon in hydronephrosis, but common in XGP.[5] When a nephrogram is present in XGP, it tends to be nonhomogeneous with irregular lucencies within the kidney, and not as uniformly distributed as in hydronephrosis.

Approximately 75 per cent of patients with XGP will have enlarged kidneys, and over one-half will have renal or ureteral calculi. There may be difficulty in visualizing the renal outline on the plain film roentgenogram because of the surrounding inflammation. In 13 of 18 cases reported by Malek *et al.*[7] the process extended to include Gerota's fascia. Occasionally scoliosis may be seen. However, blurring of the adjacent psoas muscle, fixation during respiration and sympathetic pleural effusion are more common in acute inflammatory

conditions such as carbuncle and pyelonephritis.

Over one-half of the reported cases have had no function visible on the intravenous pyelogram, and many have no visible nephrographic phase. In some series, the incidence of nonfunction has been over 80 per cent.[1] Nonfunction also may be seen in hydronephrosis, but in most cases of hydronephrosis there is a nephrographic rim seen in the periphery of the kidney surrounding the dilated calyces.

In more acute infections of the kidney, the absence of the pyelogram and nephrogram is usually limited to the region of the abscess, but this may not be helpful in the individual case, since on one hand complete nonfunction may be seen in renal abscesses, and on the other hand, particularly in tumefactive XGP, the uninvolved portion of the kidney may show a normal pyelogram. However, the clinical findings are considerably different between acute renal carbuncles and XGP. The differential diagnosis in those cases presenting as a renal mass lesion include an obstructed second collecting system, renal tumor, cyst, and tumefactive XGP. This differentiation must be made by angiography.

In a review of the angiographic findings in 15 reported cases in 1973,[5] the most consistent abnormalities were a decreased diameter of the main renal artery, and narrowing of segmental arteries which are stretched around intrarenal masses. The segmental arteries were sparse, and often occluded or attenuated. Half of the cases showed prominent capsular and periureteric vessels. There was a slow perfusion rate and an irregular nephrogram with loss of the corticomedullary junction.[5,11,16] The nephrogram, when present, was not followed by opacification of the collecting system.

Innumerable filling defects in the nephrographic phase were caused by abnormal dilated calyces and xanthogranulomatous masses.[2] The over-all appearance may simulate hydronephrosis, although sometimes the large masses are more uniform in hydronephrosis, as opposed to the irregular nephrogram and poorly marginated avascular masses in XGP.

Benign neovascularity may arise from outside of the kidney and reflect either infection or ischemia.[12,16] It consists of multiple small tortuous vessels, rather than the irregular tumor vessels seen at the margin of a necrotic hypernephroma, with lakes of contrast medium, arterial venous shunting, and a blush within the tumor.[9]

The distinction between a renal carbuncle and the tumefactive form of XGP may be impossible. The clinical history in this case is very helpful. According to the review of Himmelfarb *et al.*,[6] increased capsular and periureteric vascularity was not seen in angiograms of acute carbuncles. In subacute or chronic carbuncles, however, the arteriographic finding was very prominent neovascularity sometimes simulating a hypernephroma, but at close inspection one usually could differentiate benign from malignant neovascularity.

In 3 situations, however, the distinction between a hypernephroma and a benign lesion is much more difficult: (1) necrotic hypernephromas;[9] (2) abscesses which have a surrounding rim of abundant capsular engorgement and benign neovascularity;[3] and (3) primary avascular renal tumors, especially papillary and tubular tumors as well as benign stromal tumors and hamartomas.[8] The differential diagnosis in the avascular group of tumors not only includes masses secondary to infection, but benign cysts, polycystic kidney disease, and extensive hydronephrosis. The criteria for the differential diagnosis of cysts from tumors have been well covered in the literature. However, multiple xanthogranulomas or abscesses may not have any neovascularity and may be rather sharply marginated, making them indistinguishable from polycystic disease or hydronephrosis. In these cases, the best one can do is suspect the diagnosis of XGP, based on the correlation of the angiogram with a clinical history of a chronic low-grade infection.

SUMMARY

Xanthogranulomatous pyelonephritis should be suggested preoperatively in patients with a history of chronic urinary tract infection and certain roentgenographic findings.

These include: (1) unilateral renal enlargement, either localized or diffuse; (2) renal and/or ureteral calculi, especially large ureteral calculi; (3) nonfunction or very faint opacification of the collecting system on intravenous pyelography; and (4) angiographic demonstration of an avascular mass or masses with stretched, attenuated intrarenal vessels, benign neovascularity at the margins of the mass(es), irregular nephrogram, and prominent capsular and periureteric vessels.

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REFERENCES

1. Anhalt, M. A., Cawood, C. D., and Scott, R., Jr. Xanthogranulomatous pyelonephritis: comprehensive review with report of four additional cases. *J. Urol.*, 1971, *105*, 10–17.
2. Becker, J. A. Xanthogranulomatous pyelonephritis: case report with angiographic findings. *Acta radiol.* (Diag.), 1966, *4*, 139–144.
3. Caplan, L. H., Siegelman, S. S., and Bosniak, M. A. Angiography in inflammatory space-occupying lesions of kidney. *Radiology*, 1967, *88*, 14–23.
4. Friedenberg, M. J., and Spjut, H. J. Xanthogranulomatous pyelonephritis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 97–108.
5. Gingell, J. C., Roylance, J., Davies, E. R., and Penry, J. B. Xanthogranulomatous pyelonephritis. *Brit. J. Radiol.*, 1973, *46*, 99–109.
6. Himmelfarb, E. H., Rabinowitz, J. G., Kinkhabwala, M. N., and Becker, J. A. Roentgen features of renal carbuncle. *J. Urol.*, 1972, *108*, 846–851.
7. Malek, R. S., Greene, L. F., DeWeerd, J. H., and Farrow, G. M. Xanthogranulomatous pyelonephritis. *Brit. J. Urol.*, 1972, *44*, 296–308.
8. Melicow, M. M., and Becker, J. A. Radiographic simulation of certain solid tumors of renal corpus to renal cyst. *J. Urol.*, 1967, *97*, 592–610.
9. Ranniger, K. Selective renal arteriographic appearance of necrotic hypernephroma. *Radiology*, 1964, *83*, 414–418.
10. Rios-Dalenz, J. L., and Peacock, R. C. Xanthogranulomatous pyelonephritis. *Cancer*, 1966, *19*, 289–296.
11. Rossi, P., Myers, D. H., Furey, R., and Bonfils-Roberts, E. A. Angiography in bilateral xanthogranulomatous pyelonephritis. *Radiology*, 1968, *90*, 320–321.
12. Salmon, R. B., and Koehler, P. R. Angiography in renal and perirenal inflammatory masses. *Radiology*, 1967, *88*, 9–13.
13. Selzer, D. W., Dahlin, D. C., and DeWeerd, J. H. Tumefactive xanthogranulomatous pyelonephritis. *Surgery*, 1957, *42*, 874–883.
14. Smout, M. S., McAninch, L. N., and Wyatt, J. K. Tumefactive xanthogranulomatous pyelonephritis. *Brit. J. Urol.*, 1963, *35*, 129–132.
15. Tonelli, S., and Giannotti, P. On nature of xanthogranulomatous pyelonephritis. *Urol. Int.*, 1969, *24*, 330–343.
16. Vinik, M., Freed, T. A., Smellie, W. A. B., and Weidner, W. Xanthogranulomatous pyelonephritis: angiographic considerations. *Radiology*, 1969, *92*, 537–540.

ANGIOGRAPHY IN UNILATERAL RENAL BLEEDING WITH A NEGATIVE UROGRAM*

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EVALUATION of the patient with gross hematuria includes prompt excretory urography. In the face of a negative urogram or a urogram with nonspecific changes such as retained blood clots, cystoscopy must be performed to evaluate the bladder and document the source of bleeding. It is particularly important to visualize bloody efflux from the ureter since this will lateralize an upper tract source. In the past, renal bleeding for which no pathology was apparent has been referred to as idiopathic or "essential" hematuria. It is not surprising that renal angiography has been increasingly employed in an effort to elucidate the cause of such bleeding. During the past 7 years, we have performed angiography on 48 patients with cystoscopically documented unilateral upper urinary tract bleeding and negative or nondiagnostic excretory and retrograde urograms.

The majority of these angiograms have been negative (Table I). Of the 20 patients, who had a cause for the bleeding established by clinical and roentgenographic evaluation, 6 had angiographic abnormalities (Table II).

It is the purpose of this paper to review our experience with this group of patients.

TABLE I

ANGIOGRAPHY IN UNILATERAL RENAL BLEEDING WITH NEGATIVE UROGRAM (48 PATIENTS)

Negative angiogram—negative follow-up	28
Negative angiogram—positive follow-up	14
Abnormal angiogram	6
Total	48

TABLE II

CLINICAL-ANGIOGRAPHIC CORRELATION IN PATIENTS WITH ESTABLISHED CAUSE FOR UNILATERAL HEMATURIA AND NEGATIVE UROGRAM

Cause	No.	Abnormal Angiogram
Hemoglobin SA	6	2
Pyelonephritis	4	1
Calculi	4	—
Venous malformation	2	2
Embolus	1	1
Focal hemorrhage (chemotherapy)	1	—
Papillary tumor*	1	—
Focal nephritis	1	—
Total	20	6

* Located near ureteral orifice; may not have been cause of bleeding. See text.

PATIENT SELECTION AND METHOD

All 48 patients in this study presented with gross hematuria. Following initial clinical evaluation, all patients had excretory urography performed. In 4 cases, filling defects due to blood clots were noted. One patient had a nonrotated kidney. Aside from these nonspecific findings, all of the excretory urograms were considered to be within normal limits. Cystoscopy was the next urographic examination in all patients. This procedure ruled out lower tract pathology and established the side of the upper tract bleeding.

All patients had selective renal arteriography except 2, in whom only aortography was performed. Filming was prolonged over a 12 second duration, so that the renal vein could be visualized. In 10 patients, selective renal phlebography was also performed.

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Good retrograde venous filling was obtained by injecting 5–8 micrograms of epinephrine into the renal artery just prior to the venous injection.

This study does not include patients with known unilateral renal trauma either accidental or related to renal biopsy.

Twenty-six of the patients studied were female between the age of 5 and 68 years of age. Twenty-two of the patients were male between 6 and 77 years of age.

RESULTS

1. *Sickle cell trait.* Six patients proved to have sickle cell trait. The presence of hemoglobin SA was confirmed by means of hemoglobin electrophoresis.

The cause of hematuria in sickle cell trait is well documented. It has been shown that the sickled cells lead to vascular congestion and focal hemorrhage.[9] The medulla is frequently involved, but similar changes are also seen in the cortex. It appears that the combination of hypoxia, hyperosmolality, and decreased pH in the renal medulla are the conditions which lead to sickling.[11,18] Microscopic examination demonstrates widely dilated capillaries crammed with sickle cells. The resulting stasis is most severe in the peritubular capillaries of the medulla.[17] Eventually, extravasation of blood into the peritubular areas and into the tubular lumina is seen. Focal involvement of the renal papillae may lead to necrosis. The epithelium of the renal pelvis is also commonly affected so that hemorrhage may also originate from this area.

The basic lesion is one that leads to stasis and hypoxia. The roentgen changes that we observed in 2 of 6 patients can be explained on this basis. One patient demonstrated a normal calyceal system and renal contour on an excretory urogram and nephrotomogram (Fig. 1*A*). An early film from a selective renal arteriography showed normal distribution of the renal vasculature (Fig. 1*B*). A later film from this angiography demonstrated an area of decreased cortical perfusion in the lower pole (Fig. 1*C*). The contrast material remained in the larger segmental branches of this area, while the remainder of the arterial tree had emptied. This decreased perfusion is a manifestation of the sickling which compromises the runoff in this portion of the kidney. There may also be some associated arterial spasm.

A second patient with hemoglobin SA showed similar changes. The nephrogram was mottled throughout the lower half of the kidney. These changes can be equated with poor cortical perfusion and ischemia accompanying the sickling in multiple small vessels.

2. *Pyelonephritis.* Four of our patients proved to have pyelonephritis. The diagnosis was made by renal biopsy in 3 cases and nephrectomy in 1 case. Microscopic hematuria accompanying renal infection is quite common. Gross hematuria may occasionally accompany pyelonephritis as demonstrated by our 4 patients. The most severe bleeding in this group occurred in a 65 year old man, who presented with clot retention. Poor opacification of the infected kidney was noted on the excretory urogram. This study was followed by cystoscopy and retrograde pyelography which demonstrated lucent filling defects in the renal pelvis that were blood clots (Fig. 2*A*). The calyces and renal parenchyma appeared well maintained in this patient with no gross scarring. Selective arteriography revealed a persistent group of small tortuous vessels in the mid portion of the upper pole (Fig. 2*B*). A later film from arteriography showed prolonged opacification of these vessels (Fig. 2*C*). A tumor was suspected on the basis of these atypical angiographic findings and so nephrectomy was performed. Pathologic examination showed acute and chronic pyelonephritis with some organized thrombi in small and medium size veins. Blood was noted in the tubules as were inflammatory cells (Fig. 2*D*).

3. *Vascular malformations.* Two patients had vascular malformations. In both cases the abnormality was confined to the venous circulation. This took the form at angiography of aneurysmal dilatation of the main

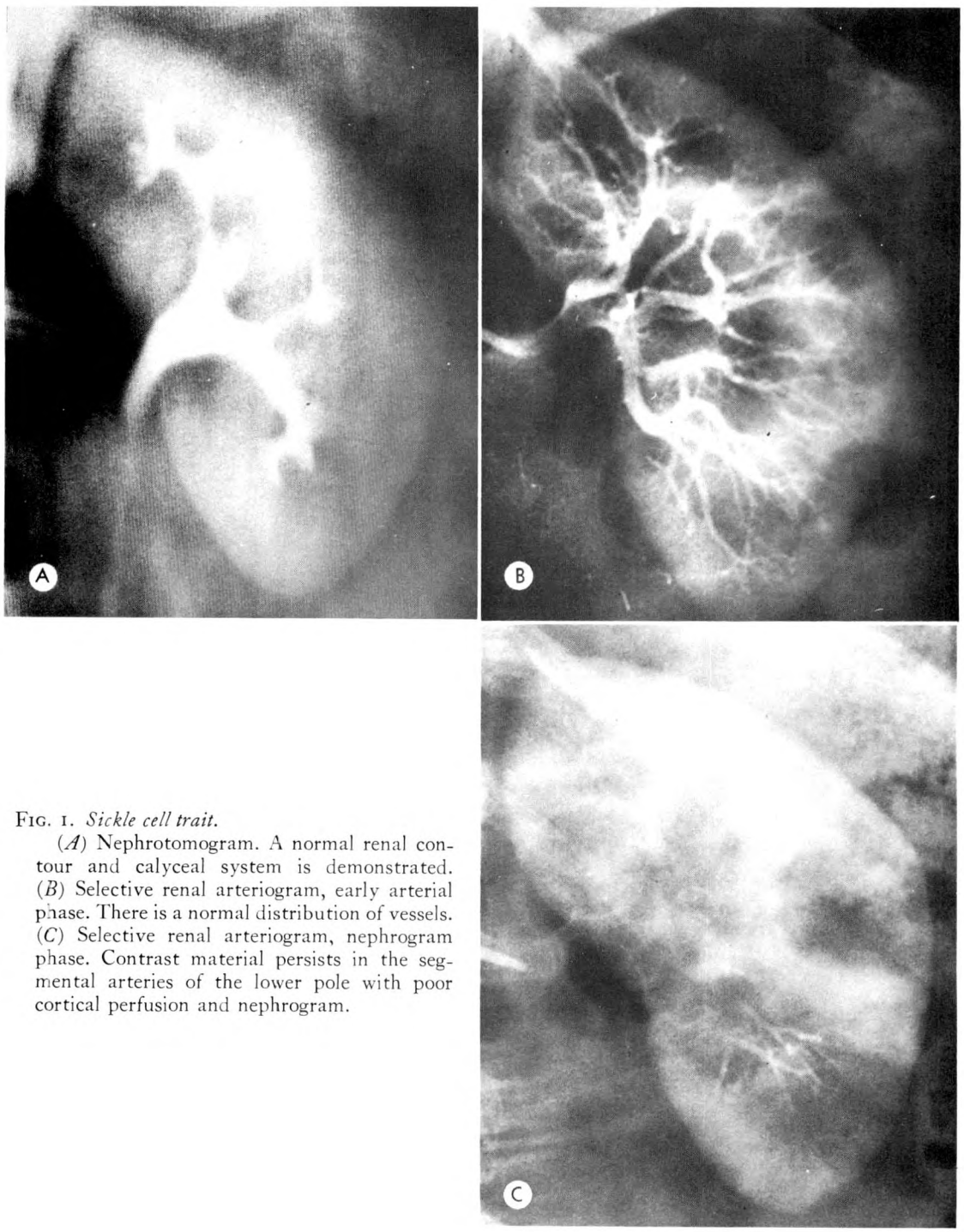


FIG. 1. *Sickle cell trait.*

(*A*) Nephrotomogram. A normal renal contour and calyceal system is demonstrated. (*B*) Selective renal arteriogram, early arterial phase. There is a normal distribution of vessels. (*C*) Selective renal arteriogram, nephrogram phase. Contrast material persists in the segmental arteries of the lower pole with poor cortical perfusion and nephrogram.

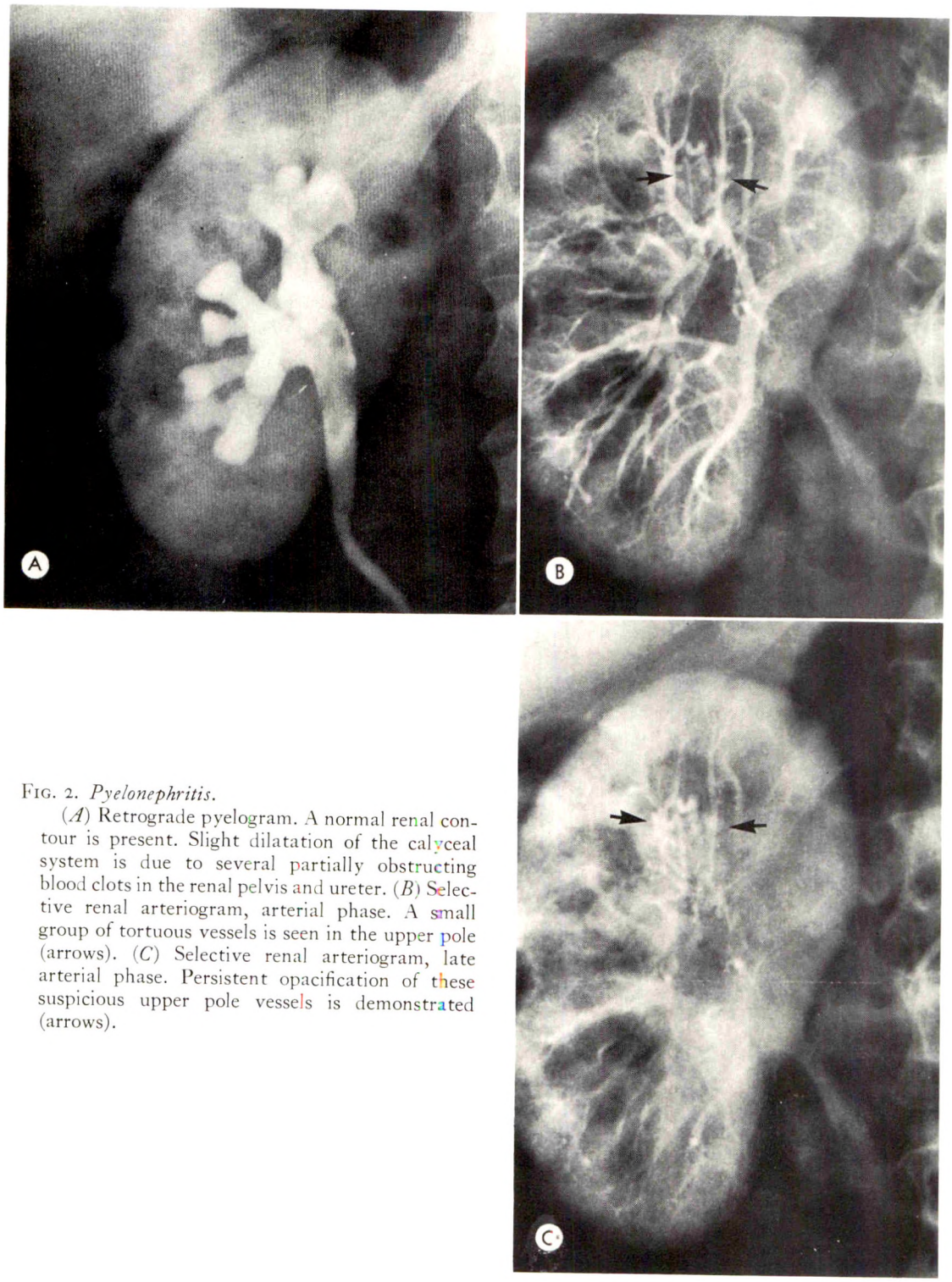


Fig. 2. *Pyelonephritis.*

(*A*) Retrograde pyelogram. A normal renal contour is present. Slight dilatation of the calyceal system is due to several partially obstructing blood clots in the renal pelvis and ureter. (*B*) Selective renal arteriogram, arterial phase. A small group of tortuous vessels is seen in the upper pole (arrows). (*C*) Selective renal arteriogram, late arterial phase. Persistent opacification of these suspicious upper pole vessels is demonstrated (arrows).

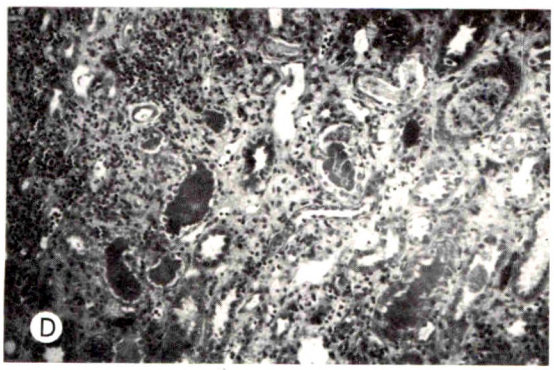


FIG. 2. (*D*) Histologic section. Pathologic examination shows acute and chronic pyelonephritis. Note the presence of inflammatory cells as well as blood in the tubules. Inflammatory cells are also present in the interstitial tissues.

renal vein in 1 patient. The presence of the lesion was confirmed by surgical exploration at another institution. The second patient was 10 years of age at the time of angiography. His admission excretory urogram was normal as was his retrograde pyelogram (Fig. 3*A*). The arterial phase of the angiography was normal (Fig. 3*B*). The venous phase demonstrated abnormal veins in the renal hilar area (Fig. 3*C*). For this reason, selective renal phlebography was performed. Tortuous pericalyceal veins were again seen as well as large dilated veins with variceal configuration extending from the main renal vein inferiorly around the ureter (Fig. 3*D*).

Surgical exploration confirmed that the bleeding was from the peripelvic portion of this venous malformation. Ligation and excision of these veins were performed.

Vascular malformations of the kidney may bleed or be detected incidentally during the evaluation of patients for other conditions.[2,5,10] In any case, demonstration of the vasculature is essential in the preoperative evaluation of such patients.

4. *Miscellaneous causes.* One patient had a small embolus to a segmental branch of the renal artery. He presented with gross hematuria and no pain. There was no history of rheumatic heart disease, myocardial infarction or arrhythmia. The admission excretory urogram showed a normal calyceal system and equal opacification bilaterally (Fig. 4*A*). Selective renal arteriography was performed, when the bloody efflux was seen to be coming from the right ureteral orifice. A persistent lucency was present extending into 2 segmental renal artery branches (Fig. 4*B*). A later film confirmed the presence of this small embolus and also showed a decreased nephrogram in that portion of the kidney supplied by the occluded branches (Fig. 4*C*).

Four patients passed small nonopaque calculi described as gravel 2 days to 4 months after angiography. Retrospective evaluation of their excretory urograms did not show filling defects or any evidence of ureteral obstruction. Renal calculi may prove to be the cause of unexplained hematuria and has been encountered by other authors.[13]

One patient proved to have focal nephritis on subsequent renal biopsy. This disease affects both kidneys. Our patient's bleeding was unilateral at the time of cystoscopy and so it is included in the series. Histologic changes in focal nephritis consist of segmental areas of glomerular hypercellularity. This form of hemorrhagic nephritis is unassociated with edema, azotemia or hypertension and is therefore considered a separate entity from glomerulonephritis.[6,12]

One patient with a large hepatoma receiving chemotherapy proved to have focal congestion and hemorrhage in the kidney at postmorten examination. There was no neoplastic involvement of the kidney.

No tumors were demonstrated by urography or angiography in this series. One patient did have a small transitional cell papilloma near the ureteral orifice detected 1 year following the initial work-up. This bladder tumor may have been missed on the original cystoscopic examination and because of its proximity to the ureteral orifice may have been the source of the bleeding originally thought to be of upper tract origin. Continued follow-up of this patient has failed to reveal any renal or ureteric lesion.

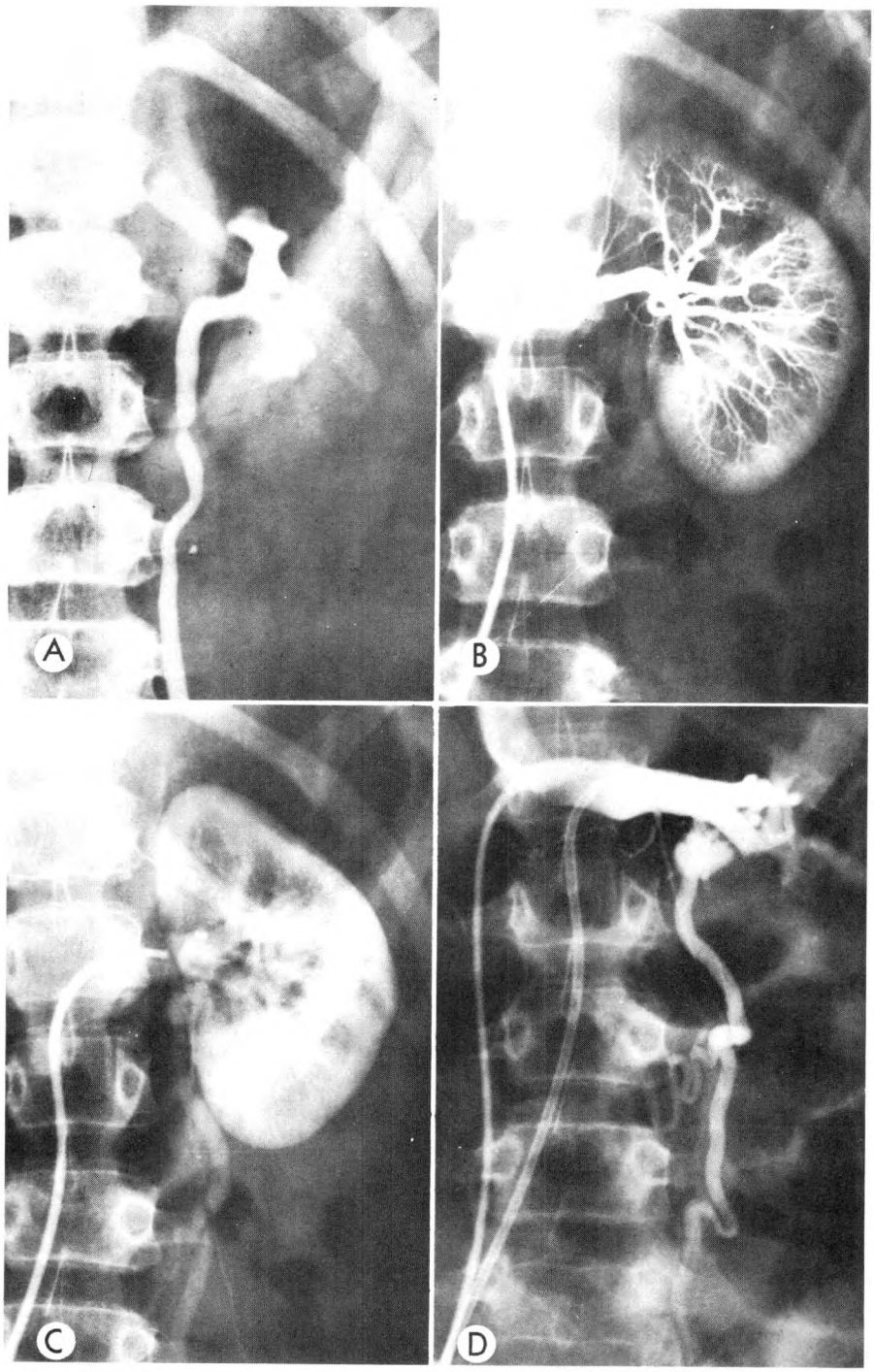
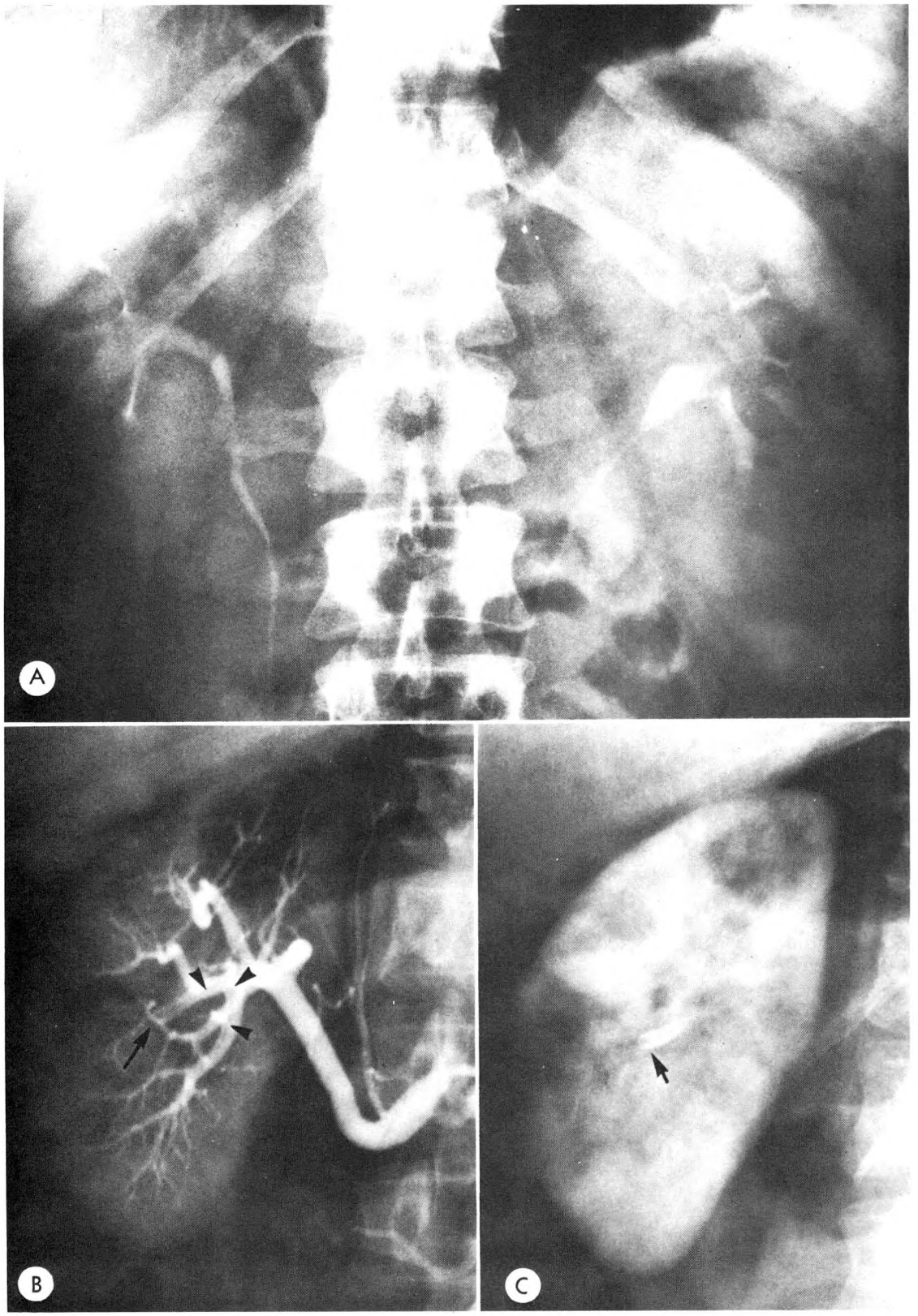


Fig. 3. *Venous malformation.*

(*A*) Retrograde pyelogram. A normal calyceal system and renal contour are seen. (*B*) Renal angiogram, arterial phase. Normal arterial vasculature is demonstrated. (*C*) Renal angiogram, venous phase. A markedly dilated vein is seen extending inferiorly from the renal hilum. (*D*) Selective renal phlebogram. Tortuous peripelvic veins are seen as well as abnormally dilated veins extending inferiorly in the region of the ureter.


A
B
C

DISCUSSION

The large majority of patients who present with hematuria will have a cause found by means of routine medical, urologic and radiologic evaluation. Careful evaluation of the patient with hematuria is essential, since there is a higher incidence of malignancy in patients with urologic symptoms and associated hematuria.[8] No cause is found after routine work-up in from 5–8 per cent of patients with hematuria.[2,15] These patients have been described as having idiopathic or "essential" hematuria. Anxiety by both the patient and clinician has led the radiologist to use all the procedures at his disposal in the work-up of these patients. For this reason, we have made it a policy over the past 7 years to study such patients by arteriography, to see if lesions not detectable by other means might be found.

Demonstration of the bleeding point by visualization of leakage of contrast material is a rare event in the nontraumatized kidney.[3,4,14] The problem of seeing such leakage at angiography is compounded by the fact that the kidney is excreting the contrast agent. We attempted to visualize leakage of contrast material in several of our patients by doing a selective renal injection after a small test injection and prior to a flush aortogram, but we failed to see any extravasation in this series.

Review of our 48 patients confirmed our impression that angiography is a low yield procedure in the evaluation of patients with unilateral renal bleeding and a negative urogram. Of the 20 patients in whom a cause for the hematuria was established, only 3 had a specific diagnosis made by arteriography. These were the 2 patients with vascular malformations and the 1 patient with an embolus. The impaired segmental arterial flow in the 2 patients with hemoglobin SA was compatible with the underlying pathology but not diagnostic. It is of interest that the 1 patient, in whom a tumor was suspected angiographically, proved to have pyelonephritis.

Many of these patients come to angiography because of the fear that a tumor might be missed by conventional urography. There are reports in the literature of a small incidence of renal carcinoma found after follow-up of patients with "essential" hematuria.[1,7] This clinical observation would seem to be evidence in favor of angiographic evaluation. In none of our cases was a renal tumor detected or has any patient in this series had a tumor discovered during the follow-up period. We included the patient with the bladder papilloma near the ureteral orifice in this series, since the tumor's relation to the original episode of hematuria remains unclear.

What of patients who bleed and have no cause demonstrated by initial clinical and radiographic evaluation as well as long term follow-up? Some of these kidneys have been studied by other authors after nephrectomy for bleeding. Careful pathologic examination of these kidneys may reveal thin walled venules beneath the uroepithelium.[19] In some cases these have been demonstrated to be the source of bleeding. MacMahon and Latorraca[16] emphasized the closeness of thin walled veins to the calyceal fornices. They were able to demonstrate calyceal-venous communication by careful sectioning of kidneys removed in 3 patients with unexplained hematuria. Similar communications were also reported by



FIG. 4. *Embolus.*

(*A*) Excretory urogram. Equal bilateral excretion is demonstrated. Some blood clot is present in the right pelvis and upper calyces. (*B*) Selective renal arteriogram, arterial phase. A persistent lucent filling defect extends into 2 segmental branches (arrowheads). One branch is completely occluded (arrow). (*C*) Selective renal arteriogram, nephrographic phase. Persistent contrast material in the occluded branch demonstrates the embolus (arrow).

Pytel.[20] Such changes might be easily overlooked in routine pathologic study of surgical specimens.

Some authors[6,12] have emphasized the role that focal glomerulonephritis may play in patients with unexplained hematuria. This condition affects both kidneys and our patients all had unilateral bleeding at the time of cystoscopy. For this reason, it is not surprising that only 1 of our patients had this entity at renal biopsy. Perhaps, repeated cystoscopic examination would have demonstrated bilateral bleeding in this patient.

Of our 28 patients with a negative angiogram and a negative follow-up, 23 had only one episode of bleeding. Although the cause for this bleeding remains unknown, routine biopsy was not performed, since the patients continued to have good renal function and no further hematuria. Some of these patients may indeed have had focal glomerulonephritis, glomerulonephritis or pyelonephritis with unilateral bleeding at the time of cystoscopy. In any case, the condition appears to have been self-limiting in these patients with no residual biochemical or urinary abnormality.

Medical management of these patients is the wisest course since surgical exploration will be negative in virtually all cases of unexplained hematuria, particularly if angiography has been part of the work-up. In 2 of our patients the ureter of the bleeding kidney was temporarily blocked by means of a catheter in an effort to increase intrapelvic pressure and stop the bleeding by a tamponading effect. The maneuver was apparently successful in 1 patient, a 6 year old boy who has not bled since this treatment. The second patient, a 41 year old woman, stopped bleeding for 8 hours following the ureteral tamponade. The importance of conservative management is best illustrated by 1 of our patients who had right-sided bleeding documented 8 times over a 7 year period. A ninth episode of bleeding was from the left side. Obviously, the original decision not to remove the kidney which first bled was correct. The crux of the matter is that a negative urogram coupled with a negative angiogram can usually be equated with a nonsurgical lesion and provides important evidence for continued conservative management of these perplexing patients.

SUMMARY

1. Angiography is a low yield procedure in the evaluation of patients with unilateral renal bleeding and a negative urogram. It is useful in the management of the rare vascular malformation or other unsuspected vascular lesion.

2. Sickle cell trait and unsuspected pyelonephritis were the commonest detectable causes of bleeding and a negative urogram in this series.

3. The major importance of the angiogram is to provide additional support in the conservative clinical management of unexplained hematuria.

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REFERENCES

1. ANDERSEN, A. J., LA COUR, and ZACHARIAE, F. Essentiell hematuri. *Ugeskrift for Laege*, 1965, *127*, 252–255.
2. BURKHOLDER, G. V., DOTIN, L. N., THOMASON, B., and BEACH, R. D. Unexplained hematuria. *J.A.M.A.*, 1969, *210*, 1729–1733.
3. CIHAK, R. J., RAINES, R. B., POLLARD, J. J., and FLEISCHLI, D. J. Spontaneous renal bleeding in hydronephrotic solitary kidney: report of case with angiographic demonstration of bleeding site. *Radiology*, 1969, *93*, 1305–1307.
4. CORNELL, S. H. Angiographic demonstration of acute renal hemorrhage. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 549–552.
5. CRUMMY, A. B., ATKINSON, R. J., and CARUTHERS, S. B. Congenital renal arteriovenous fistulas. *J. Urol.*, 1965, *93*, 24–26.
6. FERRIS, T. F., GORDON, P., KASHGARIAN, M., and EPSTEIN, F. H. Recurrent hematuria and focal nephritis. *New England J. Med.*, 1967, *276*, 770–775.
7. FLATMARK, A. Essentiell hematuri. *Nord. Med.*, 1959, *61*, 215–218.
8. GARTMAN, E. Significance of hematuria in young men. *J. Urol.*, 1956, *75*, 135–142.

9. Goodwin, W. E., Alston, E. F., and Seamans, J. H. Hematuria and sickle cell disease: unexplained, gross, unilateral, renal hematuria in Negroes, coincident with blood sickling trait. *J. Urol.*, 1950, *63*, 79–96.
10. Gunterberg, B. Renal arteriovenous malformation. *Acta radiol.* (Diag), 1968, *7*, 425–430.
11. Heller, P. Once more: pathogenic effects of sickle cell trait. Editorial. *J.A.M.A.*, 1973, *225*, 987–988.
12. Heptinstall, R. H., and Joekes, A. M. Focal glomerulonephritis. In: Ciba Foundation Symposium on Renal Biopsy. Little, Brown & Company, Boston, 1961.
13. Johnson, K. Renal angiography in patients with hematuria. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 758–765.
14. Koff, R. S., Widrich, W. C., and Robbins, A. H. Necrotizing angiitis in methamphetamine user with hepatitis B-angiographic diagnosis: five-month follow-up, results and localization of bleeding site. *New England J. Med.*, 1973, *288*, 946–947.
15. Lee, L., and Davis, E. Gross urinary hemorrhage: symptom, not disease. *J.A.M.A.*, 1953, *153*, 782–784.
16. MacMahon, H. E., and Latorraca, R. Essential renal hematuria. *J. Urol.*, 1954, *71*, 667–676.
17. Mostofi, F. K., Vorder Brugge, C. F., and Diggs, L. W. Lesions in kidneys removed for unilateral hematuria in sickle-cell disease. *A.M.A. Arch. Path.*, 1957, *63*, 336–351.
18. Perillie, P. E., and Epstein, F. H. Sickling phenomenon produced by hypertonic solutions: possible explanation for hypothenuria of sicklemia. *J. Clin. Invest.*, 1963, *42*, 570–580.
19. Priestly, J. T., and Wilbur, D. L. *Proc. Staff Meet. Mayo Clin.*, 1934, *9*, 348.
20. Pytel, A. Renal forniceal hemorrhages: their pathogenesis and treatment. *J. Urol.*, 1960, *83*, 783–789.

ADRENAL TUMORS SIMULATING INTRARENAL LESIONS*

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ADRENAL neoplasms receive their dominant arterial supply from adrenal arteries. One would expect large suprarenal tumors to cause some displacement of the subjacent kidney.

Two different adrenal neoplasms are reported which produce no renal displacement and possess an arterial supply predominantly of renal origin. Both tumors simulate intrarenal lesions.

REPORT OF CASES

CASE 1. A 20 year old Black male presented with history of excessive perspiration of approximately 4 months' duration. In association with the perspiration, the patient experienced dizzy spells and brief syncopal episodes. He denied history of headaches, diarrhea, or nervousness.

On physical examination, the patient was thin, but normally developed, with some perspiration on his brow in spite of a cool ambient temperature. The blood pressure, obtained from the right arm, while in a sitting position, was 170/120 mm. Hg. Peripheral pulses in upper and lower extremities were intact and symmetric. Cardiovascular examination revealed sinus tachycardia.

A 24 hour urine collection disclosed markedly increased excretion of urinary catecholamines and vanylmandelic acid. The anteroposterior projection of the excretory urogram was interpreted as normal. A midstream abdominal aortogram demonstrated a haphazard array of vessels projected over the left kidney. No abnormal vessels supplying the neoplasm were seen to arise directly from the aorta. Selective injection of the left renal artery in the anteroposterior projection provided a more definitive picture of the abnormal collection of vessels relating to the left kidney (Fig. 1–3). With both injections of the contrast medium (meglumine diatrizoate), the patient experienced accentuation of diastolic hypertension; this responded quickly to intravenous administration of phentolaminemethane sulfonate (Regitine, Ciba). The left inferior phrenic artery was then catheterized. Several vessels arising from the inferior phrenic artery were splayed over a mass (Fig. 4).

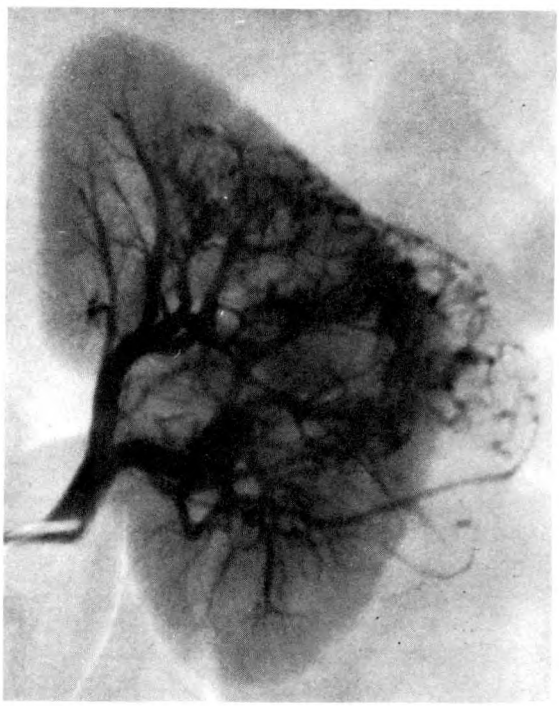


FIG. 1. Case 1. Selective renal artery injection, anteroposterior projection. An irregular tangle of vessels is noted over the midrenal area. There is virtually no distortion of intrarenal vasculature, which suggests superficial location of neoplasm.

At surgery, a large 4 × 5 × 6 cm. vascular neoplasm adherent to the anterior surface of the kidney and arising from the inferior portion of the adrenal gland was removed *in toto* and the underlying kidney preserved. Pathologic examination revealed that the tumor was a pheochromocytoma.

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The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

Postoperatively, the patient did well and returned to a normotensive state.

Case II. A 2 year old white female presented with a 15 month history of worsening acne, pubic hair development, and deepening of voice. The child had been observed to be entirely normal at the time of birth.

Physical examination showed a girl who was quite muscular and large for chronologic age. Facial acne was apparent. Pubic hair was present about the labia majora; however, no axillary hair was observed. There was slight clitoral enlargement. The vaginal mucosa showed no evidence of estrogenization. The abdomen was protuberant and a mass could be palpated in the left upper quadrant. Systolic blood pressure by palpation was 100 mm. Hg.

A 24 hour urine collection showed the 17-ketosteroids to be elevated and the 17-hydroxysteroids to be normal. One of the androgenic hormones excreted by the adrenal glands, dehydro-epiandrosterone, was elevated. Abdominal roentgenograms showed a left upper quadrant soft tissue mass containing coarse calcification (Fig. 5). The excretory urogram was interpreted as normal. Abdominal angiography demonstrated a vascular left upper quadrant tumor (Fig. 6). Selective renal artery injection disclosed that primary blood supply was derived from the renal artery (Fig. 7). Selective renal artery injection with patient in left posterior oblique position showed juxtaposition of the tumor to the kidney's surface (Fig. 8).

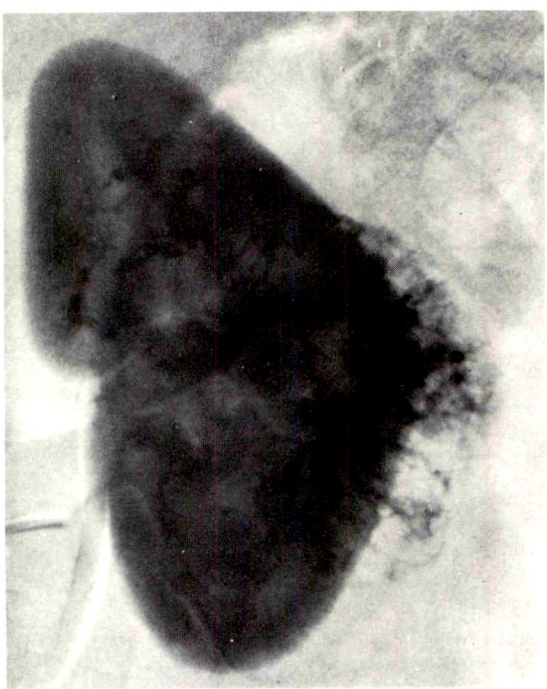


Fig. 2. Case I. Selective renal artery injection, anteroposterior projection. Late vascular phase demonstrates hypervascularity of tumor.

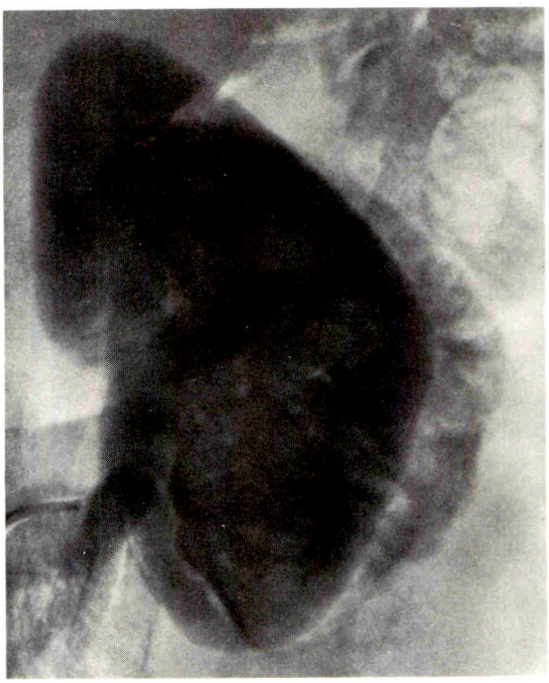


Fig. 3. Case I. Selective renal artery injection, anteroposterior projection. Venous phase of injection discloses parenchymal opacification of the neoplasm. The renal vein is identified. The vascular structure projected over the renal hilus was found to be a markedly enlarged adrenal vein at the time of surgery.

Surgery revealed a 9×6.5×4 cm. adrenal tumor. The cut surface of the tumor was brownish and lobular. Small calcific deposits were distributed throughout the neoplasm. Histologically, the tumor was an adrenal carcinoma.

The lesion was considered completely excised at the time of surgery and subsequent 2 year follow-up examination has shown no evidence of recurrent tumor.

DISCUSSION

Pheochromocytoma, as illustrated by Case I, is usually an adrenal tumor, although approximately 10 per cent are extra-adrenal and can be found in nearly any location in the body.[5] Common extra-

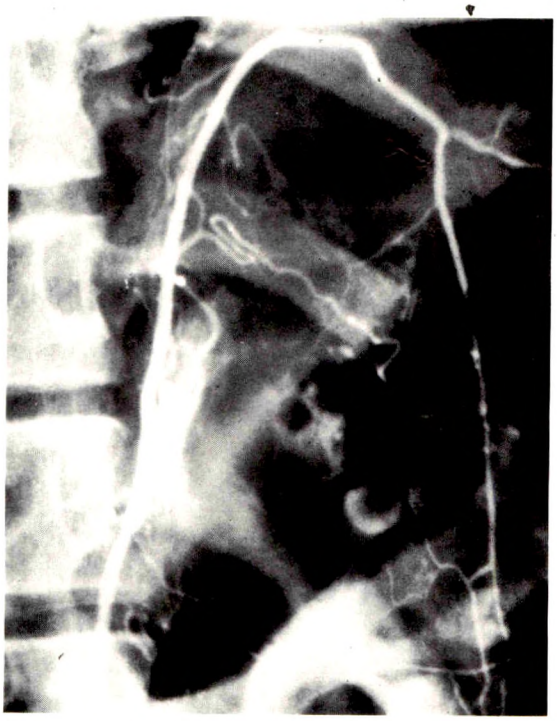


FIG. 4. Case I. Selective inferior phrenic injection. Several small vessels arising from the inferior phrenic are seen in the distribution of the adrenal tumor; however, preponderant circulation to the neoplasm is not from superior adrenal vasculature.

adrenal locations include paraspinal autonomic ganglia where accretions of chromaffin tissue occur, particularly organ of Zuckerkandl.[4] Children appear to exhibit a slightly greater incidence of extra-adrenal chromaffin tumors (28 per cent) than adults.[17] Ten to 20 per cent of pheochromocytomas are multiple;[16] multiplicity has a higher incidence in association with multiple endocrine adenomata (Sipple syndrome)[15] and in children.[17]

Adrenal carcinoma, as illustrated by Case II, can produce virilization and Cushing's syndrome. Calcification is not infrequent; this may be present in 25 per cent of tumors. The pattern of calcification is variable—flocculent, patchy, scattered, or ring-like patterns have been described.[7]

Angiography is frequently used to localize adrenal masses. Other roentgenographic methods to localize suprarenal tumors include nephrotomography, extraperitoneal pneumography,[12] adrenal venography,[13] and inferior vena cavography.[10] Pheochromocytomas and malignant cortical adrenal tumors generally possess angiographic findings, *e.g.*, neovasculature or vascular stretching.[2,3,8,9,14] Vascular supply to these tumors is typically from normal suprarenal arterial sources, *i.e.*, inferior adrenal branches from renal artery, middle adrenal branches from abdominal aorta, and superior adrenal branches from inferior phrenic artery. A few chromaffin tumors have been reported to be avascular[6] and aldosterone-secreting tumors of the adrenal cortex may be devoid of recognizable arteriographic findings.[8] Differentiation of benign and malignant adrenal tumors may be difficult. The existence of metastases or local invasion may be the only reliable hallmarks of malignancy.[1]

The 2 adrenal tumors presented are somewhat unusual because they depict primary blood supply from intrarenal vessels in addition to being projected over the midrenal areas. Characteristic displacement of the kidney was also not apparent. Cases similar to these have been described in the Swedish literature.[9] Merklin and Michels,[11]



FIG. 5. Case II. Coarse calcification within adrenal carcinoma.

in a detailed anatomic study of renal and adrenal blood supply, show the variable and complex blood supply of these organs. The adrenal glands may obtain blood through established anatomic routes *via* aorta, inferior phrenic artery, or renal arteries. Supplemental sources include lumbar, ureteric, and gonadal arteries. An intimate relationship between adrenal and renal vasculature exists. The renal artery frequently sends 2 to 5 arterial twigs to the adrenal, while superior adrenal branches arising from the inferior phrenic artery may be expected to send 1 to 4 small branches to the superior renal pole.[11] It should not be surprising that adrenal tumors potentially may obtain their primary blood supply from the renal artery. This may be expected if the tumor mass arises inferiorly within the substance of the adrenal gland. As illustrated in these 2 cases, anticipated displacement of the kidneys with sizeable suprarenal masses may not occur. This renal displacement depends entirely upon the bulk of the tumor mass; if it is low and anterior to the kidney, the neoplasm may simply drape over the surface of the under-

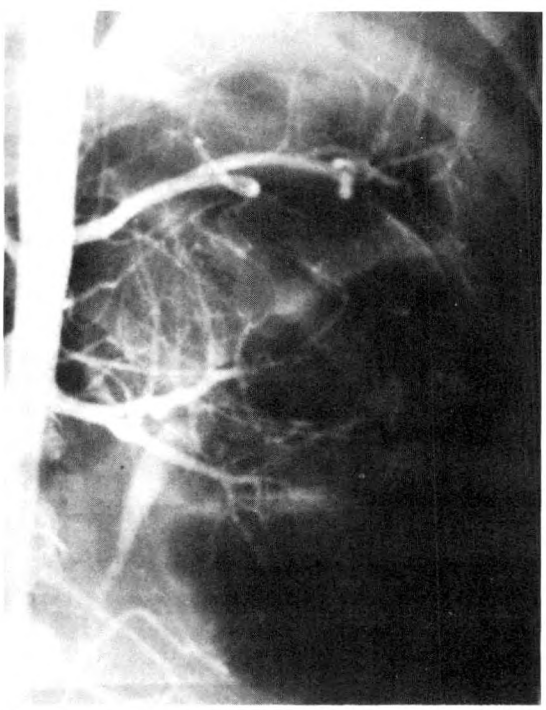


Fig. 6. Case 11. Abdominal aortogram, midstream injection. Several large, stretched midadrenal branches arise from the aorta.

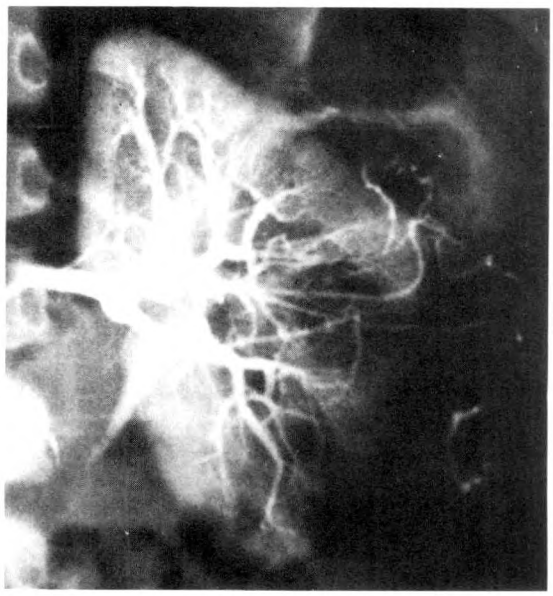


Fig. 7. Case 11. Selective renal injection, anteroposterior projection. Midarterial phase demonstrates hypervascular lesion displayed over the lateral aspect of the kidney. Minimal distortion of intrarenal vasculature is present.

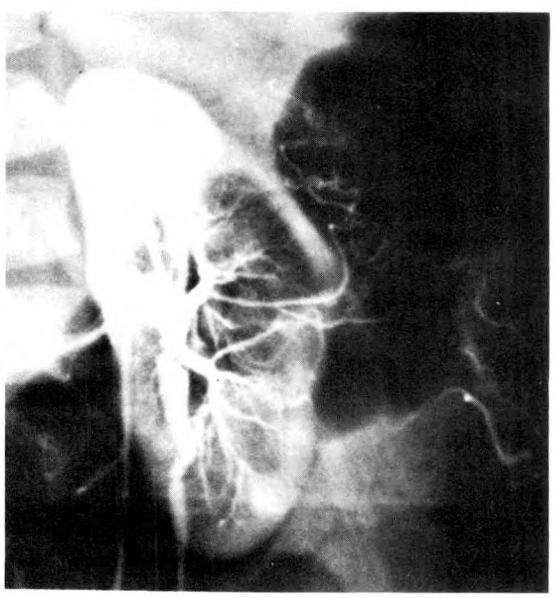


Fig. 8. Case 11. Selective renal injection, oblique projection. Midarterial phase demonstrates hypervascular neoplasm that is clearly shown to be related to the surface of the kidney and consistent with adrenal tumor.

lying organ without producing alteration of the renal axis or superior renal pole. Oblique roentgenographic projections are useful in establishing location of the mass and will provide additional evidence for diagnosis of adrenal neoplasm.

SUMMARY

The 2 large adrenal tumors discussed showed unusual but not unexpected roentgenographic findings. These include no displacement or distortion of the kidney and primary vascular supply derived from the renal artery.

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Appreciation is extended to Dr. Alphonse J. B. Palubinskas, Professor of Radiology, University of California, San Francisco, for providing material for Case II.

REFERENCES

1. BARTELS, E. C. Malignant pheochromocytoma. *S. Clin. North America*, 1959, *39*, 805–808.
2. BOIJSEN, E., WILLIAMS, C. M., and JUDKINS, M. P. Angiography of pheochromocytoma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *98*, 225–232.
3. COLAPINTO, R. F., and STEED, B. L. Arteriography of adrenal tumors. *Radiology*, 1971, *100*, 343–350.
4. CORNELL, S. H. Pheochromocytomas of lumbar sympathetic chain demonstrated by angiography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *115*, 175–178.
5. FRIES, J. G., and CHAMBERLIN, J. A. Extra adrenal pheochromocytoma: literature review and report of cervical pheochromocytoma. *Surgery*, 1968, *63*, 268–279.
6. FRY, I. K., KERR, I. H., THOMAS, M. L., and STARER, F. Value of aortography in diagnosis of pheochromocytoma. *Clin. Radiol.*, 1967, *18*, 276–281.
7. HUTTER, A. M., JR., and KAYHOE, D. E. Adrenal cortical carcinoma and clinical features of 138 patients. *Am. J. Med.*, 1966, *41*, 572–580.
8. KAHN, P. C., and NICKROSZ, L. V. Selective angiography of adrenal glands. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *101*, 739–749.
9. LANNER, L. O., and ROSENCRANTZ, M. Arteriographic appearances of phaeochromocytomas. *Acta radiol.* (Diag.), 1970, *10*, 35–48.
10. LUND, R. R., GARCIA, N. A., III, LE BLANC, G. A., GARTENLAUB, C., and RICHARDSON, J. F. Inferior vena cavography and preoperative localization of pheochromocytoma. *J. Urol.*, 1960, *85*, 768–773.
11. MERKLIN, R. J., and MICHELS, N. A. Variant renal and suprarenal blood supply with data on inferior phrenic, ureteral and gonadal arteries: statistical analysis based on 185 dissections and review of literature. *J. Internat. Coll. Surgeons*, 1958, *29*, 41–76.
12. PENDERGRASS, H. P. Retroperitoneal radiology: roentgen techniques to demonstrate pheochromocytomas and other retroperitoneal masses. *Radiol. Clin. North America*, 1963, *1*, 195–213.
13. REUTER, S. R., BLAIR, A. N., SCHTEINGART, D. E., and BOOKSTEIN, J. J. Adrenal venography. *Radiology*, 1967, *89*, 805–814.
14. ROSSI, P. Arteriography of adrenal tumours. *Brit. J. Radiol.*, 1968, *41*, 81–98.
15. SIPPLE, J. H. Association of pheochromocytoma with carcinoma of thyroid gland. *Am. J. Med.*, 1961, *31*, 163–166.
16. SJOERDSMA, A. Moderator. Pheochromocytoma: current concepts of diagnosis and treatment. Combined clinical staff conferences at the National Institute of Health. *Ann. Int. Med.*, 1966, *65*, 1302–1326.
17. STACKPOLE, R. H., MELICOW, M. M., and USON, A. C. Pheochromocytomas in children: report of 9 cases and review of first 100 published cases with followup studies. *J. Pediat.*, 1963, *63*, 315–330.

Vol. 121, No. 3

DIAGNOSIS OF PERIRENAL AND SUBCAPSULAR MASSES*

ANATOMIC-RADIOLOGIC CORRELATION

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NEW YORK, NEW YORK

THE symptoms and signs of abscess or hematoma in the subcapsular region of the kidney or in the perirenal spaces are notoriously nonspecific and the area is not accessible to the usual clinical modalities of palpation, percussion and auscultation. Diagnosis relies upon radiologic methods. However, much confusion exists regarding the terminology of these spaces and their boundaries. Anterior renal fascia, posterior renal fascia, Gerota's fascia, fascia of Zuckerkandl, fatty capsule, and capsule are all terms used and misused. Accurate roentgen localization of a subcapsular or perirenal collection is possible with an understanding of the anatomic relationships.

The purpose of this report is to define and clarify the anatomy of these extrarenal compartments and to establish the differential diagnostic criteria in the radiologic identification and localization of perirenal and subcapsular hematomas and abscesses.

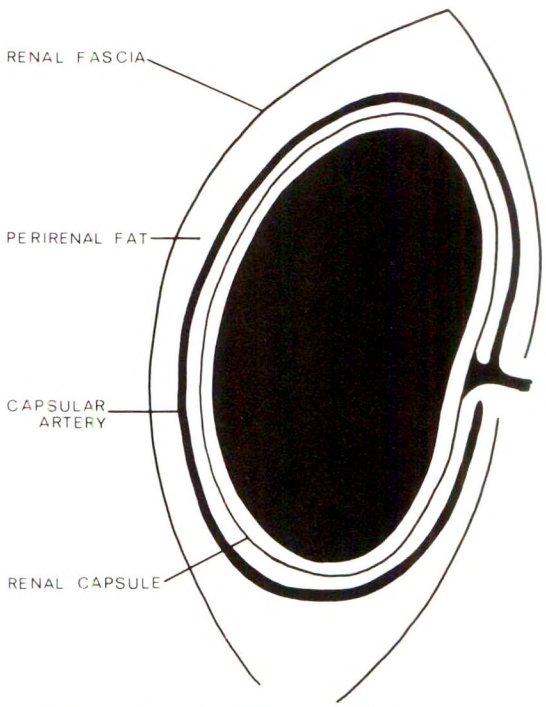


FIG. 1. Normal relationships of structures surrounding the kidney.

ANATOMIC CONSIDERATIONS

The *renal capsule* (Fig. 1) is a thin tunic which forms an intimate firm, smooth investment for the kidney. It is composed predominantly of fibrous tissue, but some smooth muscle is present within its inner layer. No adipose tissue is found between the renal parenchyma and the capsule. The capsule can be easily stripped off, but in doing so numerous fine processes of connective tissue and small blood vessels are torn through.

Perirenal fat surrounds the kidney. Its distribution is not homogeneous, but is most abundant posterior to the kidney, particularly in relation to the lower pole.[16,17]

The *capsular arteries* course through and supply primarily the perirenal fat. The somewhat confusing designation of these vessels as "capsular" is apparently derived from the old nomenclature of the perirenal fat as the "adipose capsule of the kidney." They are composed of three basic pathways —superior, middle (recurrent and perforating), and inferior capsular arteries.[2] A prominent arterial arcade is formed within the perirenal fat lateral to the kidney which communicates with renal branches perforating through the capsule.

The *renal fascia* is a condensation of the extraperitoneal connective tissue of me-

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dium or dense consistency. It is composed of two layers, anterior (fascia of Zuckerkandl) and posterior (Gerota's fascia), which envelop the kidney and perirenal fat. Laterally, these layers fuse in the flank before continuing as the single lateroconal fascia.[4,16] In the frontal projections, the renal fascial layers at their level of fusion lateral to the kidney present a plane parallel with the roentgen beam for their visualization. Medially, the renal fascia fuses with the psoas fascia and blends with the dense mass of connective tissue around the great vessels.[16–18] Superiorly, above the adrenal glands, they fuse with the diaphragmatic fascia. Inferiorly, they blend with the iliac fascia.

Pararenal fat constitutes the remainder of the extraperitoneal adipose tissue, extending between the anterior renal fascia and the peritoneum in front, and between the posterior renal fascia and transversalis fascia behind.[16]

REPORT OF CASES

CASE I. This 54 year old white female presented 3 weeks prior to admission with polydipsia, polyuria and polyphagia. Work-up revealed adult onset diabetes mellitus with diabetic acidosis. Physical examination disclosed a left upper quadrant mass and urinalysis showed 40–50 white blood cells per high power field (WBC/HPF), with many bacteria.

A plain film roentgenogram of the abdomen revealed a large soft tissue mass in the region of the lower pole of the left kidney. In addition, a linear soft tissue density projected lateral to the upper pole (Fig. 2*A*). Intravenous urography showed prompt bilateral renal function. On the left, there was evidence of a mass in the upper pole medially, and the lower pole laterally and probably medially. The strip-like density lateral to the upper pole was again evident (Fig. 2*B*). Intravenous drip urography with tomography documented these changes more clearly (Fig. 2, *C* and *D*). It was now evident, however, that a mass or masses were present in the perirenal space around the lower half of the kidney. This process displaced the renal fascia outward, accounting for the strip-like density and somewhat compressed the renal margin which otherwise appeared intact. Another mass compressed the upper pole medially. Arteriography (Fig. 2*E*) documented some hypervascularity to the process associated with the inferior and lateral portions of the kidney. The upper pole mass displaced the renal arteries but showed no neovascularity.

A left radical nephrectomy was performed, with a surgical postoperative diagnosis of hypernephroma. However, pathologic examination (Fig. 2*F*) revealed 3 perirenal abscesses, each measuring approximately 5×4 cm. and containing chocolate, foul-smelling pus. The upper pole perirenal abscess appeared somewhat organized. A thick wall was associated with the outer margin of the abscess lateral to the lower pole. No tumor was present and the parenchyma of the kidney, although distorted, appeared normal.

Comment. Renal fascia displacement is identifiable on the plain roentgenograms and urogram, and is clearly detailed on nephrotomography. Displacement is maximal over the largest perirenal abscess but the fascia can be seen to be deflected laterally at some distance from this. Hypervascularity is associated with the inflammatory process.

CASE II. This 52 year old female was admitted for evaluation of gross hematuria, mild left flank pain, and low grade temperature.

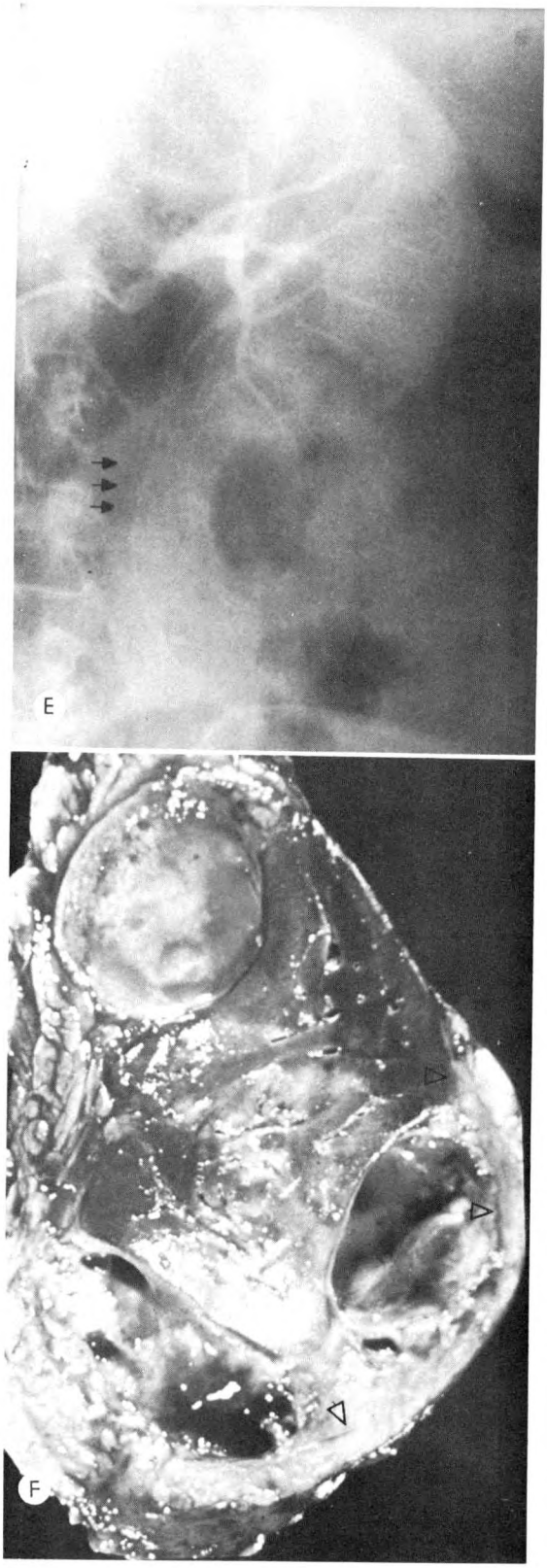
Intravenous urography (Fig. 3*A*) demonstrated local mass widening of the minor calyces in the upper pole of the left kidney. In addition, renal fascia was visualized lateral to the lower pole. The lateral margin of the kidney in this area appeared somewhat indistinct, but it was difficult to ascertain whether the renal fascia was abnormally displaced.

Renal arteriography (Fig. 3, *B* and *C*) demonstrated multiple cysts within the left kidney. Thickened renal fascia could now be identified displaced away from the renal substance of the lower pole. The superior capsular artery was also displaced from the kidney as it approached this area. On the right, a small cortical cyst in the lower pole was also noted.

At surgery, pus was immediately apparent upon opening the renal fascia. There was considerable adherence of the renal fascia and perirenal fat to the kidney. Multiple loculations of pus were found in the perirenal fat. These originated from at least 3 infected renal cysts. These cysts measured 2 to 3 cm. in diameter, were on the surface of the kidney, and con-



Fig. 2. Case 1. (*A*) Plain film roentgenogram shows a large mass in relationship to the lower pole of the left kidney. Displaced renal fascia is seen as a strip-like density (arrows) lateral to the upper pole. (*B*) Intravenous pyelogram. There is mass displacement of the renal pelvis and upper collecting system laterally and inferiorly. Most of the infundibula and calyces are not opacified. The lower pole of the kidney and the adjacent psoas muscle are not visualized. The renal fascia (arrows) is displaced from the upper renal margin. (*C* and *D*) Nephrotomograms demonstrate perirenal mass displacing the renal fascia (solid arrows) and flattening the renal margin. In its upper portion, the displaced renal fascia approaches the renal contour (upper arrows). The thickened lateral wall of the perirenal mass itself is seen (open arrows). Another nonopaque mass compresses the upper pole medially.



tained thick pus. The perirenal abscesses were drained.

Comment. Renal arteriography, by vascular opacification of the renal fascia, allows it to be identified as thickened and displaced. The capsular artery is also deviated laterally.

CASE III. This 76 year old white male presented with recent left lower quadrant abdominal pain, tenderness, and a palpable mass. Routine laboratory examination was unrevealing.

Intravenous urography (Fig. 4*A*) demonstrated a large mass in intimate association with the lateral and inferior borders of the left kidney. It extended inferiorly to the level of the iliac crest and displacement of the renal fascia outwards could be identified.

Selective renal arteriography (Fig. 4*B*) showed extension of the perforating middle capsular arteries into the large mass which caused some compression of the renal margin. No localized abnormal intrarenal vasculature was identifiable.

Aortography in the late phase (Fig. 4*C*) revealed several irregular vessels arising from the lower lumbar arteries on the left coursing in relation to the mass.

Retroperitoneal hemorrhage or, in view of the lumbar artery vascularization, neoplasm was considered.

At surgery, a large collection of blood distended the perirenal space, contained within the cone of renal fascia. Nephrectomy was performed and the pathologic examination disclosed a $3 \times 5 \times 4$ cm. infiltrating papillary adenocarcinoma of the lower renal pole. Extension through the renal capsule had obviously resulted in the perirenal hemorrhage.

Comment. The perirenal hematoma distends the cone of renal fascia to the level of the iliac crest. This is characteristic of large accumulations.[16,17] Identification of the displaced renal

FIG. 2. Case I. (*E*) Selective renal arteriogram. Ureteropelvic branches are displaced medially (arrows). Mild irregular hypervascularity is seen within the large mass associated with the lower pole. Perforating middle capsular arteries extend into the perirenal space from the lateral margin of the kidney. There is downward displacement of upper pole branches. (*F*) Gross specimen. Three large perirenal abscesses compress the kidney and displace the thickened renal fascia (arrowheads).

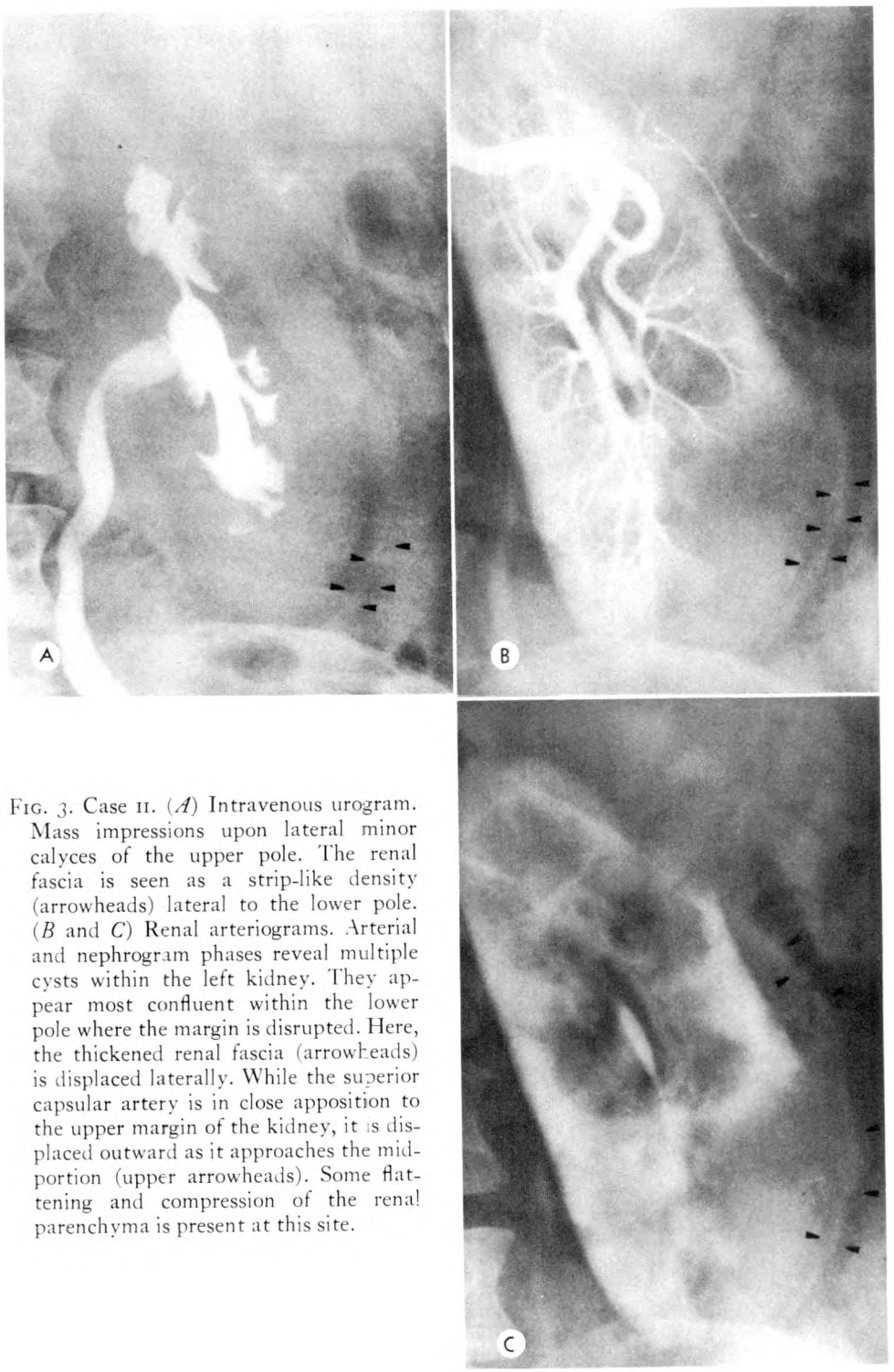


Fig. 3. Case 11. (*A*) Intravenous urogram. Mass impressions upon lateral minor calyces of the upper pole. The renal fascia is seen as a strip-like density (arrowheads) lateral to the lower pole. (*B* and *C*) Renal arteriograms. Arterial and nephrogram phases reveal multiple cysts within the left kidney. They appear most confluent within the lower pole where the margin is disrupted. Here, the thickened renal fascia (arrowheads) is displaced laterally. While the superior capsular artery is in close apposition to the upper margin of the kidney, it is displaced outward as it approaches the midportion (upper arrowheads). Some flattening and compression of the renal parenchyma is present at this site.

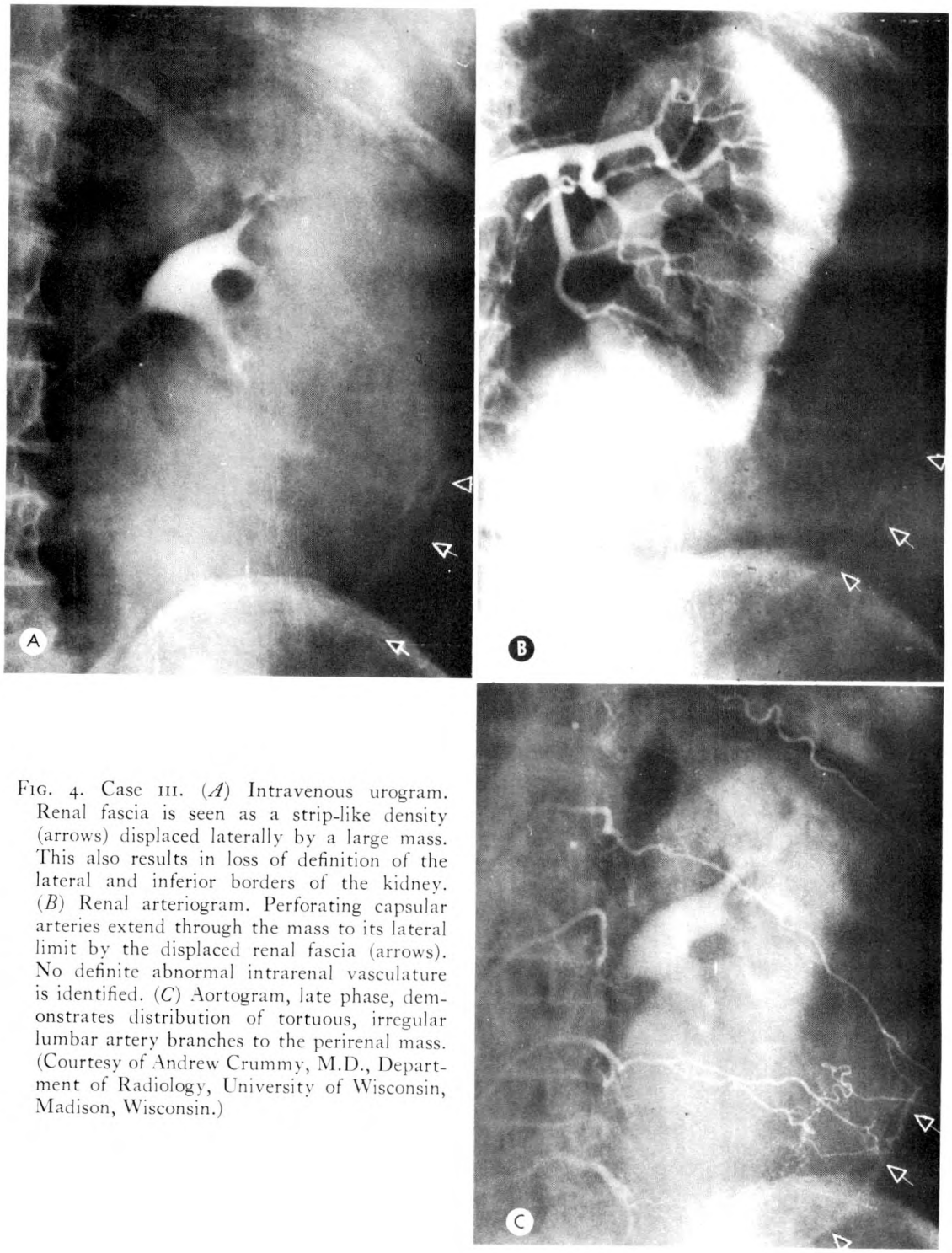


FIG. 4. Case III. (*A*) Intravenous urogram. Renal fascia is seen as a strip-like density (arrows) displaced laterally by a large mass. This also results in loss of definition of the lateral and inferior borders of the kidney. (*B*) Renal arteriogram. Perforating capsular arteries extend through the mass to its lateral limit by the displaced renal fascia (arrows). No definite abnormal intrarenal vasculature is identified. (*C*) Aortogram, late phase, demonstrates distribution of tortuous, irregular lumbar artery branches to the perirenal mass. (Courtesy of Andrew Crummy, M.D., Department of Radiology, University of Wisconsin, Madison, Wisconsin.)

fascia localizes the process to the perirenal space. Some degree of hypervascularity from the perforating middle capsular arteries and lumbar arteries is not surprising and should not be confused as a sign of diffuse extraperitoneal neoplasm. The source of the perirenal hemorrhage, a small renal carcinoma, remained occult throughout the roentgen studies.

Case IV. This 64 year old white male was previously admitted to another hospital with a 3 week history of right flank pain and fever. He was found to have a nonfunctioning kidney and to some extent responded satisfactorily to antibiotic treatment. Past medical history included an abdominal perineal resection several years previously for carcinoma of the rectum with positive lymph nodes, followed by a course of radiotherapy.

On admission, physical examination revealed a blood pressure of 190/100 mm. Hg. No abdominal masses were palpable. Urinalysis was unremarkable. Chest roentgenogram and a metastatic survey were negative.

Intravenous urography (Fig. 5*A*) now demonstrated bilateral renal function. However, on the right, there was lateral compression of the kidney which also showed some hydronephrosis.

Nephrotomography (Fig. 5, *B* and *C*) demonstrated a large mass associated with the posterolateral portion of the right kidney displacing the pelvis and calyces medially without evidence of direct invasion of the upper collecting system. Thickened renal capsule was identified circumscribing the non-opaque mass.

Selective renal arteriography (Fig. 5, *D–F*) documented arcuate displacement of the superior capsular artery paralleling the displacement of the renal capsule itself. The kidney itself was markedly compressed, but appeared intrinsically intact.

Right retrograde ureteral catheterization past the perivesical area during cystoscopy was attempted, but was unsuccessful.

At surgery, drainage of the subcapsular collection was planned. The perirenal fat was found to be quite indurated and was dissected with difficulty from the renal capsule. This was quite fluctuant and thick. A large subcapsular hematoma was present and deep pyelonephritic scars were evident on the surface of the kidney. A right nephrectomy was performed.

Pathologic examination confirmed a large, partially organized subcapsular hematoma. A thickened capsule measuring up to 8 mm. was present. A 1.2 cm. resolving abscess in the midportion of the kidney communicated with the hematoma. Hydronephrosis and generalized pyelonephritis were present.

Blood pressure after operation was 110/70 mm. Hg and remained at that level through a 3 month follow-up.

Comment. This large subcapsular collection is confined by the tense renal capsule and severely compresses the underlying parenchyma, resulting in a Page kidney. The displaced capsule is readily identified by nephrotomography and conforms precisely to the collection as further outlined by the renal compression. The level of displacement of the capsular arcade parallels exactly that of the renal capsule.

Case V. This 3½ year old female was evaluated for a 1 month history of fever, anorexia and increasing lethargy. A right upper quadrant mass was palpable. Leukocytosis was present, but urinalysis showed only 0–1 WBC/HPF.

Intravenous urography (Fig. 6) demonstrated a mass within the lower pole of the right kidney with displacement of the collecting system. The kidney appeared enlarged. Another large mass was evident causing minimal flattening of the upper pole and outlined on its external aspect by a curvilinear strip-like density. The findings were interpreted as most compatible with a necrotic Wilms' tumor. Inferior vena cavography showed some displacement but no invasion.

At surgery, a large subcapsular abscess containing creamy white pus and some old blood clot was found. The abscess had clearly dissected the capsule off the kidney. It communicated with a large infected thick-walled intrarenal cyst through a small tract along the lateral border of the kidney. It was evident that the cyst accounted for the calyceal deformity, and the subcapsular collection also posterior to the kidney accounted for the renal magnification. Drainage and excision of both were accomplished.

Comment. The tense, displaced renal capsule is clearly identifiable on intravenous urography. The effects of total body opacification in a child probably enhances its contrast visualization.

Case VI. This 33 year old white female, with a history of systemic lupus erythematosus for 17 years, was admitted for accidental coumadin overdosage. She had been discharged 5 days

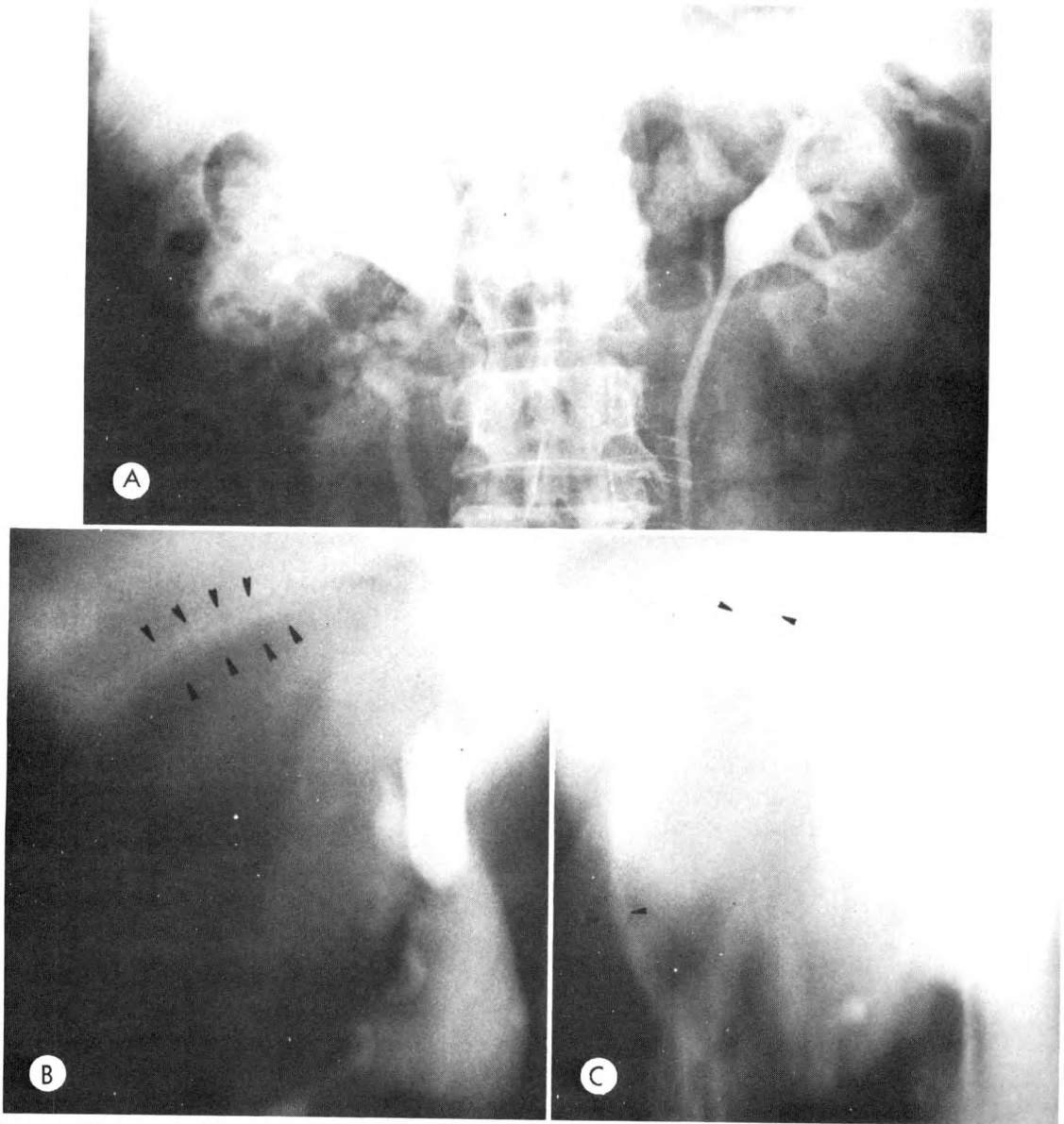


FIG. 5. Case IV. (*A*) Intravenous urogram. The right kidney is displaced medially. Dilatation of the collecting system and ureter is present. (*B* and *C*) Nephrotomograms at different levels demonstrate the displaced and thickened renal capsule (arrowheads) confining the nonopaque mass which is compressing the lateral contour of the kidney.

previously from another hospital after treatment for acute thrombophlebitis of the leg and had mistakenly been taking 20 mg. coumadin daily since.

Examination revealed acute distress from marked left flank pain and tenderness and multiple ecchymoses on the abdomen and extremities. Blood pressure was 90/60 mm. Hg, pulse 155, hematocrit 21 per cent, prothrombin time 70 seconds, and the urine was grossly bloody.

Intravenous urography with nephrotomography demonstrated distinct separation of both the renal capsule and renal fascia from the left lower pole indicating both a subcapsular and a perirenal collection of blood. Some mild irregularity of contour and compression were evident in the subjacent renal parenchyma (Fig. 7, *A* and *B*). Arteriography showed displacement of

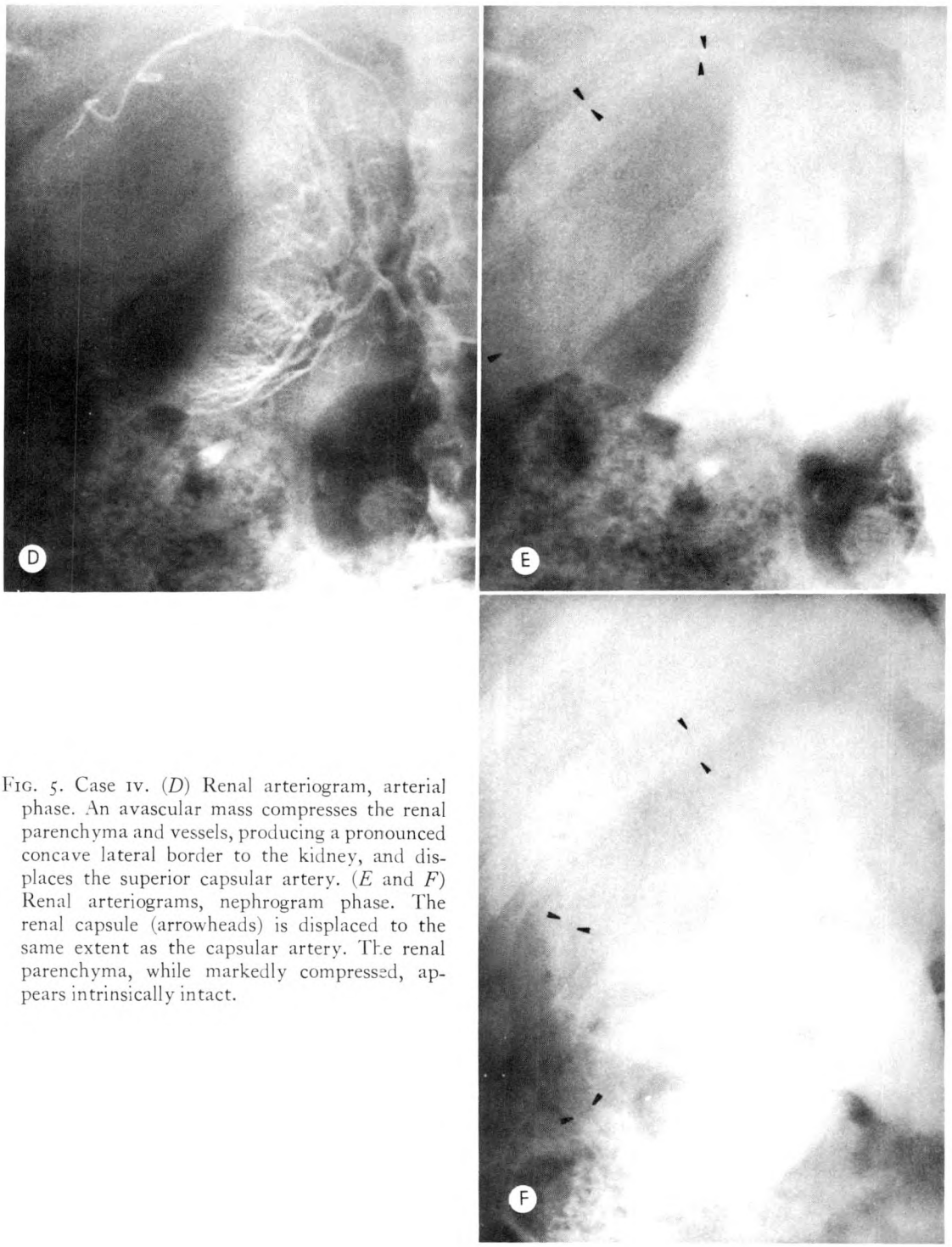


Fig. 5. Case IV. (*D*) Renal arteriogram, arterial phase. An avascular mass compresses the renal parenchyma and vessels, producing a pronounced concave lateral border to the kidney, and displaces the superior capsular artery. (*E* and *F*) Renal arteriograms, nephrogram phase. The renal capsule (arrowheads) is displaced to the same extent as the capsular artery. The renal parenchyma, while markedly compressed, appears intrinsically intact.

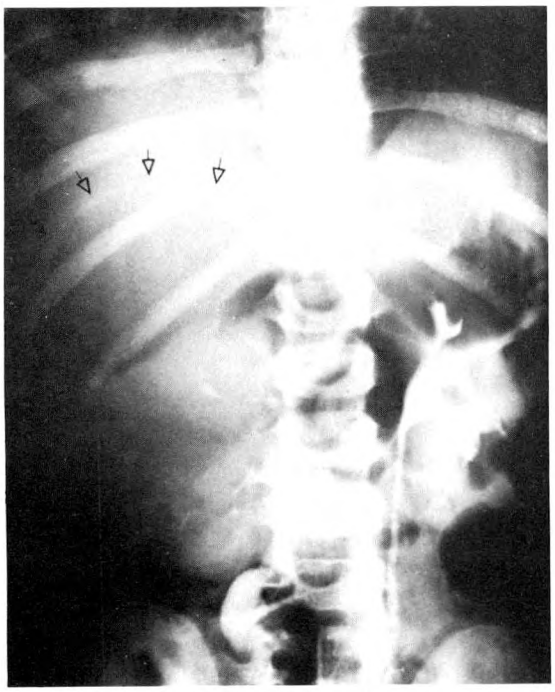


FIG. 6. Case v. On the right, the calyces are displaced by a large, relatively lucent mass in the lower half of the kidney. The renal capsule (arrows) is displaced superiorly by a prominent collection which also results in mild compression of the underlying renal margin. The apparent enlargement of the right kidney is secondary to magnification since the kidney is also pushed forward. Decreased function is evident.

the capsular artery from the lower pole (Fig. 7*C*).

She was treated with vitamin K and fresh frozen plasma, with an excellent response.

Follow-up nephrotomography 13 days later showed no abnormalities. Specifically, there was no evidence of renal fascial or capsular displacement.

Comment. Simultaneous displacement of both renal capsule and fascia can be identified. Since virtually all perirenal collections originate from the kidney, some degree of capsular displacement may also be present but this is probably too localized in most cases to be identified. Hypervascularity accompanies the process.

CASE VII. This 53 year old white female underwent a total abdominal hysterectomy and bilateral salpingo-oophorectomy for uterine fibroids. Eight days postoperatively, she developed fever, right flank pain, and evidence of urinary tract infection.

Intravenous urography with nephrotomography (Fig. 8) demonstrated obstruction at the right ureterovesical junction with dilatation of the collecting system. Extravasation from an upper pole calyceal fornix extended laterally and superiorly into the renal parenchyma and then beyond the renal capsule into the perirenal fat. The renal fascia was displaced laterally. Some peripelvic lymphatic opacification was also identifiable.

The partial distal ureteral obstruction was attributed to edema or operative trauma. Pain and fever rapidly subsided following antibiotics and the patient was discharged from the hospital.

A follow-up intravenous urogram, 1 month postoperatively, showed only minimal narrowing of the distal right ureter at the level of the bladder with a short localized tortuous segment. Function was prompt and there was no evidence of caliectasis, extravasation, or perirenal mass.

Comment. This type of urine extravasation directly across the capsule into the perirenal fat has been noted only rarely in the past.[26] Contrast material distribution first parallels the dependent lateral border of the kidney, but urine extravasation throughout the perirenal fat is evidenced by the lateral displacement of the renal fascia.

DISCUSSION

Perirenal abscesses are almost invariably secondary to a site of renal infection which perforates through the capsule to contaminate the perirenal fatty compartment. The organisms most commonly involved include *E. coli*, *Aerobacter aerogenes* and the *Clostridia* group.[6,17] In children, occasionally, perirenal abscess may be a consequence of hematogeneous spread from a remote infection such as furunculosis.[6,17]

Extrarenal hematomas, whether subcapsular or perirenal in location, are generally considered either traumatic or spontaneous (nontraumatic). Over 300 cases have been reported in the literature.[26] Polkey and Vynalek[23] reported a comprehensive study of the causes of spontaneous hematomas. Of 178 cases reviewed, the location was subcapsular in 18.5 per cent and extracapsular (perirenal or pararenal or a combination of the 2) in 81.5 per cent. Lesions of the kidneys and its blood vessels accounted

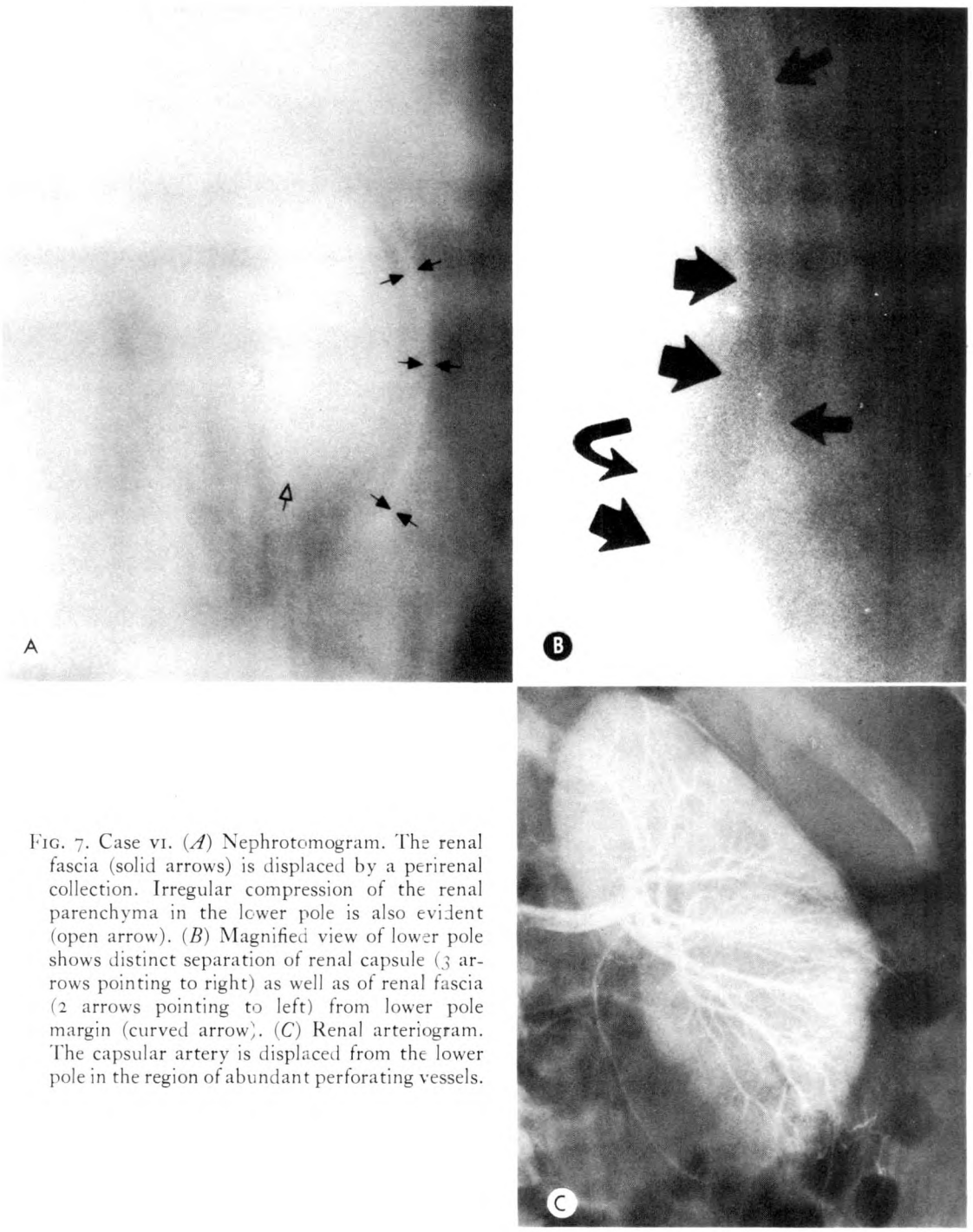


Fig. 7. Case VI. (*A*) Nephrotomogram. The renal fascia (solid arrows) is displaced by a perirenal collection. Irregular compression of the renal parenchyma in the lower pole is also evident (open arrow). (*B*) Magnified view of lower pole shows distinct separation of renal capsule (3 arrows pointing to right) as well as of renal fascia (2 arrows pointing to left) from lower pole margin (curved arrow). (*C*) Renal arteriogram. The capsular artery is displaced from the lower pole in the region of abundant perforating vessels.

for 92 per cent of the cases. The underlying etiologies in the order of their relative frequency included nephritis, neoplasms, aneurysms of the renal artery, arteriosclerosis, hydronephrosis, periarteritis nodosa, tuberculosis, renal cysts, and blood dyscrasias.

In recent years, a surprisingly large number of cases of extrarenal hemorrhage due to periarteritis nodosa and neoplasm have been reported.[22,24,25,28] It has been pointed

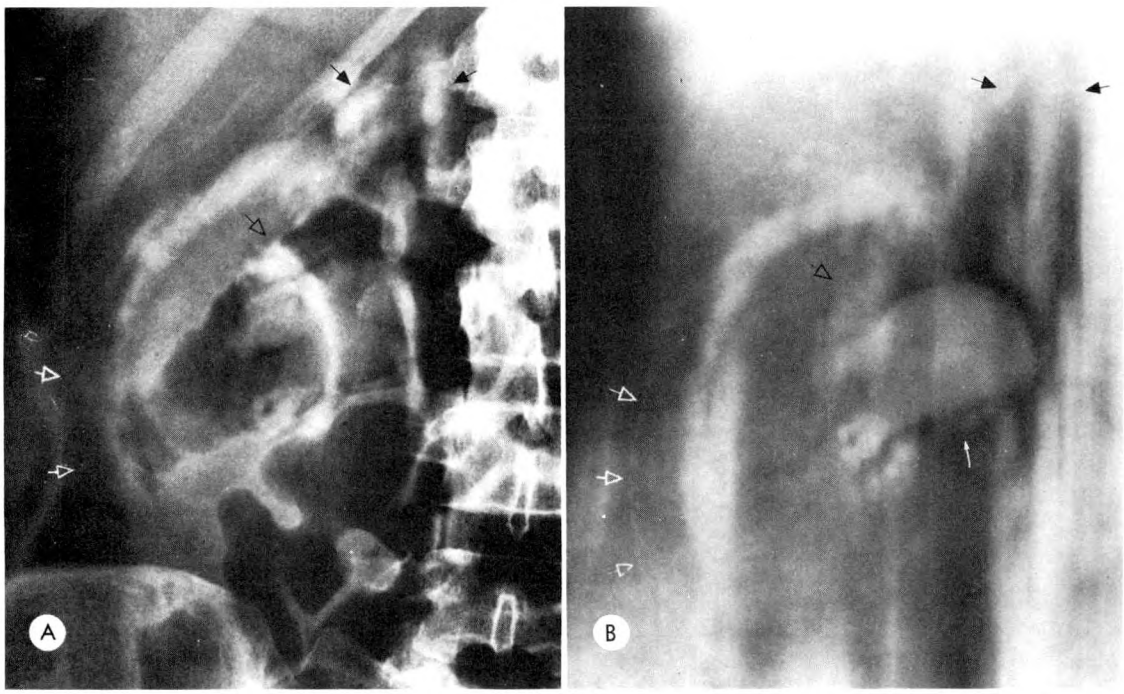


FIG. 8. Case VII. (*A*) Intravenous urogram and (*B*) nephrotomogram demonstrate extravasation from an upper pole calyx (open black arrow), part of a dilated obstructed collecting system. The extravasation extends directly across the renal parenchyma, through the capsule, and into the perirenal fat. Here it resides along the lateral contour of the kidney, following its plane of obliquity, and extends superiorly around the adrenal gland (solid black arrows). Renal fascia is displaced laterally (open white arrows). Peripelvic lymphatic opacification can also be identified (small white arrow).

out that many of the earlier cases diagnosed as nephritis may have been actually periarteritis nodosa or lupus erythematosus. One mechanism of hematoma formation begins with cortical infarcts.[20,28] The hemorrhage may be confined by the relatively rigid capsule; at other times the blood immediately breaks through the capsule but is confined within the dimensions of the renal fascia. A hematoma within the distensible perirenal compartment can develop to an enormous size before sufficient pressure occurs to cause tamponade of the bleeding site. Spontaneous subcapsular or perirenal hemorrhages masking occult and often surprisingly small renal tumors are also being recognized with increasing frequency.[24,25,28]

The clinical diagnosis of subcapsular or perirenal abscess or bleeding is rarely made. Signs and symptoms are often subtle, delayed, nonspecific or misleading.

With acute bleeding, the clinical picture may consist of pain, tenderness and rigidity which may be associated with nausea, vomiting and abdominal distention. Concomitant signs of internal bleeding may be present, but this may be manifested only by a drop in the hemoglobin or hematocrit. A mass may not be palpable, especially if the hematoma lies posteriorly to the kidney. If the hematoma is subcapsular, it may not become particularly large because of the confining effect of the renal capsule. At other times, the capsule may show considerable elasticity and the collection may approximate the size of the kidney itself[25] (Case IV). With sudden and profuse hemorrhages, many types of retroperitoneal and intraperitoneal catastrophies may be mimicked. If the hematoma extends downward in the retrocecal region, the patient may be explored for an acute appendicitis.[12,14] Rarely, perforation into the peritoneal

cavity may occur causing a generalized peritonitis, or a massive intraperitoneal hemorrhage.

With subacute or chronic bleeding, pain may not be a conspicuous feature and the principal findings may be only anemia and perhaps a palpable mass.

Hypertension may result from the constrictive renal effects of a large subcapsular[5,13] or perirenal[10] hematoma, producing the "Page kidney."[21] As illustrated by Case IV, the hypertension may be easily corrected by decompression or nephrectomy.

The diagnosis of subcapsular or perirenal hemorrhage or abscess can be indicated on plain film roentgenography or intravenous urography, and can be clearly documented by nephrotomography or angiography. Localization of the process to a specific extrarenal compartment is based on recognition of characteristic changes involving the renal capsule, renal fascia, kidney margin, and capsular arteries. These are illustrated in Figure 9, and are as follows:

(1) *Visualization of the displaced renal capsule or fascia.* Either of these structures, displaced outward from the renal margin, can be seen as a strip-like density 1–4 mm. thick. Visualization of the renal fascia can be achieved on plain film roentgenography and urography in cases of perirenal abscess or hematoma (Fig. 2, *A* and *B*; 3*A*; and 4*A*). In diffuse processes of the perirenal space, it may be recognized over a considerable length (Fig. 10). At times, urography will demonstrate a displaced renal capsule (Fig. 6).

Opacification of the displaced renal capsule and fascia is achieved in nephrotomography and arteriography, presumably because of their vascularity. Nephrotomographic identification of the capsule in cases of spontaneous subcapsular hemorrhage was first reported by Frank and Wieche[7] in 1967, and its diagnostic value further substantiated by Pollack and Popky[24] in 1972. Contrast is further afforded by the nonopaque abscess or hematoma on one side and radiolucent fat on the other. We have noted a type of displacement which, when present, distinguishes subcapsular from perirenal collections. The renal capsule is sharply deflected over the margin of a sub-

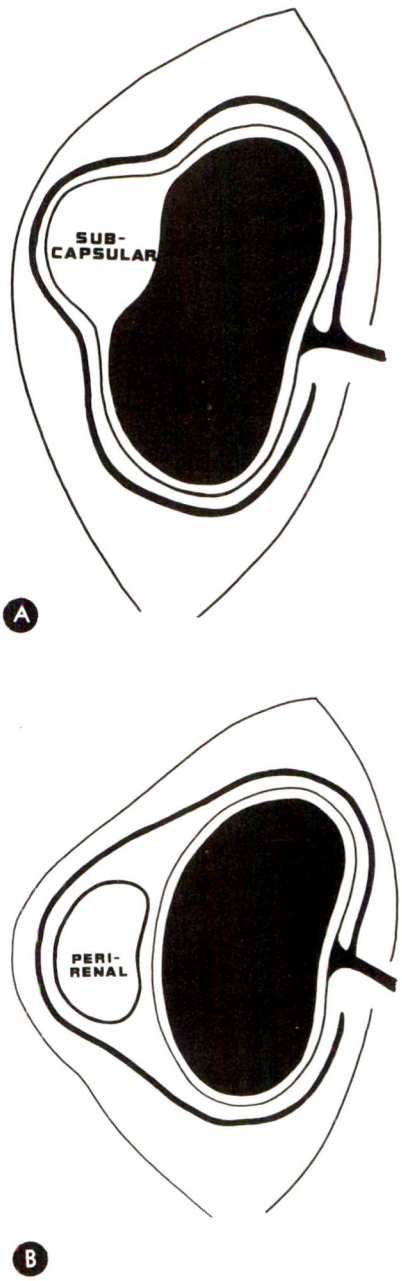


Fig. 9. Major findings distinguishing (*A*) a subcapsular from (*B*) a perirenal collection. Note particularly the relationships of the displaced renal capsule, perirenal fascia and capsular arteries at the borders of the mass. Flattening of the underlying renal parenchyma is more commonly found in subcapsular collections.

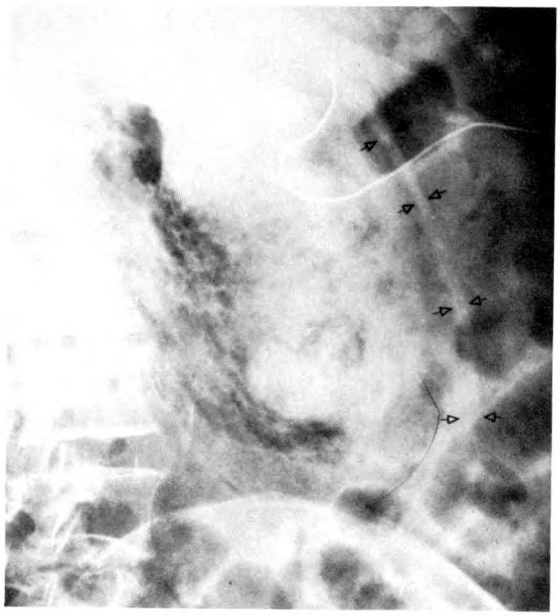


FIG. 10. The displaced renal fascia is identifiable as a long strip-like density (arrows) in a case of gas-producing perirenal abscess.

capsular mass. Even with huge collections, its point of displacement intimately conforms to the border of the hematoma (Fig. 5*B*; and 5*E*). It appears that this is a consequence of the relatively rigid inelastic nature of the renal capsule. In contrast, renal fascia is often displaced laterally from the margin of the kidney at some distance from a coalescent perirenal collection. Its maximum deflection is at the site of the perirenal hematoma or abscess but it can be seen to depart from close to the renal border both above and below (Fig. 2, *C* and *D*). This is somewhat analogous to Codman's triangle which is seen in bone lesions and to the myelographic sign of an extradural defect. It is a reflection of the yielding perirenal fat and renal fascia.

(2) *Visualization of the hematoma or abscess.* The subcapsular or perirenal collection is easily recognized as a nonopaque mass between the opacified renal parenchyma on one side and the elevated renal capsule or fascia on the other. Within the perirenal space, the most common site for coalescence and localization of a fluid collection is in relationship to the lower pole posterolaterally. This is a consequence of the rich perirenal fat in this area and the path of least tissue resistance.[6,16,17]

(3) *Flattening and compression of the kidney.* While this may occur with tense perirenal collections, it is more typical of subcapsular hematoma. The pressure exerted by a confined subcapsular hematoma typically causes flattening of the subjacent renal parenchyma.

(4) *Displacement of capsular arteries.* We do not believe that "the location of the capsular arteries will help to distinguish between subcapsular and perinephric bleeding"[24] since they may be displaced externally in either condition. Examples of conspicuous arcuate displacements and stretching of the capsular artery system in cases of both subcapsular[24] and perirenal[3,19] hematomas and abscesses have been amply documented in the literature. Rather, it appears that the level of displacement of the capsular arterial arcade is the angiographic key to the differential diagnosis. If the vessel conforms closely to the border of the mass, a subcapsular collection is indicated (Fig. 5*D*). If deviation of the capsular artery begins at some distance from the extrarenal mass, a perirenal collection is indicated (Fig. 3*B*).

As a differential point, it must be recognized that the capsular arteries will be separated from the cortical margin in cases of renal atrophy (Fig. 11). Perirenal adipose replacement in instances of acquired shrinkage of the kidney tends to increase the distance between the capsular artery and the atrophied kidney.[15] This separation can be easily distinguished from a subcapsular or perirenal mass displacing the capsular artery.

The marked increase in intrarenal vascular resistance produced particularly by a large subcapsular hematoma may result in striking slowing of arterial flow with failure of opacification of the small intrarenal and capsular vessels. Watnick and associates[28] have recently illustrated this in a case harboring a 3 cm. hypernephroma as the cause of the bleeding.

(5) *Structure and function of the renal collecting system.* Distortion of the calyces and renal pelvis may accompany any gross displacement of the kidney itself. Since perirenal abscesses result from a site of renal infection which has perforated through the capsule, chronic inflammatory changes involving the calyces may be evident. An intrarenal abscess or hematoma may produce mass displacement upon the collecting system.

Failure of excretion of contrast medium on the involved side ("unilateral anuria") can result from the compression of either a perirenal[9] or subcapsular[1,11,28] hematoma. Koehler and associates[11] have recently emphasized the association of subcapsular hematomas with the enlarged nonfunctioning kidney.



Fig. 11. In a case of chronic atrophic pyelonephritis, the nephrographic phase of a selective renal arteriogram shows the capsular arcade widely separated from the shrunken renal parenchyma.

These observations in the localization of subcapsular and perirenal collections are of considerable help in planning the most appropriate therapy. It is important to consider whether the underlying cause is due to predominantly unilateral renal disease or to a systemic condition involving both kidneys. With the recognition that most extrarenal abscesses are secondary to an infection of the kidney, conservative treatment perhaps with surgical drainage or nephrectomy is determined by the extent of involvement. In subcapsular or perirenal hematoma in the presence of a renal tumor, hydronephrosis, renal artery aneurysm, lithiasis or unilateral renal tuberculosis, nephrectomy does not present any significant long term problem. On the other hand, if the etiology is nephritis, arteriosclerosis, periarteritis nodosa or a blood dyscrasia and nephrectomy must be performed as a life-saving procedure, careful follow-up observation of the remaining kidney must be maintained. If the hematoma is not large and adequate visualization of the bleeding site can be obtained so that it can be controlled with sutures, a biopsy of the involved renal tissue may be preferable to nephrectomy.

SUMMARY

The anatomy and terminology of the structures involved in the compartmentalization of subcapsular and perirenal hematomas and abscesses are defined and clarified. These relationships include the renal capsule, perirenal fat, capsular arteries and renal fascia.

The etiology, pathogenesis and clinical features are considered.

The specific roentgen features permitting the identification and localization of these processes on plain film roentgenography, urography, nephrotomography and arteriography are described and illustrated.

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REFERENCES

1. ASCHNER, P. W., and KLINGER, M. E. Subcapsular renal hemorrhage causing anuria of single kidney. *J. Urol.*, 1951, *65*, 777–780.
2. BOIJSEN, E. Angiographic studies of anatomy of single and multiple renal arteries. *Acta radiol.*, 1959, Suppl. 183.
3. ČAPEK, V., and FOJTÍK, F. Significance of selective renal angiography in injuries of kidney in childhood. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1963, *90*, 75–80.
4. CONGDON, E. D., and EDSON, J. N. Cone of renal fascia in adult white male. *Anat. Rec.*, 1941, *80*, 289–313.
5. ENGEL, W. J., and PAGE, I. H. Hypertension due to renal compression resulting from subcapsular hematoma. *J. Urol.*, 1955, *73*, 735–739.
6. EVANS, J. A., MEYERS, M. A., and BOSNIAK, M. Acute renal and perirenal infections. *Seminars in Roentgenol.*, 1971, *6*, 274–291.
7. FRANK, I. N., and WIECHE, D. R. Nephrotomographic appearance of spontaneous subcapsular hemorrhage. *Radiology*, 1967, *89*, 477–482.
8. GEROTA, D. Beiträge zur Kenntnis des Befestigungsapparates der Niere. *Arch. Anat. Entwcklngsgesch.*, 1895, 265–286.
9. HARROW, B. R., and SLOANE, J. A. Nontraumatic perirenal hemorrhages. *South. M. J.*, 1964, *57*, 363–369.
10. HELLEBUSCH, A. A., SIMMONS, J. L., and HOLLAND, N. Renal ischemia and hypertension from constrictive perirenal hematoma. *J.A.M.A.*, 1970, *214*, 757–759.
11. KOEHLER, P. R., TALNER, L. B., FRIEDENBERG, M. J., and KYAW, M. M. Association of subcapsular hematomas with nonfunctioning kidney. *Radiology*, 1973, *101*, 537–542.
12. MACKENZIE, A. R. Spontaneous subcapsular renal hematoma: report of case misdiagnosed as acute appendicitis. *J. Urol.*, 1960, *84*, 243–245.
13. MARSHALL, W. H., JR., and CASTELLINO, R. A. Hypertension produced by constricting capsular renal lesions ("Page" kidney). *Radiology*, 1971, *101*, 561–565.
14. MARTIN, K. W. Spontaneous circumrenal haematoma: review and report of two cases. *Brit. M. J.*, 1949, *1*, 1118–1120.
15. MEYERS, M. A., FRIEDENBERG, R. M., KING, M. C., and MENG, C-H. Significance of renal capsular arteries. *Brit. J. Radiol.*, 1967, *40*, 949–956.
16. MEYERS, M. A., WHALEN, J. P., PEELLE, K., and BERNE, A. S. Radiologic features of extraperitoneal effusions: anatomic approach. *Radiology*, 1972, *104*, 249–257.
17. MEYERS, M. A. Acute extraperitoneal infection. *Seminars in Roentgenol.*, 1973, *8*, 445–464.
18. MITCHELL, G. A. G. Renal fascia. *Brit. J. Surg.*, 1950, *37*, 257–266.
19. OLSSON, O., and LUNDERQUIST, A. Angiography in renal trauma. *Acta radiol.* (Diag.), 1963, *1*, 1–23.
20. OUIMET-OLIVA, D., CHARRON, J., and BÉLANGER, R. Renal infarct with perirenal hematoma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *98*, 70–76.
21. PAGE, I. H. Production of persistent arterial hypertension by cellophane and perinephritis. *J.A.M.A.*, 1939, *113*, 2046–2048.
22. PETERSON, C., JR., WILLERSON, J. T., DOPPMAN, J. L., and DECKER, J. L. Polyarteritis nodosa with bilateral renal artery aneurysms and perirenal haematomas: angiographic and nephrotomographic features. *Brit. J. Radiol.*, 1970, *43*, 62–66.
23. POLKEY, H. J., and VYNALEK, W. J. Spontaneous non-traumatic perirenal and renal hematomas: experimental and clinical study. *Arch. Surg.*, 1933, *26*, 196–218.
24. POLLACK, H. M., and POPKY, G. Spontaneous subcapsular renal hemorrhage: its significance and roentgenographic diagnosis. *J. Urol.*, 1972, *108*, 530–533.
25. PRESMAN, D., ROLNICK, D., and PORTNEY, F. Spontaneous perinephric hematoma secondary to renal tumor. *Am. J. Surg.*, 1961, *102*, 586–593.
26. SILVER, T. M., KOFF, S. A., and THORNBURY, J. Unusual pathway of urine extravasation associated with renal colic. *Radiology*, 1973, *109*, 537–538.
27. USON, A. C., KNAPPENBERGER, S. T., and MELICOW, M. M. Nontraumatic perirenal hematomas: report based on 7 cases. *J. Urol.*, 1959, *81*, 388–394.
28. WATNICK, M., SPINDALA-FRANCO, H., and ABRAMS, H. L. Small hypernephroma with subcapsular hematoma and renal infarction. *J. Urol.*, 1972, *108*, 534–536.

Vol. 121, No. 3

ANGIOGRAPHIC FEATURES OF ALPORT'S SYNDROME*

HEREDITARY NEPHRITIS

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IN 1927 Alport described the association of hereditary nephritis with deafness, now known as Alport's syndrome.[1] Since then several families with the syndrome have been described.[3,6,7,9,10] A recent study suggested that hereditary nephritis is not uncommon and should be considered in every patient with unexplained hematuria, proteinuria or renal failure.[3] The wide variety of renal pathology that occurs in this entity, however, makes its early diagnosis difficult.

Although an increased number of angiograms are being obtained in patients with renal parenchymal disease, the angiographic abnormalities which occur in Alport's syndrome have been described in only 1 case report.[4]

We have recently studied 2 patients with Alport's syndrome by angiography in different stages of the disease.

REPORT OF CASES

CASE I. A 16 year old boy was first seen in mid-August, 1973, because of bilateral flank pain and general malaise. A urine sediment was most consistent with glomerular disease. He was admitted to Wayne County General Hospital in October, 1973, with proteinuria and hematuria. An audiogram revealed severe, bilateral, high-pitched hearing loss. His father had no renal problems but had hearing loss; his mother was hypertensive and proteinuric; 2 sisters had chronic urinary tract infections and 1 cousin had a nephrotic syndrome. Urinalysis revealed 3+ protein, 5–10 red blood cells per high power field, 2–3 white blood cells per high power field, a rare red cell cast and hyaline and granular casts. During the hospitalization his serum creatinine increased from 3.2 to 5.6 mg. per cent and his blood urea nitrogen from 24.5 to 31 mg. per cent. His blood pressure varied from 150/100 to 170/100 mm. Hg. The creatinine clearance rate was 16 and 20 cc./min. on 2 occasions.

A renal biopsy in October, 1973, showed increased mesangial matrix and sclerosis of the glomeruli with prominent epithelial crescent formation in some glomeruli. Slight periglomerular fibrosis, focal tubular atrophy and interstitial aggregates of foam cells were present. The pattern was considered to be consistent with hereditary nephritis.

Renal angiography, done 4 weeks after renal biopsy because of persistent hematuria, revealed no arteriovenous fistula. The angiographic features of both kidneys were identical (Fig. 1, *A–C*). The main renal arteries, segmental arteries and the proximal interlobar arteries were normal. The distal interlobar arteries showed moderate tortuosity and marked pruning. Virtually no arcuate arteries were seen. The nephrogram was faint and homogeneous with indistinctness of the corticomedullary junction. The renal blood flow was extremely slow, approximately 2 cc. per second in either kidney. The size of both kidneys was normal.

CASE II. A 21 year old woman had an abnormal urinary sediment at age 2½ years. A hearing defect, first noted at the same age, became progressive. She had an episode of gross hematuria at age 10 requiring hospitalization. Proteinuria and hematuria were persistent during a subsequent pregnancy and several hospitalizations were necessary for similar symptoms following delivery. She also had intermittent facial and pedal edema and frequent flank pain. A 14 year old sister and 10 year old brother had renal insufficiency and hearing deficits; 2 second cousins on the maternal side, 1 female and 1 male, died at 18 and 19 years of renal failure.

A renal biopsy in 1971 revealed progressive glomerulonephritis with significant interstitial changes, consistent with hereditary nephritis.

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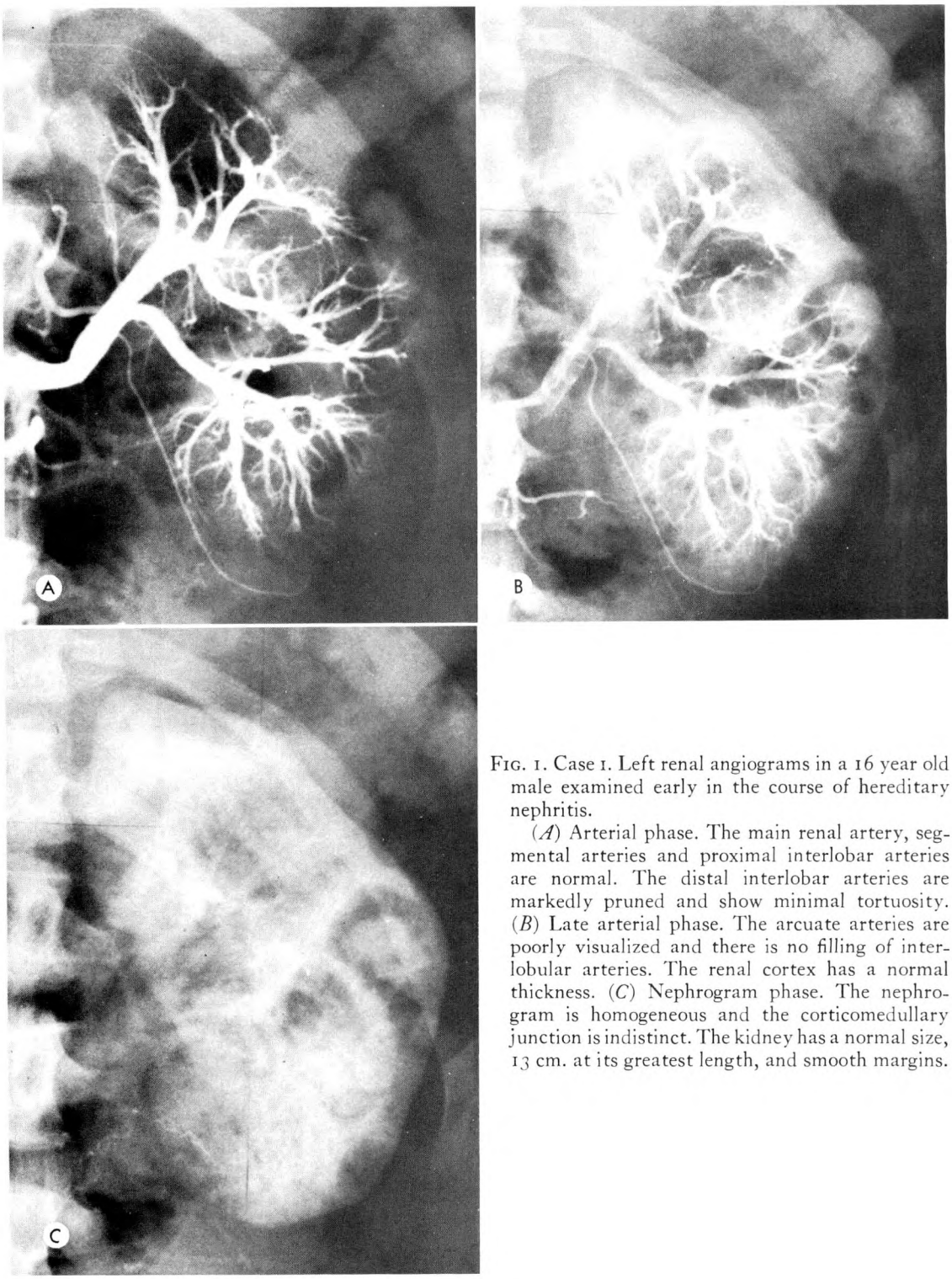


FIG. 1. Case 1. Left renal angiograms in a 16 year old male examined early in the course of hereditary nephritis.

(*A*) Arterial phase. The main renal artery, segmental arteries and proximal interlobar arteries are normal. The distal interlobar arteries are markedly pruned and show minimal tortuosity. (*B*) Late arterial phase. The arcuate arteries are poorly visualized and there is no filling of interlobular arteries. The renal cortex has a normal thickness. (*C*) Nephrogram phase. The nephrogram is homogeneous and the corticomedullary junction is indistinct. The kidney has a normal size, 13 cm. at its greatest length, and smooth margins.

The laboratory studies during the hospitalization in March, 1973, included a blood urea nitrogen of 45 mg. per cent and creatinine of 8.8 mg. per cent.

Renal and pelvic angiographies were done as part of a transplantation evaluation. The angiographic features were identical in both kidneys (Fig. 2, *A* and *B*). The kidneys were small, measuring 8 cm. in length. The main renal and segmental arteries showed only a reduced caliber. The interlobar arteries were extremely tortuous and crowded, and the arcuate arteries were poorly seen. The corticomedullary junction was indistinct, resulting in poor delineation of the renal cortex. Renal blood flow was extremely slow, less than 1 cc. per second. The nephrogram was homogeneous and the renal margins smooth. The findings were typical of end-stage disease.

DISCUSSION

Alport's syndrome is characterized by hereditary renal disease, frequently coexisting with nerve deafness, and occasionally with ocular abnormalities. Although the sex distribution of the disease is equal, the affected male has a much worse prognosis and usually dies of renal failure at an early age, whereas the affected female usually lives to an old age. The clinical manifestations of the renal disease include episodic hematuria, proteinuria, pyuria and renal failure. Hypertension may occur late in the disease. Nerve deafness is characterized by a progressive, severe, symmetric, high frequency hearing loss. Ocular abnormalities include congenital cataracts, con-

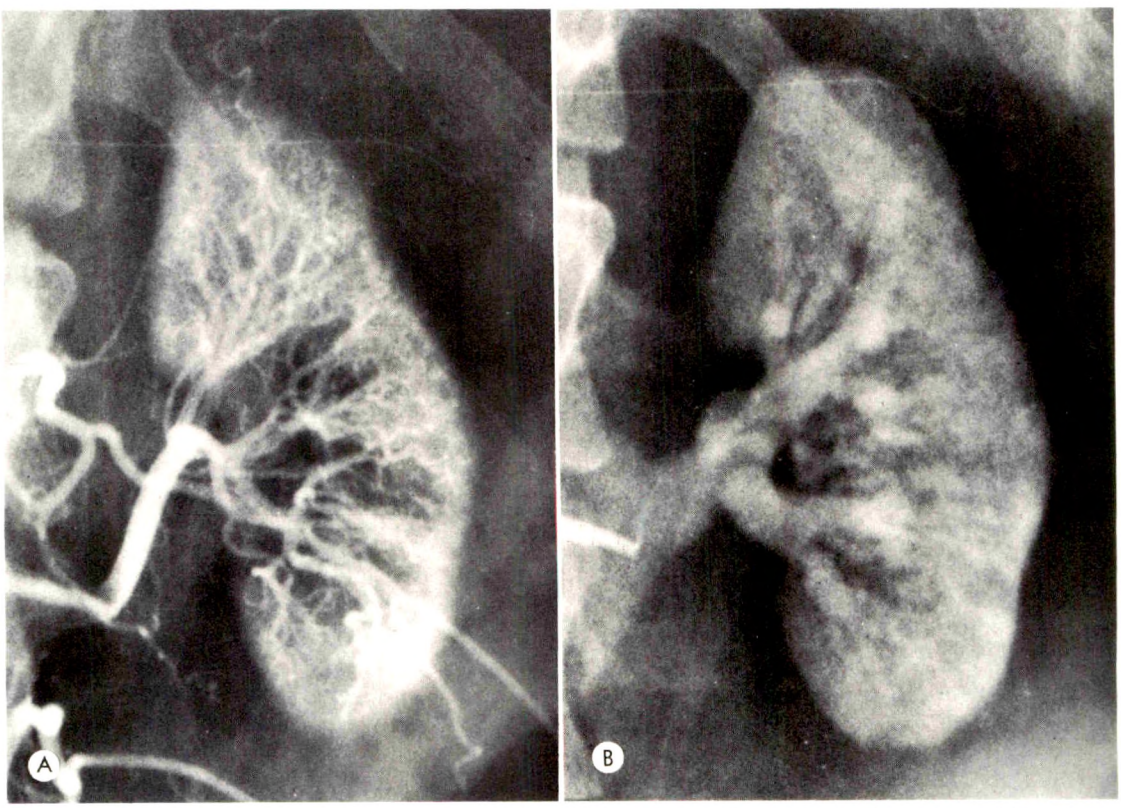


Fig. 2. Case 11. Left renal angiograms in a 21 year old female examined late in the course of hereditary nephritis.

(*A*) Arterial phase. The main renal and segmental arteries appear normal, but the interlobar arteries are crowded and show marked tortuosity. (*B*) Nephrogram phase. Although the kidney is small and contracted, measuring 8 cm. in length, the margins are smooth and the nephrogram homogeneous. The veins are well demonstrated because of a prolonged injection of contrast medium.

genital nystagmus, myopia, spherophakia and anterior lenticonus.

The renal pathology in Alport's syndrome varies greatly, depending on the stage of the disease. In moderately advanced cases the kidneys are shrunken and atrophied, with a marked decrease in weight. The surfaces are finely granular and pale, but the coarse scarring seen in chronic pyelonephritis almost invariably is lacking. The renal pelvis and calyces are normal. The microscopic findings vary from typical interstitial pyelonephritis in some patients to glomerulonephritis in others. More commonly a "mixed" nephritis is present, with alternating areas of severe tubular atrophy and tubular dilatation combined with patchy glomerular involvement. The presence of foam cells throughout the cortex, but most numerous near the corticomedullary junction, is an important feature.[6,7,10]

The strong family history of renal disease and auditory deficit in our patients is typical of this syndrome. The mechanism of inheritance is unsettled. Two major hypotheses are: (1) transmission by partially sex-linked dominant inheritance; (2) transmission by preferential segregation and chromosomal association.[10]

Our patients were studied at different stages in the progression of their disease. The first, with a 2 month history of renal insufficiency, was examined early. His markedly slowed blood flow of less than 2 cc. per second to each kidney is consistent with his severely impaired glomerular filtration rate. The pruning of the interlobar arteries, nonfilling of intralobular arteries, indistinctness of the corticomedullary junction and the homogeneous, faint nephrogram reflect renal vascular bed obliteration in the cortex due to severe glomerular disease. The normal cortical thickness and the normal renal size indicate the relatively early stage of the disease. Thus, the angiographic features of this case are the same as those reported for a rapid progressive glomerulonephritis.[8] The only other reported case of Alport's syndrome studied by angiography[4] has angiographic changes similar to this patient.

Our second patient has small, contracted kidneys, typical of end-stage renal parenchymal disease. This agrees with the patient's long history. However, one feature worth mentioning is the bilaterally smooth renal margins observed at angiography. This correlates well with the finely granular surface and absence of coarse scarring seen at autopsy in patients with hereditary nephritis.[6]

Problems in differential diagnosis include glomerulonephritis, arteriolar nephrosclerosis and pyelonephritis. The findings parallel those of glomerulonephritis in both the early and late stages. Arteriolar nephrosclerosis may be difficult to differentiate from both glomerulonephritis and hereditary nephritis. In arteriolar nephrosclerosis the main renal arteries are normal or dilated, the interlobar arteries are markedly pruned, and fine lucencies occur in the nephrogram, caused by microinfarcts.[5,8] Although interstitial pyelonephritis is histologically a component of hereditary nephritis, the angiographic findings are different from those of chronic pyelonephritis. In hereditary nephritis the kidneys maintain a smooth cortical margin and homogeneous distribution of contrast medium in the parenchymal phase, in contrast to the contracted, scarred kidneys and unhomogeneous accumulation of contrast medium in the parenchymal phase which is typical of chronic pyelonephritis.

Our patients did not have intravenous urography. Such studies in several patients with the disease reported in the literature have been normal. Chang *et al.*,[2] however, showed a case with diffuse enlargement of both kidneys and minimal blunting of the minor calyces.

CONCLUSION

The angiographic findings in 2 patients with Alport's syndrome are described.

The first patient, examined at an early stage of the disease, had pruning of interlobar arteries with indistinctness of the

corticomedullary junction, as well as slowed renal flow compatible with severe glomerular disease. These findings are similar to the angiographic appearance of rapidly progressive glomerulonephritis.

The second patient had the typical findings of end-stage renal parenchymal disease.

The marked differences in the angiographic appearances reflect the different stages of the syndrome in which the patients were examined.

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REFERENCES

1. Alport, A. C. Hereditary familial congenital haemorrhagic nephritis. *Brit. M. J.*, 1927, *1*, 504–506.
2. Chang, C. H., Gaskell, J. R., and Chun, C. S. Alport's syndrome. *South. M. J.*, 1972, *65*, 635–636.
3. Chazan, J. A., Zacks, J., Cohen, J. J., and Garella, S. Hereditary nephritis: clinical spectrum and mode of inheritance in five new kindreds. *Am. J. Med.*, 1971, *50*, 764–771.
4. Demetropoulos, K. C., Hoskins, P., and Rapp, R. Angiographic study of hereditary nephritis (Alport's syndrome). *Radiology*, 1973, *108*, 539–540.
5. Halpern, M. Angiography in chronic renal disease and renal failure. *Radiol. Clin. North America*, 1972, *10*, 467–494.
6. Heptinstall, R. H. Pathology of the Kidney. Little, Brown & Company, Boston, 1966.
7. Krickstein, H. I., Gloor, F. J., and Balogh, K. Renal pathology in hereditary nephritis with nerve deafness. *A.M.A. Arch. Path.*, 1966, *82*, 506–517.
8. Mena, E., Bookstein, J. J., and Gikas, P. Angiographic diagnosis of renal parenchymal disease. *Radiology*, 1973, *108*, 523–532.
9. Perkoff, G. T., Nugent, C. A., Dolowitz, D. A., Stephens, F. E., Carnes, W. H., and Tyler, F. H. A follow-up study of hereditary chronic nephritis. *A.M.A. Arch. Int. Med.*, 1958, *102*, 733–746.
10. Strauss, M. B., and Welt, L. G. Disease of the Kidney. Little, Brown & Company, Boston, 1971.

ACUTE RENAL FAILURE AFTER ANGIOGRAPHY*

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THE potential nephrotoxic effects of the earlier angiographic contrast media such as acetrizoate (Urokon) are well recognized, and acute renal failure was one reported clinical manifestation of this toxicity.[13,23,25] The newer contrast media, such as the iothalamate and diatrizoate salts, however, have been considered relatively non-nephrotoxic when used in doses up to 5 mg./kg.[11,31]

This report describes 8 instances of acute oliguric renal failure occurring after angiography. Currently used contrast media were given in doses generally considered not excessive and in no patient was bilateral renal artery catheterization performed.

METHOD

During the years 1968 through 1972, 8 patients undergoing evaluation at the Mayo Clinic developed acute renal failure after an angiographic procedure. These 8 patients quite likely do not represent the total number of complications of this nature because our data were obtained by retrospective review of records of patients for whom nephrologic consultation was requested. During this same period, approximately 7,400 patients underwent angiographic studies.

Percutaneous arteriography was performed by a modified Seldinger technique[18] *via* the femoral or, occasionally, the axillary artery. Cardiac catheterization and coronary arteriography were done by the brachial cut-down technique. The contrast media used were sodium and methylglucamine diatrizoates (Renografin 76 per cent; Renovist) and sodium iothalamate (Vascoray). The routine preparation included an oral laxative on the previous evening, abstinence from oral intake 8 to 10 hours before angiography, a local anesthetic at the artery puncture site, and intravenous titration of Innovar. Vital signs were monitored frequently during the procedure and for at least 1 hour in the recovery room.

The type of angiographic procedure, indications, and findings are listed in Table 1. Two patients had angiocardiography for low-output heart failure, 2 had aortography for atherosclerotic or hypertensive disease, 2 had aortography with unilateral renal arteriography for suspected renal masses, and 2 had selective arteriography of the celiac axis (one for arteriovenous fistulas and the other for hepatic vein obstruction). Angiographic renal findings included benign renal cysts in 1 patient and bilateral renal artery stenosis in 2 others.

Acute renal failure was documented, 1 to 3 days after angiography, by a marked change in serum creatinine concentration or the occurrence of oliguria, or both.

RESULTS

The course of the acute renal failure in our 8 patients is described in terms of serial serum creatinine levels, urinary volumes, and response to diuretics. The severity and duration of renal function impairment, as measured by serial serum creatinine determinations, are shown in Figure 1. In all patients, serum creatinine levels increased to more than twice the base-line value, indicating a loss of more than 50 per cent of renal function and involvement of both kidneys. As early as the first day after angiography, a change in serum creatinine was noted in the 2 patients for whom this information was available. For the remaining patients who did not have serum creatinine determinations until the second or

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TABLE I

ANGIOGRAPHIC PROCEDURES, INDICATIONS, AND FINDINGS

Case No.	Age (yr.)	Procedure	Indication	Finding
1	60	Ventriculography+coronary arteriography	Mitral insufficiency	Mitral insufficiency, coronary artery disease
2	42	Visceral arteriography	Budd-Chiari syndrome	Hepatic vein thrombosis
3	66	Visceral arteriography	Hereditary telangiectasia	Angiomatosis of liver
4	59	Aortography	Renovascular hypertension	Bilateral renal artery stenosis
5	23	Ventriculography	Congenital heart disease	Complete atrioventricular canal
6	80	Aortography+left renal arteriography	Renal mass	Renal cysts, benign
7	49	Aortography	Occlusion of multiple arteries	Renal artery stenosis, occlusion of multiple arteries
8	49	Aortography+left renal arteriography	Renal mass	Adrenal tumor

third day, an average daily increase of more than 1 mg./dl. was observed. This increase is similar to that seen in acute renal failure from other causes.

The duration of oliguria, defined as urinary output of less than 20 ml./hr. or 500 ml./da., is depicted in Figure 2. Low urine volumes were first noted within 12 hours after angiography in 4 patients and before 24 hours in 2 other patients. In the remaining 2 patients, no adequate measurements of urine volume were made until 31 and 34 hours, respectively, after the angiographic procedure. Most likely, oliguria began in all patients shortly after the procedure but was not documented because urine volumes were not measured in every case during the first 24 hours. Thus, 7 of the 8 patients had either a significant change in serum creatinine level or documented oliguria within 24 hours after angiography.

The severe nature of the renal failure is indicated by the poor response to large doses of diuretics and a further increase in serum creatinine for 2 to 4 days after attempts at diuresis in 5 patients. However, recovery of renal function usually commenced within 1 week; this is earlier than expected for most patients with acute

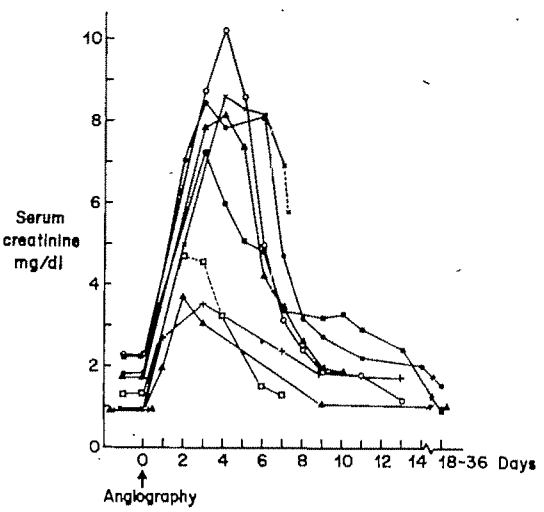


FIG. 1. Serum creatinine values before and after angiography.

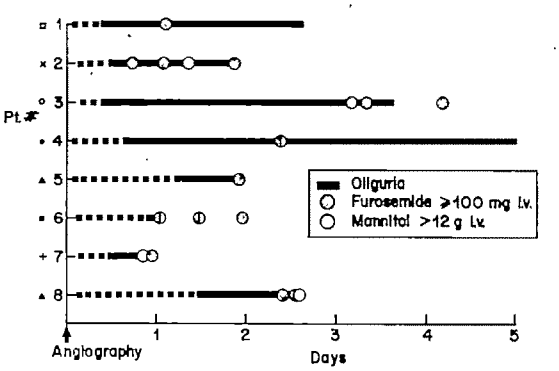


FIG. 2. Duration of oliguria. Symbols identify curves in Figure 1.

oliguric renal failure. The importance of early recognition and adequate treatment of this complication is exemplified by Case 1, in which severe fluid overload and hyperkalemia were of such magnitude that peritoneal dialysis was required as early as the second day. One death occurred (Case 2) during the recovery phase, despite hemodialysis performed for severe hepatic failure, and was probably unrelated to renal failure. In the 7 other patients, gross recovery of renal function to base-line levels occurred before hospital dismissal. The longest recovery time was approximately 16 days.

DISCUSSION

Currently used angiographic contrast media such as the diatrizoate and iothalamate salts have been considered to be safe by many investigators.[6,19,31] Although acute renal failure has not been reported in animal experiments with these media, several studies have suggested a transient and mild decrease in renal function.[27,34,36,38] Few clinical instances of severe impairment of renal function have been reported and most have occurred under specific circumstances. An overdose of contrast medium or a concurrent multiple myeloma disorder was the likely cause for several reported cases of renal damage after angiography in adults.[5,24,31,33] Two cases of angiographically induced renal failure in children also have been documented, in a patient with severe heart disease and in a newborn infant.[15,17]

The literature contains few reports of unexplainable acute renal failure after angiography.[24,35,37] Renovascular hypertension was described in 2 patients and diabetes mellitus in a third. In a fourth case, the patient had aortic insufficiency and later developed severe proteinuria; renal biopsy findings in this patient were thought to be compatible with hypersensitivity-induced glomerulonephritis.[4]

PATHOPHYSIOLOGIC MECHANISMS

There are many possible causes for acute renal failure after angiography. Because the time correlation between the angiographic procedure and the onset of renal failure was well established for our patients, certain explanations are possibilities, such as dosage and nephrotoxicity of contrast medium, cardiovascular effects after angiography, renal status prior to angiography, and allergic reactions.

Effect of contrast medium dosage and type. The type and amount of contrast medium used in our patients are shown in Table II. The dosages and the frequencies were similar to our total experience for all angiographic studies during this period at the Mayo Clinic. Recorded doses include all test injections and averaged 193 ml. or 3.4 ml./kg.; the concentrations were 70 to 80 per cent. In the 4 patients whose total dose was the highest, the amount was close to

TABLE II

TYPE AND AMOUNT OF CONTRAST MEDIUM USED

Case	Type	Amount Administered		
		ml. (70–80%)	ml./kg.	I, g./kg.
1	Renografin-76	285	4.6	1.70
2	Renografin-76	210	4.1	1.52
3	Renografin-76	185	4.2	1.34
4	Renografin-76	144	3.4	1.26
5	Renovist	230	4.9	1.82
6	Vascoray	110	1.4	0.56
7	Angio-Conray	70	1.3	0.62
8	Renografin-76	310	3.4	1.26

the proposed upper limit of 5 ml./kg. Some workers[36] consider this average dose to be too high. The 2 patients who had a relatively small amount of contrast medium had been exposed to contrast agents only 1 and 3 days previously. Two other patients had had a similar exposure within 1 week; the 4 others had had no studies with contrast medium for more than 1 month prior to angiography. Renal function impairment had not been detected after these preceding studies, and all patients had adequate urinary output prior to angiography.

The direct mechanism by which contrast medium might cause renal damage is still controversial.[16] Hypertonicity of the medium may cause crenation of erythrocytes with a secondary decrease in renal blood flow.[10] Vasoconstriction and subsequent vasodilatation immediately after exposure to contrast medium and release of histamine, particularly after a repeated study, have also been implicated.[2,9,20,21]

In our patients, a relatively high dosage or repeated administration of contrast medium may have been an etiologic factor. However, no patient in this series underwent bilateral selective renal angiography, a procedure that often produces maximal exposure of the kidneys to contrast medium. Usually, aortography utilizing 60 ml. of contrast medium and bilateral selective renal artery injections with 12 to 15 ml. per kidney is used for studies performed because of renovascular hypertension or a renal mass. Renal artery stenosis is not a contraindication for selective injection if occlusion of the renal orifice can be avoided. More than 2,700 such bilateral studies were performed at the Mayo Clinic during the period of review and no renal complications were observed in this group.

Cardiovascular effects. A prolonged decrease in blood pressure was not observed in any of our patients despite close monitoring. In only 1 instance was there a brief, moderate decrease in blood pressure, which responded immediately to a vasopressor agent (Vasoxyl).

Decreased peripheral and possibly renal perfusion after angiography may have occurred in several patients even though their blood pressure remained normal. In Case 1 there was a short episode of atrial fibrillation without associated signs of impaired peripheral perfusion. The congenital heart disease in Case 5 was associated with a chronically low cardiac output. Large arteriovenous fistulas in Case 3 produced a shunt of approximately 5 liters/min., and this may have impaired perfusion to other organs. A suspected pulmonary embolus after venography in Case 2 and a recurrent gastrointestinal hemorrhage in Case 3 also could have resulted in significantly decreased renal perfusion. However, blood pressures remained stable during these episodes.

Dehydration has been considered as an etiologic factor in angiographically induced complications.[12] The preparation, with fluid restriction and a laxative, may have induced a mild dehydration in our patients. However, none was prepared for simultaneous renal vein renin determinations—a procedure that often produces marked dehydration. In the same 4 year period, there were no recognized instances of acute renal failure in more than 500 patients who had combined renal vein renin and angiographic study in the salt-depleted and dehydrated state. Some of these salt-depleted patients, however, did receive 500 to 1,000 ml. of saline after the renal venous blood sampling. Dehydration *per se* apparently did not play a significant role in the development of acute renal failure in our patients. Other causes of decreased renal perfusion cannot be specifically excluded.

Renal disease. Renal abnormalities were present prior to arteriography in most of our patients and frequently were related to systemic disorders. Five of the 8 patients had an increased serum creatinine value, which clearly indicated a decreased glomerular filtration rate, before the angiographic study. The impairment of function was mild to moderate in all cases. Likely conditions responsible for these renal abnormalities included diabetic glomerulopathy, ur-

ate nephropathy, bilateral renal artery stenosis, and obstructive uropathy in Cases 1, 3, 4, and 6, respectively. Numerous patients with more marked impairment of renal function were subjected to arteriography during the same period without apparent complications. In our experience[14] with high-dose urography in patients with decreased renal function, there has not been a deleterious effect of the contrast medium. Diabetic patients may be an exception, however.

The combination of renal and hepatic impairment might increase the toxicity of the contrast medium, because of the limitations in both major pathways of excretion.[32] Although hyperbilirubinemia has been suggested[8] as a predisposing condition for the development of acute renal failure experimentally, clinical experience[7] does not support this. Of our 8 patients, 3 had marked abnormalities in liver function tests; 2 of these patients also had renal insufficiency. Perhaps there is a greater susceptibility to renal complications in patients with hepatic insufficiency who undergo arteriography.

Proteinuria may produce acute renal failure after angiography by precipitation of casts or proteinaceous material. Urinary Tamm-Horsfall protein[3] and the paraproteinuria of multiple myeloma,[28] particularly in the dehydrated patient, have been proposed as precipitating factors in the development of acute renal failure. Although present in the majority of our patients, proteinuria was only mild to moderate and was not associated with the nephrotic syndrome. Protein electrophoresis (obtained in 6 patients) did not suggest the presence of an abnormal serum protein and in no case was multiple myeloma suspected or subsequently diagnosed.

Diabetes mellitus was present in 1 patient, who also had probable diabetic nephropathy. We have observed the development of acute renal failure after excretory urography in 6 diabetic patients during the years 1968 through 1972. Pillay *et al.*[29] and Teal *et al.*[37] also suggested an association of renal failure with exposure to contrast medium in diabetic patients, but the mechanism for this has not been clarified.

According to Martin,[22] hyperuricemia can delay the excretion of contrast medium. Increased urinary uric acid excretion induced by these agents has been noted by Mudge[26] and Postlethwaite and Kelley[30] and is a potential hazard to renal function, particularly in dehydrated patients. Serum uric acid levels were abnormally high in 2 of our 4 patients in whom this value was available.

Hypersensitivity allergic reactions could cause acute renal failure as a consequence of induced vasculitis. An interstitial nephritis was implicated in 1 previously reported case.[4] The methylglucamine cation present in most media has been thought to release histamine[16] and thus cause bronchospasm.[1] In our patients, no overt allergic phenomena were observed, and histories of allergies were negative except for 1 patient who reported previous reactions.

PREVENTION

This review of potential causes for acute renal failure in our patients suggests that multiple factors, alone or in combination, are responsible for this nephrologic complication. The factors that identify the patient at risk are not clear, but all of our patients had severe medical disorders. The trend toward the use of angiography in more severely ill patients and the use of greater amounts of contrast medium should be tempered with an increased awareness of potential renal complications. More frequent consultation between the clinician and the radiologist, particularly when planning a study in severely ill patients, seems warranted. We suggest that consideration be given to the prophylactic administration of mannitol by intravenous infusion prior to and during the procedure and that fluid restriction be avoided whenever possible. Careful monitoring of urinary output and serum creatinine for at least 24 hours after angiography may allow the

early recognition of this complication so that appropriate therapeutic attempts to increase urine output, when impending oliguria is suspected, can be instituted earlier.

SUMMARY

Acute oliguric renal failure developed in 8 patients after catheterization angiography without bilateral renal arteriography.

The cause for this complication remains obscure. Severe dehydration, shock, overdose of contrast medium, or multiple myeloma could not be implicated. From retrospective analysis of these cases, multiple factors seem to be operative in these generally severely ill patients.

Early recognition and adequate management may prevent life-threatening effects of this infrequent, but potentially serious angiographic complication.

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REFERENCES

1. Ansell, G. Adverse reactions to contrast agents: scope of problem. *Invest. Radiol.*, 1970, *5*, 374–384.
2. Aperia, A., Broberger, O., and Ekengren, K. Renal hemodynamics during selective angiography. *Invest. Radiol.*, 1968, *3*, 389–396.
3. Berdon, W. E., Schwartz, R. H., Becker, J., and Baker, D. H. Tamm-Horsfall proteinuria: its relationship to prolonged nephrogram in infants and children and to renal failure following intravenous urography in adults with multiple myeloma. *Radiology*, 1969, *92*, 714–722.
4. Borra, S., Hawkins, D., Duguid, W., and Kaye, M. Acute renal failure and nephrotic syndrome after angiocardiography with meglumine diatrizoate. *New England J. Med.*, 1971, *284*, 592–593.
5. Bunnell, I. L. Selective Renal Arteriography: Its Application to the Diagnosis of Renal Vascular and Parenchymal Lesions. Charles C Thomas, Publisher, Springfield, Ill., 1968, p. 35.
6. Davidson, A. J., Abrams, H. L., and Stamey, T. A. Renal extraction of PAH in man following abdominal aortography. *Radiology*, 1965, *85*, 1043–1046.
7. Davidson, A. J., Becker, J., Rothfield, N., Unger, G., and Ploch, D. R. Evaluation of effect of high-dose urography on previously impaired renal and hepatic function in man. *Radiology*, 1970, *97*, 249–254.
8. Dawson, J. L. Jaundice and anoxic renal damage: protective effect of mannitol. *Brit. M. J.*, 1964, *1*, 810–811.
9. Dean, R. E., Andrew, J. H., and Read, R. C. Red cell factor in renal damage from angiographic media: perfusion studies of *in situ* canine kidney with cellular and acellular perfusates. *J.A.M.A.*, 1964, *187*, 27–31.
10. Derrick, J. R., Brown, R. W., Livanec, G., Bond, T. P., and Guest, M. M. Experimental effects of selective arteriography on microcirculation. *Am. J. Surg.*, 1968, *116*, 712–714.
11. Doust, B. D., and Redman, H. C. Myth of 1 ml/kg in angiography: study to determine relationship of contrast medium dosage to complications. *Radiology*, 1972, *104*, 557–560.
12. Dudzinski, P. J., Petrone, A. F., Persoff, M., and Callaghan, E. E. Acute renal failure following high dose excretory urography in dehydrated patients. *J. Urol.*, 1971, *106*, 619–621.
13. Edling, N. P. G., and Helander, C. G. On renal damage due to aortography and its prevention by renal tests: with reports of five cases of severe renal damage. *Acta radiol.*, 1957, *47*, 473–480.
14. Fulton, R. E., Witten, D. M., and Wagoner, R. D. Intravenous urography in renal insufficiency. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *106*, 623–634.
15. Gilbert, E. F., Khoury, G. H., Hogan, G. R., and Jones, B. Hemorrhagic renal necrosis in infancy: relationship to radiopaque compounds. *J. Pediat.*, 1970, *76*, 49–53.
16. Grainger, R. G. Renal toxicity of radiological contrast media. *Brit. M. Bull.*, 1972, *28*, 191–195.
17. Gruskin, A. B., Oetliker, O. H., Wolfish, N. M., Gootman, N. L., Bernstein, J., and Edelmann, C. M., Jr. Effects of angiography on renal function and histology in infants and piglets. *J. Pediat.*, 1970, *76*, 41–48.
18. Kincaid, O. W., and Davis, G. D. Renal Angiography. Year Book Medical Publishers, Inc., Chicago, 1966, pp. 37–38.
19. Knox, F. G., Bunnell, I. L., Elwood, C. M., and Stigman, E. M. Effect of selective renal angiography on glomerular filtration rate and renal plasma flow in man (abstract). *Physiologist*, 1965, *8*, 210.
20. Lasser, E. C., Walters, A., Reuter, S. R.,

and LANG, J. Histamine release by contrast media. *Radiology*, 1971, *100*, 683–686.
21. LINDGREN, P. Hemodynamic responses to contrast media. *Invest. Radiol.*, 1970, *5*, 424–435.
22. MARTIN, C. M. Myelography with sodium diatrizoate (Hypaque). *California Med.*, 1971, *115*, 57–59.
23. MCAFEE, J. G. Survey of complications of abdominal aortography. *Radiology*, 1957, *68*, 825–838.
24. MCEVOY, J., MCGEOWN, M. G., and KUMAR, R. Renal failure after radiological contrast media. *Brit. M. J.*, 1970, *4*, 717–718.
25. MILLER, G. M., WYLIE, E. J., and HINMAN, F., JR. Renal complications from aortography. *Surgery*, 1954, *35*, 885–896.
26. MUDGE, G. H. Uricosuric action of cholecystographic agents: possible factor in nephrotoxicity. *New England J. Med.*, 1971, *284*, 929–933.
27. MULLADY, T. F., WAKIM, K. G., HUNT, J. C., and KINCAID, O. W. Effects of diatrizoate sodium on kidney function in dogs. *J.A.M.A.*, 1963, *184*, 716–718.
28. PERILLIE, P. E., and CONN, H. O. Acute renal failure after intravenous pyelography in plasma cell myeloma. *J.A.M.A.*, 1958, *167*, 2186–2189.
29. PILLAY, V. K. G., ROBBINS, P. C., SCHWARTZ, F. D., and KARK, R. M. Acute renal failure following intravenous urography in patients with long-standing diabetes mellitus and azotemia. *Radiology*, 1970, *95*, 633–636.
30. POSTLETHWAITE, A. E., and KELLEY, W. N. Uricosuric effect of radiocontrast agents: study in man of four commonly used preparations. *Ann. Int. Med.*, 1971, *74*, 845–852.
31. ROBBINS, L. L. Golden's Diagnostic Radiology. Section 18. Selective Angiography. Williams & Wilkins Company, Baltimore, 1972, p. 9.
32. SAXTON, H. M. Urography. *Brit. J. Radiol.*, 1969, *42*, 321–346.
33. SIDD, J. J., and DECTER, A. Unilateral renal damage due to massive contrast dye injection with recovery. *J. Urol.*, 1967, *97*, 30–32.
34. SORBY, W. A., and HOY, R. J. Renal arteriography and renal function: short-term effect of aortography, selective renal arteriography, adrenalin suppression and renal venography on PAH extraction ratio. *Australasian Radiol.*, 1968, *12*, 252–255.
35. STARK, F. R., and COBURN, J. W. Renal failure following methylglucamine diatrizoate (Renografin) aortography: report of case with unilateral renal artery stenosis. *J. Urol.*, 1966, *96*, 848–851.
36. TALNER, L. B., and DAVIDSON, A. J. Effect of contrast media on renal retraction of PAH. *Invest. Radiol.*, 1968, *3*, 301–317.
37. TEAL, J. S., RUMBAUGH, C. L., SEGALL, H. D., BERGERON, R. T., and SCANLAN, R. L. Acute renal failure following spinal angiography with methylglucamine iothalamate: case report. *Radiology*, 1972, *104*, 561–562.
38. WOLLOWICK, H. E., FOSTER, J. H., SNYDER, H. E., YOUNGER, R., and KILLEN, D. A. Effect of abdominal aortography on proximal renal tubule function. *J. Surg. Res.*, 1966, *6*, 346–353.

VOL. 121, No. 3

ARTERIOGRAPHY AND PHLEBOGRAPHY OF THE LIMBS IN PLANNING SHUNT AND FISTULA OPERATIONS IN PATIENTS WITH CHRONIC RENAL FAILURE*

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REGULAR dialysis is well established in the treatment of chronic renal failure. The techniques used to create arteriovenous fistulae and Scribner shunts have been fully documented.[1,8,9,11]

The complications of these procedures include local sepsis, stenoses of the arteries and veins, thromboses, pulmonary and arterial emboli and septicemia, some of which may lead to failure of the shunts.[7,9,11]

For the patient on regular dialysis, shunt or fistula failure creates a crisis until a new site for vascular access is fashioned. It is helpful in managing these patients to know all possible future sites for shunts and fistulae.

For a successful shunt or fistula operation a patent artery and nearby superficial vein are required. An occluded vein may be reconstituted by grafting if necessary. For home management, in the majority of patients, a fistula in the left forearm is most convenient but failing this, any access site for dialysis in the arms or legs may have to be used.

While clinical examination and a knowledge of the usual anatomic pattern of arteries and veins is helpful in deciding where to place shunts or fistulae, the most accurate method of doing so is by arteriography and phlebography of both arms and legs. Only one report of such angiography has appeared in the literature.[13]

This paper describes the techniques used for 4 limb angiography and indicates its value in planning shunt and fistula operations.

METHODS OF INVESTIGATION

Three techniques have been employed:

1. Catheterization technique. A 100 cm., French 8, open-ended, radiopaque catheter with a curved tip is introduced into the subclavian arteries under screen control.[4,5] Hand injections of 30 ml. of meglumine iothalamate 60 per cent are made and 2 separate series of films, one of the arm and the other of the forearm, are exposed using an A.O.T. serial film changer. The arm is kept in the anatomic position to separate the images of the radius and ulna from the arteries and veins.

The catheter is then exchanged for a straight one with side holes and positioned just above the aortic bifurcation. Sixty ml. of the same contrast medium is injected at a rate of 12 ml./second and films exposed of both legs in the arterial and venous phases using a serial changer with programmed automatic moving table top.

2. Direct cannulation technique. Either the axillary or brachial arteries, and the femoral arteries are cannulated. Hand injections of 30 ml. of meglumine iothalamate 60 per cent are made for each of the 2 arm positions, and 50 ml. for each leg. Films to show the arteries and veins of the arms and legs are exposed as described above. The metal cannula with a short guidewire described by Sheldon[10] for carotid arteriography is easy to introduce, but any short cannula introduced over a needle is suitable.

3. Phlebography. This is carried out using our standard technique.[6] This consists essentially of injection of contrast material into a foot vein, films being exposed on a tilting screening table. A tourniquet is applied above the ankle to direct the contrast material into the deep venous system, but is not applied so tightly as to occlude the

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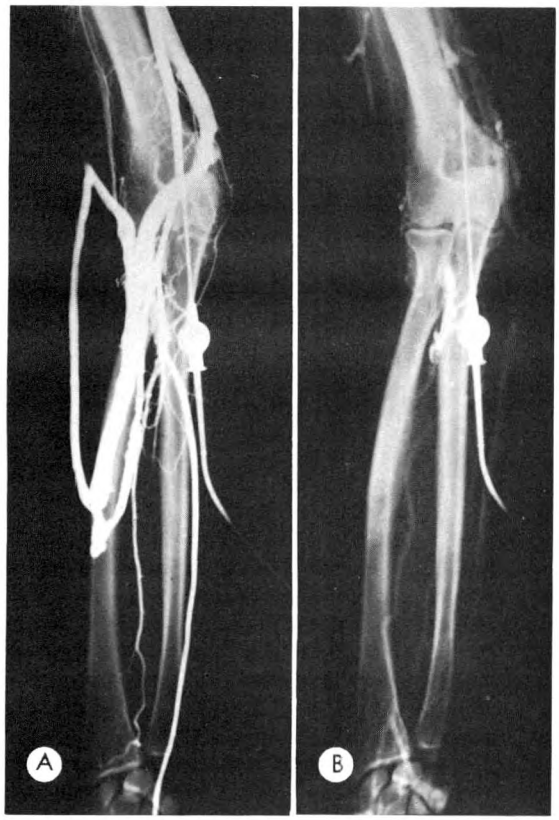


FIG. 1. Case 1. (*A* and *B*) Patent ulnar artery and basilic vein in right forearm. The fistula is demonstrated in *A*.

superficial veins. An exactly similar technique is used for the arms.

REPORT OF CASES

CASE 1. A 28 year old woman presented in renal failure due to chronic glomerulonephritis and hypertension. Many operations on 2 arm fistulae and 2 leg shunts were required to maintain hemodialysis. A renal transplant was rejected. A further right arm fistula was created (Fig. 1*A*). This fistula proved difficult for the patient to manage at home and a 4 limb angiography was carried out with a view to finding a more suitable site.

This angiography was carried out by bilateral femoral and brachial artery cannulations. Separate bilateral ascending phlebograms were obtained to show the leg veins.

The examination showed that there were patent ulnar arteries and basilic veins in both forearms (Fig. 1, *A* and *B*; and 2, *A* and *B*). The radial artery and cephalic vein were occluded in the left forearm (Fig. 2, *A* and *B*). The fistula was demonstrated in the right forearm (Fig. 1*A*).

In the right leg the peroneal artery was patent, the anterior and posterior tibial arteries were occluded (Fig. 3*A*). The long saphenous vein was occluded. A few small tributaries of the short saphenous vein were shown (Fig. 3*B*). In the left leg the peroneal artery and the long saphenous vein were patent (Fig. 4, *A* and *B*). The anterior tibial artery was occluded and the posterior tibial artery extremely small (Fig. 4*A*).

CASE II. A woman aged 44 years presented in chronic renal failure due to glomerulonephritis. Three shunts and 1 fistula operation were performed before renal transplantation. Rejection of the graft occurred after 15 months and this was followed by 5 further shunt operations.

A 4 limb angiography was carried out by bilateral femoral and brachial artery cannula-



FIG. 2. Case 1. (*A* and *B*) Patent ulnar artery and basilic vein in left forearm. Occlusion of the radial artery and cephalic vein.

tions. A separate bilateral phlebography was performed to show the leg veins. The examination showed that there were normal and patent ulnar arteries and basilic veins in both arms. Both radial arteries were occluded. In the right leg the shunt was demonstrated on the anterior tibial artery. The posterior tibial and peroneal arteries were normal. In the left leg the posterior tibial artery was occluded, the anterior tibial and peroneal arteries were patent. The long and short saphenous veins were normal on both sides.

A fistula was created in the right forearm using a homograft from the long saphenous vein. In the left thigh the proximal end of the saphenous vein was anastomosed to the femoral artery as an additional site for dialysis.

Case III. A woman aged 41 presented in chronic renal failure due to pyelonephritis. Two shunt and 2 fistula operations were required for regular dialysis.

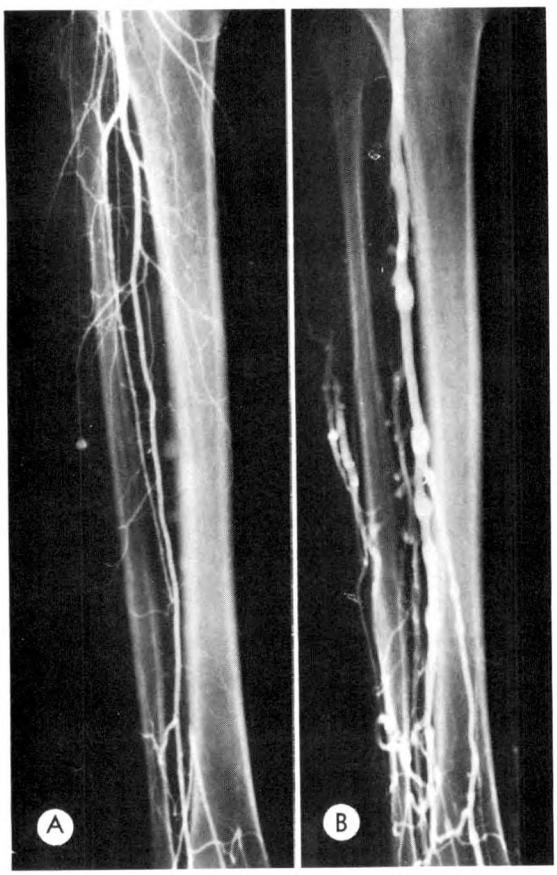


Fig. 3. Case 1. (*A*) In the right leg the peroneal artery is patent, the anterior and posterior tibial arteries are occluded. (*B*) The long saphenous vein is occluded and a few small tributaries of the short saphenous vein are shown.

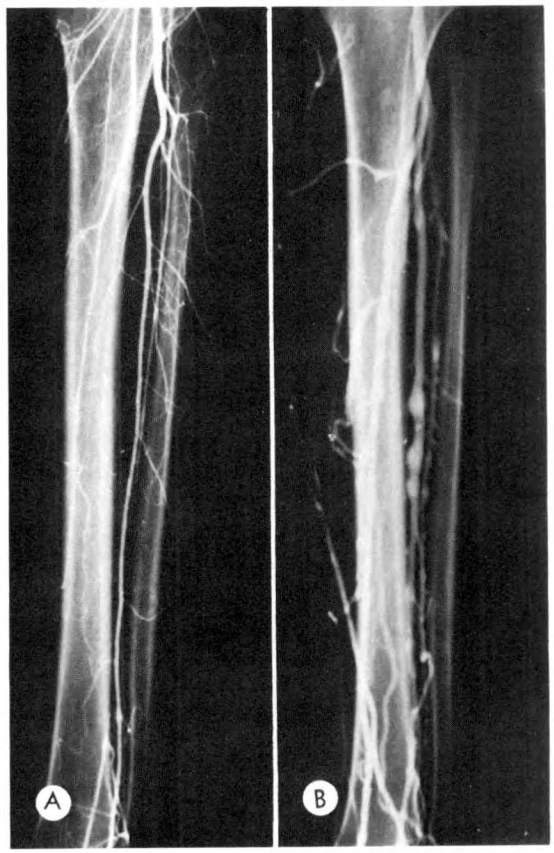


Fig. 4. Case 1. (*A* and *B*) In the left leg the peroneal artery and the long saphenous vein are patent. In *A* the anterior tibial artery is occluded and the posterior tibial artery is extremely small.

A 4 limb angiography was performed by bilateral selective catheterization of both subclavian and femoral arteries.

The examination showed normal radial and ulnar arteries and basilic vein in the left arm. In the right arm the radial artery was occluded. The veins were normal.

In the right leg the anterior and posterior tibial arteries and the peroneal arteries were normal. The long saphenous vein was also normal.

In the left leg the shunt was shown on the posterior tibial artery. The anterior tibial and peroneal arteries were normal. The long saphenous vein was patent, although stenosed at the shunt.

An A.V. shunt was created in the right leg and the fistula was refashioned in the left forearm.

CASE IV. A man aged 39 presented with renal failure due to polycystic disease. Four shunt operations on the left leg were required for dialysis. After a failed renal transplantation many more shunt and fistulae operations were required.

A 4 limb angiography was performed by bilateral brachial and femoral artery cannulations and a separate phlebogram was obtained to show the leg veins.

In the right forearm the ulnar artery was patent and the radial artery occluded. The basilic vein was patent and the cephalic vein occluded. In the left forearm the radial and ulnar arteries and the cephalic vein were occluded. The basilic vein was patent. In the right leg the anterior tibial, the peroneal arteries and the long saphenous vein were normal. The posterior tibial vein was occluded. In the left leg the anterior and posterior tibial arteries and long saphenous vein were normal. The shunt was demonstrated on the anterior tibial artery. There were no major venous tributaries on the lateral aspect of the calf.

CASE V. A woman aged 46 presented in chronic renal failure due to glomerulonephritis.

Seven consecutive shunt operations were required for regular dialysis treatment.

A 4 limb angiography was carried out by bilateral catheterization of the subclavian and femoral arteries.

The examination showed that in the right arm the ulnar artery and the basilic and cephalic veins were patent. The radial artery was occluded. In the left arm the main arteries and veins were normal. The main arteries and veins of the right leg were normal. In the left leg a functioning shunt was demonstrated. The main arteries and veins were normal.

A fistula was created in the right forearm.

CASE VI. A woman aged 34 years presented in chronic renal failure due to chronic glomerulonephritis. She had secondary hyperparathyroidism with vascular calcification.

Ten consecutive fistula and shunt operations were required for regular dialysis treatment.

A 4 limb angiography was performed by bilateral catheterization of the subclavian and femoral arteries.

The examination showed that in the left arm the brachial and ulnar arteries were patent, and the radial artery occluded. The veins were normal.

In the right forearm the radial artery was occluded. The veins were normal. In both legs the anterior tibial and peroneal arteries were normal. Both posterior and tibial arteries were occluded in the mid calf but reconstituted distally. The distal portions of the long and short saphenous veins were patent.

A fistula was created in the right forearm.

CASE VII. A woman aged 54 presented with chronic renal failure due to pyelonephritis. Two arm fistulae and 6 shunt operations were required for regular dialysis.

A 4 limb angiography was carried out by bilateral selective catheterization of the subclavian and femoral arteries.

In the left arm the ulnar artery, the basilic vein and medial vein of the forearm were patent. The radial artery and cephalic vein were occluded. In the right arm the ulnar artery, the basilic and cephalic veins were patent. The radial artery was occluded.

In the right leg the anterior tibial artery was normal. The posterior tibial and peroneal arteries were occluded. The veins were not demonstrated satisfactorily.

In the left leg the anterior tibial and peroneal arteries were normal. The posterior tibial artery and the long saphenous vein were the site of an A.V. shunt. A fistula was created in the left antecubital fossa.

CASE VIII. A man aged 44 presented with renal failure due to glomerulonephritis.

Two shunt operations and 2 forearm fistulae were required for regular dialysis before an unsuccessful renal transplantation.

After the transplant operation the fistulae in both arms thrombosed.

A 4 limb angiography was performed by bilateral selective catheterization of the subclavian and femoral arteries.

The examination showed that in the left forearm the ulnar artery and cephalic and basilic veins were normal. The fistula, which had apparently recanalized, was demonstrated connecting the radial artery and cephalic vein.

In the right arm the ulnar artery and the basilic and cephalic arteries were normal. The radial artery was occluded.

In the left leg the posterior tibial artery was very small following a previous shunt. The anterior tibial and peroneal arteries were normal. The veins were not demonstrated.

In the right leg the 3 main arteries were normal. The veins were not demonstrated.

Case ix. A 50 year old woman presented in chronic renal failure due to pyelonephritis. Many shunt operations in both legs had failed due to thromboses. A fistula in each arm functioned poorly.

A 4 limb angiography was carried out by axillary cannulation in the right arm, brachial cannulation in the left arm and bilateral femoral artery cannulations. Phlebography was performed to show the leg veins and the veins of the left arm.

The examination showed that the left ulnar artery and the basilic and cephalic veins were normal. The radial artery was occluded (Fig. 5, *A* and *B*). The brachial artery was shown by retrograde injection and appeared normal (Fig. 6*A*). A fistula was shown in the antecubital fossa (Fig. 6*B*). In the right arm the ulnar artery and the basilic vein were patent.

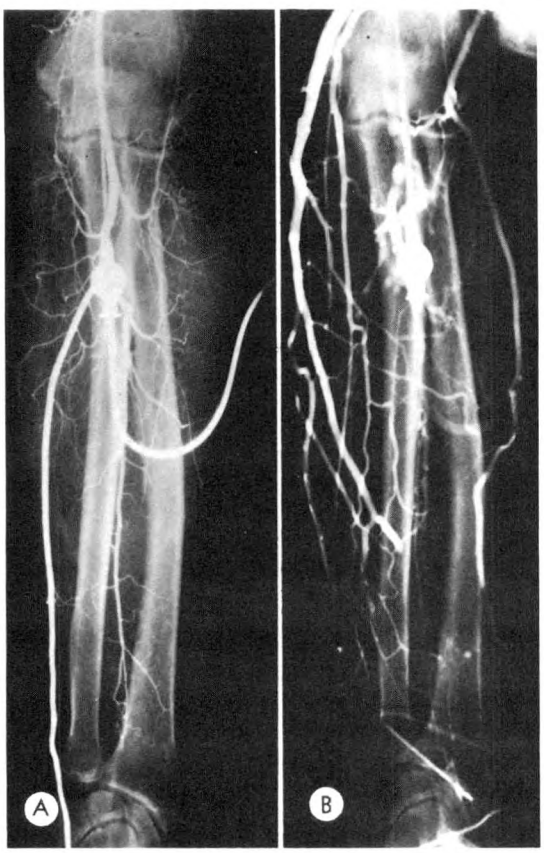


Fig. 5. Case ix. (*A* and *B*) Patent ulnar artery and basilic vein in the left forearm. Occlusion of the radial artery.



Fig. 6. Case ix. (*A* and *B*) Patent ulnar artery and basilic vein in the right forearm. Occlusion of the radial arteries.

In the right leg the main arteries and veins were normal (Fig. 7, *A* and *B*). In the left leg the anterior tibial and peroneal arteries were normal. The long saphenous vein was patent from the mid calf upwards. The saphenous vein in the thigh was occluded (Fig. 8).

Case x. A 38 year old woman presented with renal failure due to pyelonephritis. A shunt in each leg and a fistula in the right arm were required to maintain regular dialysis. The fistula, although patent, was not functioning well.

Angiography was performed by left brachial and bilateral femoral artery cannulations and separate phlebograms of the left arm and both legs were obtained to show the veins.

In the left arm, the radial and ulnar arteries were patent. The cephalic vein was occluded in the antecubital fossa but reconstituted in its upper third. A normal brachial artery was shown by retrograde injection. The arteries and veins of the right arm were not examined. In the right leg all the main arteries to the calf



FIG. 7. Case IX. (*A* and *B*) In the right leg the main arteries and veins are normal.

were occluded. The saphenous vein was occluded in the calf but patent in the thigh. In the left leg the peroneal artery was patent; the anterior and posterior arteries were occluded. The long saphenous vein was patent from the mid-calf upwards. The short saphenous vein was occluded. The long saphenous vein in the thigh was patent.

DISCUSSION

Our experience suggests that the quickest method for 4 limb angiography is by cannulation of the axillary arteries with follow-through to show the veins in the upper limbs, and bilateral femoral artery cannulation, with separate bilateral ascending phlebography to show the veins of the lower limbs. The hemodilution of the contrast medium is less in the arms than the legs and follow-through phlebography from the arteries produces an adequate demonstration of the arm veins, provided a sufficiently large volume of contrast material is used (Fig. 1*B*; and 2*B*). In the legs however, it is usually necessary to show the veins by phlebography (Fig. 3*B*; and 4*B*), as follow-through from arterial injections often shows the veins poorly (Fig. 8).

Using our technique for leg phlebography, which is the same as that used to show the veins in venous thrombosis, the tourniquet at the ankle is adjusted so that the superficial veins as well as the deep are demonstrated.[6] The same principle of not occluding the superficial veins is used should arm phlebography be required (Fig. 5*A*).

The amount of the contrast medium for a complete demonstration of the arteries and veins of all 4 limbs, including phlebograms of the legs, is at least 320 ml. We use

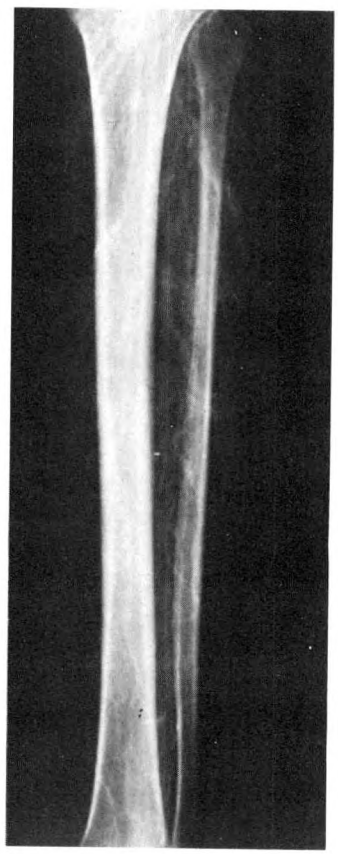


FIG. 8. Case IX. In the left leg the anterior tibial and peroneal arteries are normal. The long saphenous vein is patent from the mid calf upwards.

meglumine iothalamate 60 per cent, as a sodium containing contrast medium in this quantity could precipitate pulmonary edema in patients with incipient cardiac failure. Renal failure can be produced by contrast medium,[12] but in patients already committed to regular dialysis this consideration can be ignored and we believe that the relatively large volume of contrast medium used is safe.

The investigation is usually carried out under a light general anesthetic. This is not essential, but the examination tends to be prolonged and this group of patients who have always had a large number of minor surgical procedures tend to be intolerant of long investigations. The anesthetic introduces the risk of hyperkalemia and the plasma potassium is always estimated at the end of the procedure. To minimize this risk, the examination is carried out as rapidly as possible.

In all the patients reported every effort was being made to maintain long term dialysis to prepare them to treat themselves at home. Although some shunts last for several years, it is not unusual to encounter failure after only a few months. A well established fistula may last indefinitely if a patient's treatment is otherwise stable, but in some patients it is difficult to establish a fistula which is good enough for self-dialysis techniques. Special problems in vascular access surgery are encountered in patients with vascular calcification due to secondary hyperparathyroidism (Case VI) and with arteriosclerosis (Cases VII, and IX). Clotting of shunts is more likely to occur in patients who are the least anemic, as is often the case with polycystic kidneys (Case IV). However, in our experience, the greatest problems have occurred during the months following a failure of a transplant (Cases I, II, IV, and VIII). It is in these situations that 4 limb angiography has proved valuable in planning future sites for shunt and fistula operations.

The most convenient site for a shunt or fistula is in a calf or forearm but the upper arms and thighs may have to be used in some patients if no suitable artery and vein is available at these more accessible sites. For this reason the radiographic technique for 4 limb angiography should include demonstration of the arteries and veins of the upper arms and thighs.

Demonstration of the saphenous veins by phlebography is also important quite apart from the siting of shunts or fistulae, as these veins may need to be used as a homograft to bridge an occluded vein to create a fistula.

SUMMARY

The techniques used for 4 limb angiography in 10 patients with chronic renal failure on long term hemodialysis are described.

It is suggested that to show the arteries of the upper and lower limbs bilateral axillary and femoral artery cannulation should be used. In the arms follow-through to the venous phase is usually adequate to show the veins but in the legs, because of hemodilution, separate bilateral ascending phlebograms are required.

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REFERENCES

1. Brescia, M. J., Camino, J. E., Appel, K., and Herwich, B. J. Chronic hemodialysis using venepuncture and surgically created arteriovenous fistula. *New England J. Med.*, 1966, *275*, 1089–1092.
2. Cohen, B. H., Chinitz, J. L., Soll, K. H., Ramirez, O., Onesti, G., Kim, K. E., and Swartz, C. Radiological contrast study of arteriovenous dialysis cannulae. *Radiology*, 1970, *94*, 603–606.
3. De Palma, J. R., Golding, A., and Maxwell, M. H. Evaluation of A. V. cannula malformation and clotting using shunt angiography. *Trans. Am. Soc. Artif. Intern. Organs*, 1969, *15*, 278–284.
4. Hinck, V. C., and Dotter, C. T. Appraisal of current techniques for cerebral angiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 626–630.
5. Huick, V., Judkins, M., and Parton, H. Simplified selective femoro-cerebral angiogram. *Radiology*, 1969, *89*, 1048–1052.

6. LEA THOMAS, M. Phlebography. *A.M.A. Arch. Surg.*, 1972, *104*, 145–151.
7. LOWENSTEIN, E., LITTLE, J. W., III., and Lo, H. H. Prevention of cerebral embolization from flushing radial artery cannulas. *New England J. Med.*, 1971, *285*, 1414–1415.
8. QUINTON, W., DILLARD, D., and SCRIBNER, B. H. Cannulation of blood vessels for prolonged hemodialysis. *Trans. Am. Soc. Artif. Intern. Organs*, 1960, *6*, 104–109.
9. SCRIBNER, B. H., CANER, J. E. Z., BURI, R., and QUINTON, W. Technique of continuous hemodialysis. *Trans. Am. Soc. Artif. Intern. Organs*, 1960, *6*, 88–103.
10. SHELDON, P. Percutaneous cannulation of carotid artery. *Brit. J. Radiol.*, 1964, *37*, 526–530.
11. SWARTZ, C., and TEPLICK, J. G. Radiographic consideration in maintenance dialysis. *Radiol. Clin. North America*, 1972, *10*, 511–528.
12. TALNER, L. B. Urographic contrast media in uremia. *Radiol. Clin. North America*, 1972, *10*, 421–432.
13. WING, A. J., JONES, N. F., LEA THOMAS, M., and THOMPSON, A. E. Peripheral angiography of arms and legs as aid to planning shunt and fistula operations. *Proc. European Dialysis & Transplant A.*, 1971, *7*, 510–512.

CONGENITAL SUBCLAVIAN STEAL*

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KOOK SANG OH, M.D., *and* JOHN P. DORST, M.D.
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WHILE Contorni in 1960 was the first to demonstrate by angiography retrograde collateral flow in the vertebral artery of man, it was Reivich *et al.*,[12] in 1961, who showed the relationship of this phenomenon to symptoms of cerebral ischemia. They showed that a block of the proximal portion of a subclavian artery resulted in blood being stolen from the cerebral circulation to supply the affected upper limb. In such cases, some of the blood delivered to the brain through the internal carotid arteries and the contralateral vertebral artery flowed through the Circle of Willis and down the vertebral artery to the patent distal portion of the subclavian artery beyond the obstruction. This type of collateral circulation is termed a subclavian steal. When it results in symptoms of cerebral ischemia it is called the subclavian steal syndrome.

The subclavian steal syndrome occurs most often in adults who have atherosclerotic obstruction of the proximal subclavian artery. In children, subclavian steal is infrequent, usually asymptomatic, and is caused by a congenital anomaly of the thoracic aorta. We know of reports of 29 cases of congenital subclavian steal demonstrated by angiography.[9,11,17] Our experience with an additional 5 cases forms the basis of this communication.

CASE MATERIAL

Since 1963 congenital subclavian steal has been demonstrated in 7 patients by angiography at The Johns Hopkins Hospital. Three of these patients were reported previously.[1,11] One of these cases[11] and the 4 other cases are presented in this article.

REPORT OF CASES

CASE I. P.M. (JHH# 142 98 60) was a healthy 4 year old girl referred for evaluation because of a murmur noted on routine physical examination. Blood pressure was 110/94 mm. Hg in the right arm and 130/68 in the left arm. Neither pulse nor blood pressure was detected in the lower limbs. She had a Grade 3/6 continuous murmur best heard beneath the left scapula. It was also present in the left second intercostal space and radiated to the neck. The electrocardiogram (ECG) was normal. A cardiac series showed left ventricular enlargement and a posterior indentation in the barium filled esophagus that ran obliquely cephalad from left to right. At cardiac catheterization left ventricular pressure was 134/9 mm. Hg and the femoral artery pressure was significantly lower (106/74). Left ventricular angiography (Fig. 1, *A–C*) showed coarctation of the aorta and an aberrant right subclavian artery that arose distal to the coarctation. The aberrant right subclavian artery filled from the vertebral artery and other collateral vessels.

CASE II. B.A. (JHH# 90 76 44), an 18 year old girl, was first seen in 1954 following discovery of a murmur. Since then she has had no symptoms except occasional cold feet. Blood pressure was 150/110 mm. Hg in the left arm and 120/105 in the right arm. Femoral pulses were barely palpable and the blood pressure was unobtainable in the lower limbs. A thrill was palpable over the right carotid artery. The first heart sound was decreased and an early ejection sound was present. She had an apical Grade 2/4 blowing pansystolic murmur that radiated into the axilla. The chest roentgenogram showed a normal heart size with a right aortic arch and a ring like calcification in the most proximal portion of the descending thoracic aorta. Angiography showed a high right aortic arch with a long coarctation and a

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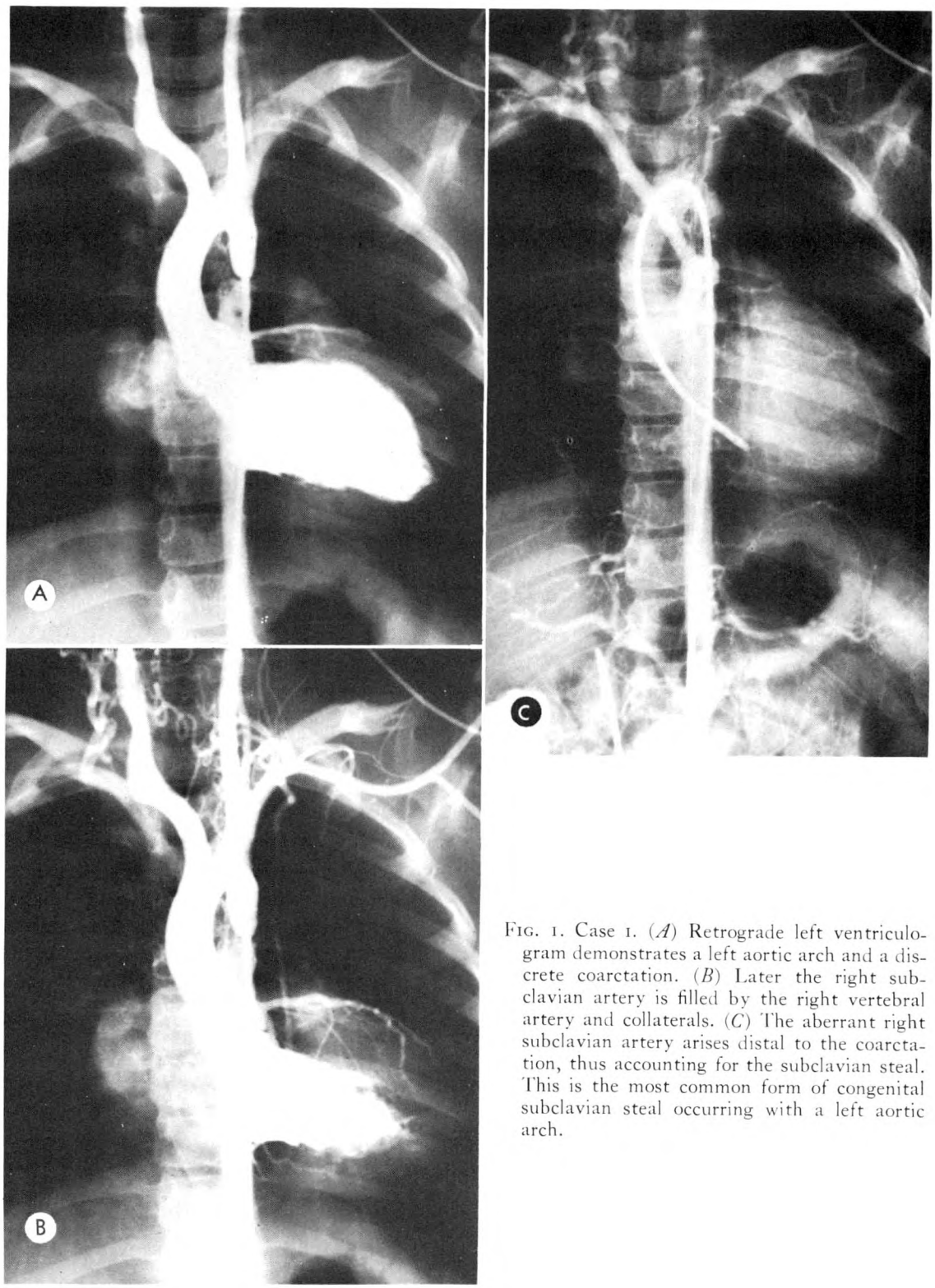


FIG. 1. Case 1. (*A*) Retrograde left ventriculogram demonstrates a left aortic arch and a discrete coarctation. (*B*) Later the right subclavian artery is filled by the right vertebral artery and collaterals. (*C*) The aberrant right subclavian artery arises distal to the coarctation, thus accounting for the subclavian steal. This is the most common form of congenital subclavian steal occurring with a left aortic arch.

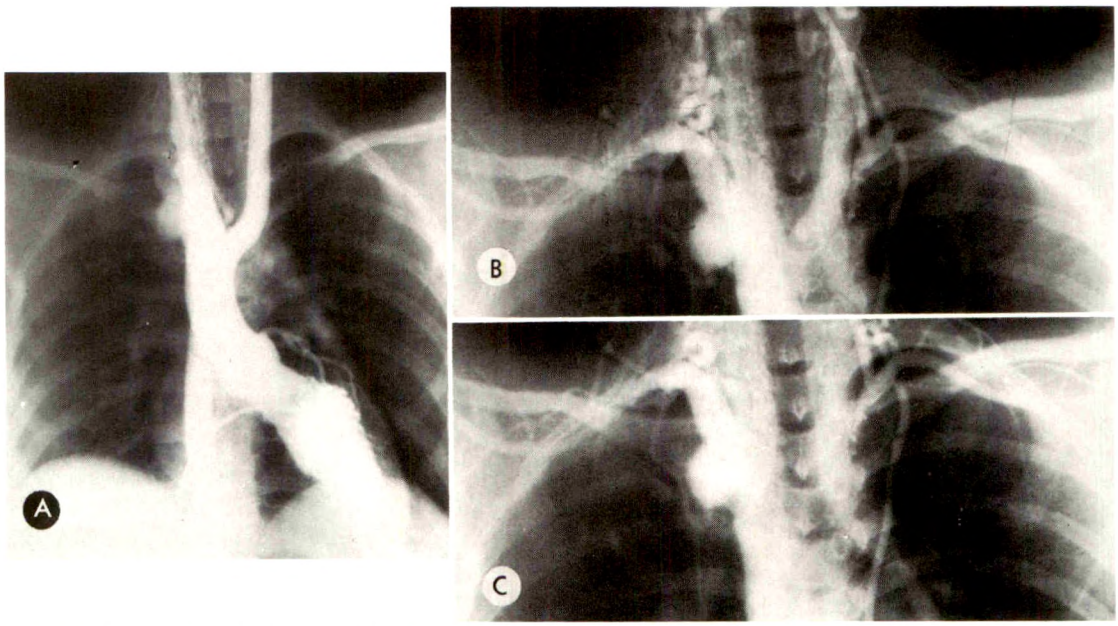


Fig. 2. Case II. (*A–C*) Transseptal left ventriculograms. A right aortic arch, coarctation, and a calcified aneurysm arising immediately distal to the coarctation are demonstrated. The left subclavian artery is isolated from the aortic arch and fills *via* a large left vertebral artery. The right subclavian artery arises at the level of the coarctation and also fills by the right vertebral artery and collaterals.

calcified aneurysm immediately distal to the coarctation (Fig. 2, *A–C*). The left subclavian artery did not fill directly from the aorta but instead filled from retrograde flow in the left vertebral artery and other collateral vessels. The right subclavian artery arose from the aortic aneurysm and was also filled in part by collateral vessels.

Case III. C.G. (JHH# 136 33 41), a 3,600 gm. boy, developed severe congestive heart failure at 15 days of age and was transferred to Johns Hopkins Hospital for evaluation. He was dusky with marked tachypnea. Respirations were 80 per minute and pulse 156. The blood pressure was 100/65 mm. Hg in the right arm and leg and 80/60 in the left arm. The left radial and brachial pulses were absent. He had a gallop rhythm and hepatomegaly caused by heart failure. The ECG showed biatrial enlargement, left ventricular hypertrophy and an indeterminate axis. A right aortic arch, cardiomegaly, increased pulmonary vascularity, and a small posterior esophageal indentation were noted on the roentgenograms. Cardiac angiography demonstrated a large ventricular septal defect with a left-to-right shunt. The left subclavian artery did not fill from the aortic arch but instead was opacified by retrograde flow in the left vertebral artery and other collaterals. A diverticulum was demonstrated in the distal aortic arch.

Case IV. D.B. (JHH# 106 06 33) was a normally developed 13 year old girl who had a murmur first noted at 3 years of age. On physical examination, the blood pressure was 150/0 mm. Hg in the right arm and 165/90/70 in the left arm. Femoral pulses were decreased and the right brachial pulse was present only when the head was turned to the right. She had a left ventricular heave, a thrill in the suprasternal notch and an aortic ejection click preceded by a harsh, Grade 2/4 aortic ejection murmur. The ECG showed left axis deviation and a right-sided conduction delay. The chest roentgenogram showed bilateral cervical ribs, normal heart size and a "figure of 3 sign" indicating coarctation of the aorta. At cardiac catheterization systolic pressures in the left ventricle and proximal aorta were 140 mm. Hg, while in the distal aorta, systolic pressure was 82 mm. Hg. Aortography demonstrated coarctation of the aorta with bicuspid aortic valve and an aberrant right subclavian artery that originated distal to the coarctation. The right subclavian artery filled by retrograde flow from numerous

collateral vessels, including the vertebral artery.

CASE V. R.K. (JHH# 12 00 93), a 53 year old male, suddenly lost the ability to speak following a coughing spell. His past medical history included longstanding congestive heart failure, hypertension and occasional headaches and dizziness. On physical examination, blood pressure in the right arm was 140/90 mm. Hg and in the left arm it was 120/90. The left carotid pulse was diminished. Left hemiparesis, left hemianesthesia, left homonymous hemianopsia, and a left central facial palsy were present. Lumbar puncture, echo encephalogram, brain scan, and ophthalmodynamometry were normal. He improved rapidly and a diagnosis of transient ischemia to the post central region of the right cerebral hemisphere was considered likely. A thoracic aortogram showed a right aortic arch with early bifurcation of the left common carotid artery. The left subclavian artery did not fill directly from the aortic arch but instead opacified from the left vertebral artery and collateral vessels from the left external carotid artery. A right carotid arteriogram showed very tortuous vessels without stenosis or recent occlusion.

DISCUSSION

Significant obstruction or absence of the proximal subclavian artery is associated with the development of collateral circulation to the ischemic upper limb. The most common pathways of collateral blood flow include: (1) from the external carotid artery to the superior thyroidal artery, then to the inferior thyroidal artery, the thyrocervical and costocervical arteries and finally the subclavian artery; (2) from the contralateral internal mammary artery to the ipsilateral internal mammary artery, thence to the subclavian artery; (3) from the aorta and posterior intercostal arteries to the superior intercostal artery, then to the costocervical artery and finally the subclavian artery; (4) from the external carotid artery to the occipital artery, then to the muscular branches of the vertebral artery and finally to the subclavian artery; and (5) from the contralateral vertebral artery to the basilar artery to the ipsilateral vertebral artery and finally the subclavian artery. This last anastomotic network, because of its size, is usually the major collateral pathway.

The degree of obstruction in the subclavian artery which is necessary for retrograde vertebral flow has been the subject of numerous investigations. Flow is reversed when the mean pressure in the basilar artery exceeds that in the vertebral-subclavian system. Reivich *et al.*[12] found that blood flow reversed in man with a drop in systemic artery pressure greater than 10 per cent across the stenotic segment of the subclavian artery. This corresponded to a 50 per cent reduction in the diameter of the lumen. Ekestrom and Retamal[4] noted subclavian steal with a mean pressure gradient of 30 mm. Hg across the stenosis while Sammartino and Toole[13] found that a range of 21–40 mm. Hg was necessary. The steal may be intermittent, fluctuating with changes in pressure and resistance. Because exercise of a limb increases flow and lowers vascular resistance, it probably increases the pressure gradient across the obstruction and facilitates retrograde flow. A compensatory increase in flow in the carotid arteries and contralateral vertebral artery has been observed in the experimental animal and man. It does not, however, compensate for flow lost *via* the ipsilateral vertebral artery and thus, there is a net decrease in cerebral flow.[6,7]

In the adult, intermittent symptoms of cerebral or limb ischemia or both frequently accompany subclavian steal, reflecting inadequate collateral supply to these regions. Exercise may precipitate both central nervous system and limb symptoms. Pain, easy fatigability, and numbness are most frequently noted in the arm, while dizziness, vertigo, headache, and visual symptoms are the most common central nervous system complaints.[8] The spectrum of reported symptoms, however, is great, particularly those related to brain stem ischemia. Since most cases of subclavian steal syndrome are due to atherosclerotic obstruction of the subclavian

artery, it is not surprising that coexistent occlusive disease is found in cerebral vessels in approximately 30 to 40 per cent of patients. In a collective review of the subclavian steal syndrome, only 10 per cent of adult patients were asymptomatic.[10] In infants and children, however, most patients are asymptomatic. Pieroni *et al.*[11] found only 5 of 26 children with congenital subclavian steal who had symptoms definitely ascribable to ischemia. These symptoms usually were parasthesia or exercise intolerance of the affected limb. Of our 5 cases, only Case V, a 53 year old man, had symptoms referable to the central nervous system. The degree to which his subclavian steal contributed to his transient ischemic event is open to speculation. However, 2 patients, 30 and 33 years old have been described with central nervous system symptoms attributable to congenital subclavian steal.[2] Although the natural history of congenital subclavian steal is not known, it appears likely that development of collateral channels does not always compensate fully for the anatomic abnormality.

Fifty-five potential causes for congenital subclavian steal have been described.[2] Many of these have not been observed in man. In the group studied by angiography, the following observations have been made: (1) congenital subclavian steal occurs approximately twice as frequently with a right than with a left aortic arch; (2) when a right aortic arch and subclavian steal coexist, the affected side is usually the left and is caused by hypoplasia, atresia or isolation of the left subclavian artery.[2] Three of our 5 patients had a right aortic arch; all had a left subclavian steal. Two of these patients had a barium esophagogram. Case II had no posterior esophageal indentation, indicating isolation of the left subclavian artery.[15] The posterior esophageal indentation in Case III could have been caused by an atretic band behind the esophagus that connected the aorta to the distal subclavian artery or, more rarely, an interrupted double aortic arch, Subtype III.[16]

Congenital subclavian steal in patients with left aortic arch is almost always associated with localized coarctation or complete interruption of the aortic arch. Eight of 9 patients reviewed by Pieroni *et al.*[11] had coarctation or aortic interruption. Our 2 patients had an aberrant right subclavian artery that arose distal to the coarctation. With a left aortic arch, the side of the subclavian steal is not predictable, since it depends on the relative positions of the subclavian artery and the site of the obstruction.

The incidence of congenital heart disease is increased in patients with congenital subclavian steal. An increased incidence of patent ductus arteriosus, ventricular septal defect or both are particularly common in patients with congenital subclavian steal and a left aortic arch. With a right aortic arch and mirror image branching of the aorta, cardiac malformations are much more common than with right aortic arch and aberrant right subclavian artery, regardless of the presence or absence of subclavian steal.

Cronquist[3] demonstrated retrograde opacification of the opposite vertebral artery in 20 per cent of normal patients with selective vertebral artery injections. Gonzalez *et al.*[5] showed retrograde vertebral artery flow when contrast material was injected in the contralateral brachial artery of a patient who subsequently had a normal aortic arch study; *i.e.*, false positives are possible with selective brachial or vertebral studies, and angiography of the aortic arch is essential for accurate diagnosis.

SUMMARY

Five cases of congenital subclavian steal demonstrated by angiography are reported.

The anatomy, hemodynamics, clinical findings and angiographic features are reviewed.

Ischemic symptoms are infrequently present in infants and children and most cases are discovered incidentally during evaluation of congenital heart disease and/or coarctation of the aorta. The relative lack of symptoms in infants and chil-

dren when compared to adults reflects in part more collateral development and the absence of associated occlusive disease of the cerebral vessels in children. While the natural history of this disorder is not known, there is some evidence that cerebral ischemic symptoms may arise as early as the fourth decade.

Subclavian steal associated with a right aortic arch usually occurs on the left side and is caused by hypoplasia, atresia or isolation of the proximal portion of the left subclavian artery. Subclavian steal associated with a left aortic arch is usually caused by coarctation of interruption of the aortic arch with the affected subclavian artery arising distal to the obstruction.

Arch aortography is the best method to evaluate possible congenital subclavian steal.

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REFERENCES

1. ANTIA, A. U., and OTTESEN, O. E. Collateral circulation in subclavian stenosis or atresia. *Am. J. Cardiol.*, 1966, *18*, 599–604.
2. BECKER, A. E., BECKER, M. J., and EDWARDS, J. E. Congenital anatomic potentials for subclavian steal. *Chest*, 1971, *60*, 4–13.
3. CRONQUIST, S. Vertebral catheterization via femoral artery. *Acta radiol.*, 1961, *55*, 113–118.
4. EKESTROM, S., and RETAMAL, E. Haemodynamic condition in subclavian steal syndrome. *Scandinav. J. Thor. & Cardiol. Surg.*, 1967, *1*, 161–164.
5. GONZALEZ, L. L., WIOT, J. F., and BOYD, A. D. Retrograde flow in vertebral artery. *A.M.A. Arch. Surg.*, 1965, *91*, 185–194.
6. HANDA, J., ISHIKAWA, S., HUBER, P., and MEYER, J. S. Experimental production of "subclavian steal" electromagnetic flow measurements in monkey. *Surgery*, 1965, *58*, 703–712.
7. HARDESTY, W. H., WHITACRE, W. B., TOOLE, J. F., RANDALL, P., and ROYSTER, H. P. Studies on vertebral blood flow in man. *Surg., Gynec. & Obst.*, 1963, *116*, 662–664.
8. HEATH, R. D. Subclavian steal syndrome. *J. Bone & Joint Surg.*, 1972, *54-A*, 1033–1039.
9. KINKHABWALA, M. N., and RABINOWITZ, J. G. Subclavian steal syndrome: three unusual cases in pediatric age group. *J. Canad. A. Radiologists*, 1972, *23*, 259–263.
10. PICCONE, V. A., JR., and LEVEEN, H. H. Subclavian steal syndrome. *Ann. Thoracic Surg.*, 1970, *1*, 51–75.
11. PIERONI, D. R., BRODSKY, S. T., and ROWE, R. D. Congenital subclavian steal. *Am. Heart J.*, 1972, *84*, 801–807.
12. REIVICH, M., HOLLING, H. E., ROBERTS, B., and TOOLE, J. F. Reversal of blood flow through vertebral artery and its effect on cerebral circulation. *New England J. Med.*, 1961, *265*, 878–885.
13. SAMMARTINO, W. F., and TOOLE, J. F. Reversed vertebral artery flow. *A.M.A. Arch. Neurol.*, 1964, *10*, 590–594.
14. SHUFORD, W. H., SYBERS, R. G., and EDWARDS, F. K. Three types of right aortic arch. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 67–74.
15. SHUFORD, W. H., SYBERS, R. G., and SCHLANT, R. C. Right aortic arch with isolation of left subclavian artery. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 75–83.
16. SHUFORD, W. H., SYBERS, R. G., and WEENS, H. S. Angiographic feature of double aortic arch. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *116*, 125–140.
17. SUNDERLAND, C. O., LEES, M. H., BONCHER, L. I., KIDD, H. J., and ROSENBERG, J. A. Congenital pulmonary artery—subclavian steal. *J. Pediat.*, 1972, *81*, 927–931.
18. VICTORICA, B. E., MIEROP, L. H. S., and ELLIOTT, L. P. Right aortic arch associated with contralateral congenital subclavian steal syndrome. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *108*, 582–590.

Vol. 121, No. 3

ANGULATED VIEWS IN THE SAGITTAL PLANE FOR IMPROVED ACCURACY OF CINECORONARY ANGIOGRAPHY*

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IN recent years, the diagnostic contribution of selective coronary arteriography has been considerable and its importance has been fully acknowledged. However, as stressed in previous reports, the method cannot invariably be considered as foolproof.[6,8,9,12,16] In our experience, discrepancies between the angiographic data and subsequent pathologic findings are particularly frequent, when lesions are located near the bifurcation of the left coronary artery. In this critical area, superimpositions of vessels not infrequently occur in all standard transverse angiographic projections and stenoses can be overlooked or underestimated. The purpose of this communication is to stress the usefulness of special cranial and caudal projections in a sagittal plane. These views clearly separate the proximal divisional branches of the left coronary artery and greatly improve visualization of stenoses in this area.

STANDARD CINECORONARY ANGIOGRAPHIC VIEWS

The equipment used in our laboratory is a Picker Saturn apparatus with a C-Arm.* This equipment allows a full 180° transverse or horizontal rotation of the x-ray tube and image intensifier about the thorax of the patient. The patient remains still on the table, lying flat on his back with both arms folded behind his neck. Using a percutaneous femoral approach and preformed polyethylene catheters,†[3] the usual transverse or horizontal projections of both coronary arteries can be easily and rapidly obtained. These projections include a frontal, true lateral, 30° right anterior oblique (RAO) and 45° to 60° left anterior oblique (LAO) view (Fig. 1*A*). In this standard transverse or horizontal plane of rotation, the x-ray beam is perpendicular to the longitudinal axis of the middle and distal segments of the left coronary artery (Fig. 1*B*; and 2). Pluridimensional views of the mid and distal segments of the left anterior descending (LAD) and left circumflex (LCX) coronary arteries can thus be obtained and provide for adequate visualization of any narrowing that may be present.

However, the proximal portions of the left anterior descending, first diagonal, left circumflex and left marginal arteries frequently overlap one another in these standard views, especially in the frontal, right anterior oblique and left anterior oblique projections (Fig. 3, *A–C*). Moreover, in their initial segments, these vessels are oriented in a similar transverse axis and tend to parallel the path of the x-ray beam directed posteroanteriorly (Fig. 2). Therefore, these vessels may be visualized tangentially or as a cut section of their proximal portion. Angiographic interpretation of lesions in these segments may be very difficult (Fig. 3*D*).

USEFULNESS OF SAGITTAL ANGULATIONS

Angulation of the x-ray beam caudally or cranially (Fig. 4*A*) often overcomes these difficulties. The plane of rotation is then

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† Bourassa Catheters, United States Catheter and Instrument Corporation, Billerica, Mass.

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Supported in part by the Jean-Louis Levesque Foundation.
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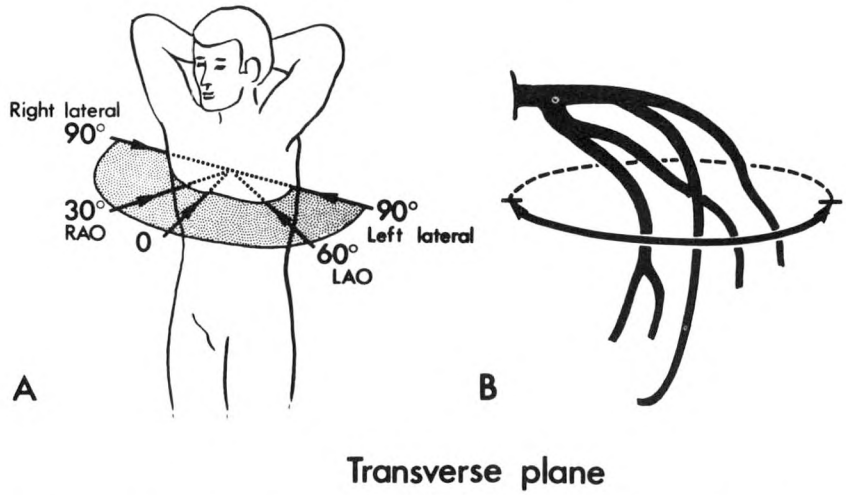


FIG. 1. Diagrammatic representation of the routine transverse or horizontal plane of rotation during selective cinecoronary arteriography. Diagram *A* illustrates the 180° rotation of the image intensifier and x-ray tube about the transverse or horizontal plane of the thoracic cage. This rotation allows for the projections commonly obtained in coronary arteriography (frontal, 30° RAO, 60° LAO and 90° right and left lateral). Diagram *B* shows that, in this plane of rotation, the x-ray beam is perpendicular to the longitudinal axis of the middle and distal segments of the left coronary artery, providing a good pluridimensional view of these segments.

more perpendicular to the proximal segments of the left coronary artery (Fig. 4*B*). Therefore, the frequently overlapped coronary artery branches are easily separated from each other. Secondly, these proximal segments are visualized from an entirely different angle, allowing a better definition of eccentric or slit-like stenoses. Thirdly, these segments are now projected lengthwise on the film, avoiding the tangential or cross-sectional view obtained in the transverse plane.

DESCRIPTION OF THE TECHNIQUE

Several angiographic installations currently used for coronary arteriographic studies including the Picker Saturn-C-Arm,* C.G.R. Arcus,† and G.E. Fluoricon-C,‡ offer the possibility of additional angulations of the x-ray tube and image intensifier in a sagittal plane (Fig. 5). A recent modification of this system, the U-Arm suspension (Philips Cardiodiagnost,§ Sie-

* Picker X-Ray Corporation, White Plains, N. Y.
† Compagnie Générale de Radiologie, Paris, France.
‡ General Electric Company, Milwaukee, Wisc.
§ Philips Electronics Ltd., Eidhoven, Netherlands.

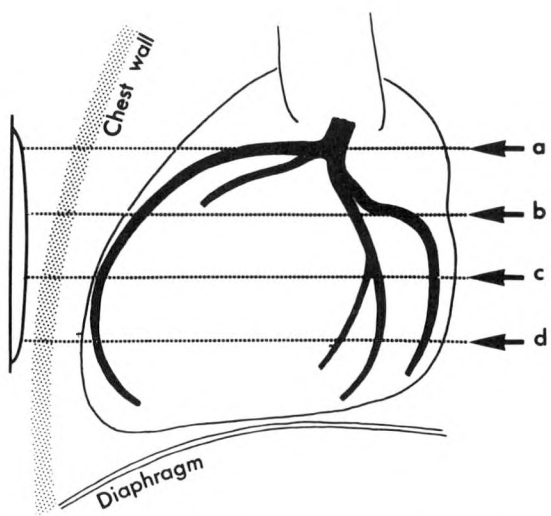


FIG. 2. Direction of the x-ray beam in the standard transverse plane. The incident x rays are almost perpendicular to the middle and distal segments of the LAD and LCX arteries, providing a good visualization of these segments (*c* and *d*). In contrast, the posteroanterior x-ray beam is often parallel to the proximal segments of the LAD and LCX arteries (*a* and *b*). In this situation, the vessels are seen tangentially or as a cut section of their proximal segment. Angiographic interpretation of lesions in these segments may be very difficult.

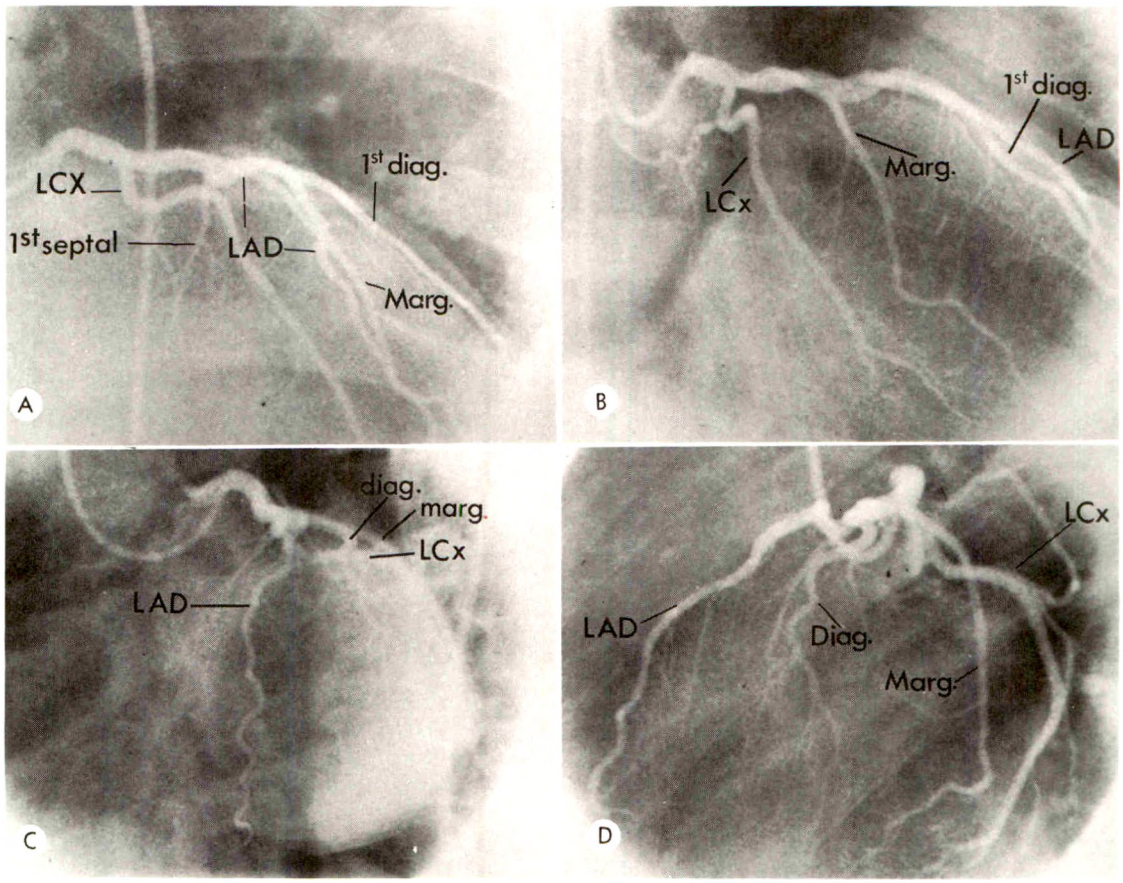


Fig. 3. (*A*) Routine frontal and (*B*) 30° RAO projections of the left coronary artery. Both projections show marked overlapping of the proximal segments of the anterior descending (LAD), first diagonal (1st diag.), and left marginal (Marg.) arteries. (*C*) In the 60° LAO projection, the proximal LAD, first diagonal (diag.) and left marginal (marg.) arteries are seen tangentially and again overlap each other. (*D*) The 90° lateral projection shows excellent visualization of the middle and distal segments of LAD and LCx arteries. However, in this projection, the initial portion of the first diagonal (Diag.) and left marginal (Marg.) branches are seen tangentially and, in addition, are superimposed on the proximal LAD and LCx arteries

mens Cardoskop U,‖ and G.E. Fluoricon-U) also permit the use of sagittal angulations. This can be done by rotating the catheterization table to a right angle with the U-Arm apparatus. This maneuver is, however, somewhat cumbersome and requires ample free space around the angiographic table in the catheterization laboratory.

Since the major purpose of these special angulations is to better visualize a limited portion of the coronary vascular system, the shutters of the x-ray tube can be moved closer to each other and the angiographic field reduced to a small rectangle (Fig. 6*B*; 7*C*; 8*D*; 9*B*; 10*C*; and 11*B*). This decreases secondary radiations and increases the contrast and detail of the film.

‖ Siemens Aktiengesellschaft, Erlangen, Germany.

A. CRANIO-CAUDAL SAGITTAL ANGULATION

In this first special view, the x-ray tube is angulated by approximately 25° towards the head of the patient. The image intensifier is thus inclined in a caudal direction (Fig. 6*A*). This sagittal angulation is usually obtained in a straight posteroanterior or frontal plane. However, a slight 10° RAO plane may also be used, whenever it is required to clear the thoracic spine or

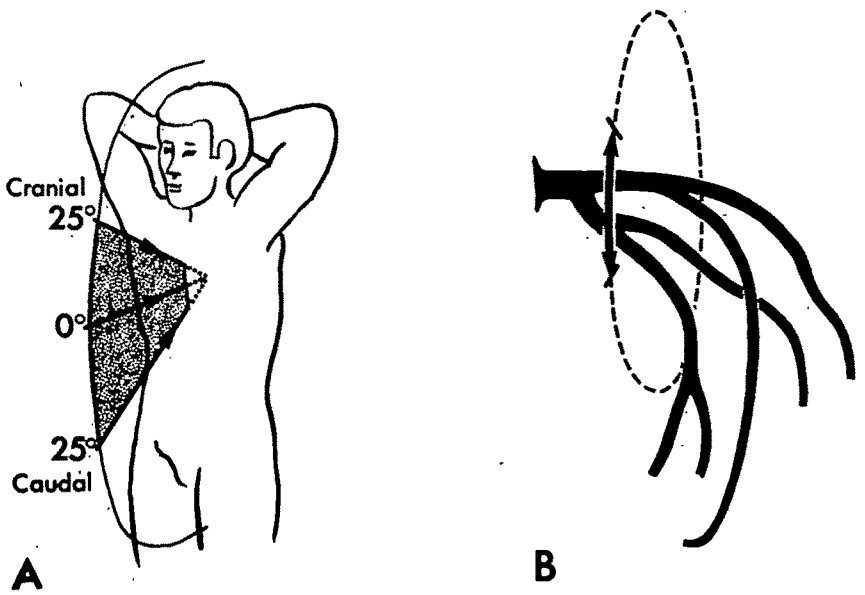


FIG. 4. Diagrammatic representation of special cranial and caudal angulations in the sagittal plane of the patient's chest. Diagram *A* illustrates the slight superior and inferior rotation of the image intensifier and x-ray tube along the longitudinal axis of the patient's chest. Diagram *B* shows that the plane of rotation is almost perpendicular to the transversely oriented proximal segments of the LAD and LCX arteries.

the ascending branch of the opaque catheter. The resultant incident cranio-caudal x-ray beam displaces the left anterior descending and diagonal arteries towards the top, and the left circumflex artery and its marginal branch towards the bottom of the film (Fig. 6*B*). In this view, the proximal portion of the left circumflex artery is almost perpendicular to the incident x-ray beam (Fig. 6*A*). This angulation clearly separates the origin and proximal segments of the left circumflex and marginal arteries from the left anterior descending artery (Fig. 7). Short diaphragmatic stenoses on the proximal portion of the left circumflex artery can be well visualized (Fig. 8).

B. CAUDO-CRANIAL SAGITTAL ANGULATION

In this second special view, the x-ray tube is angulated towards the feet of the patient. The image intensifier is thus inclined in a cranial direction and this results in an incident caudo-cranial beam (Fig. 9*A*). In this projection, the proximal left

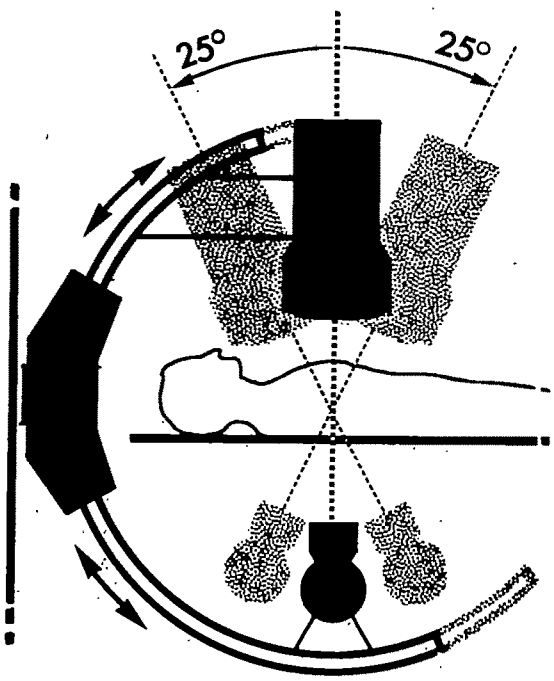


FIG. 5. Cranial and caudal sagittal angulation of the x-ray tube and image intensifier obtained with the C-Arm device.

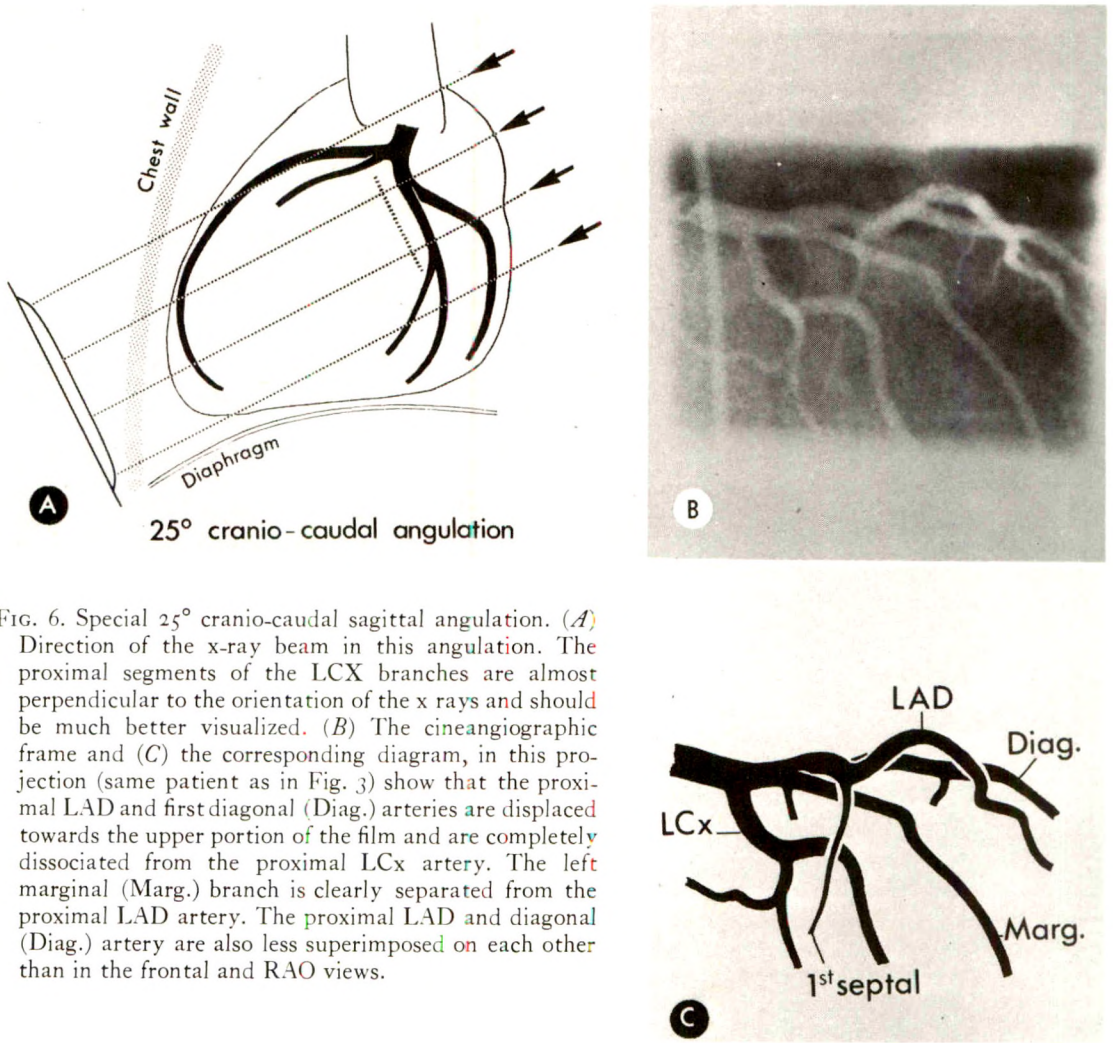


Fig. 6. Special 25° cranio-caudal sagittal angulation. (*A*) Direction of the x-ray beam in this angulation. The proximal segments of the LCX branches are almost perpendicular to the orientation of the x rays and should be much better visualized. (*B*) The cineangiographic frame and (*C*) the corresponding diagram, in this projection (same patient as in Fig. 3) show that the proximal LAD and first diagonal (Diag.) arteries are displaced towards the upper portion of the film and are completely dissociated from the proximal LCx artery. The left marginal (Marg.) branch is clearly separated from the proximal LAD artery. The proximal LAD and diagonal (Diag.) artery are also less superimposed on each other than in the frontal and RAO views.

anterior descending and diagonal arteries are seen more perpendicularly along their longitudinal axis and this view clearly demonstrates stenoses on the proximal segment of these vessels (Fig. 9*B*; and 10).

The caudo-cranial sagittal angulation may be adequately performed in the frontal plane (Fig. 9*B*; and 10*C*). However, in this plane, overlapping of segments of the anterior descending artery with branches of the circumflex artery is not infrequent (Fig. 9*B*). This difficulty is easily overcome by the use of a slight 15° LAO rotation, which permits separation of the left anterior descending from the left circumflex coronary artery branches (Fig. 11, *A* and *B*).

DISCUSSION

The occurrence of angina pectoris with normal coronary arteries has been recognized for several decades. Relative or functional myocardial ischemia can be induced by severe anemia, aortic valvular disease and systemic hypertension.[7] More recently, extensive use of selective coronary arteriography has led to the identification of patients or groups of patients with angina pectoris or myocardial infarction and normal coronary arteriograms. These paradoxical findings have been related to several mechanisms including abnormal oxygen-hemoglobin dissociation,[5] increased vasomotor activity,[14] primary abnormalities of myocardial metabolism[2,13] and, less likely,

FIG. 8. Cineangiographic views illustrate the importance of the cranio-caudal angulation to visualize stenosis of the left marginal artery. (*A*) The frontal and (*B*) 30° RAO projections show only minor irregularities (→) of the proximal segments of the LAD and LCx arteries. (*C*) On the lateral view, however, a stenosis can be suspected on the proximal segment of the left marginal (marg.) artery (→). The degree of this stenosis is very difficult to evaluate because of overlapping of the marginal (marg.) branch with the distal LCx artery in this projection. Note also that this segment of the marginal artery is seen tangentially in all standard transverse projections. (*D*) The sagittal cranio-caudal angulation permits a more perpendicular view of this left marginal artery (marg.) and excellent visualization of a severe localized proximal stenosis (→).

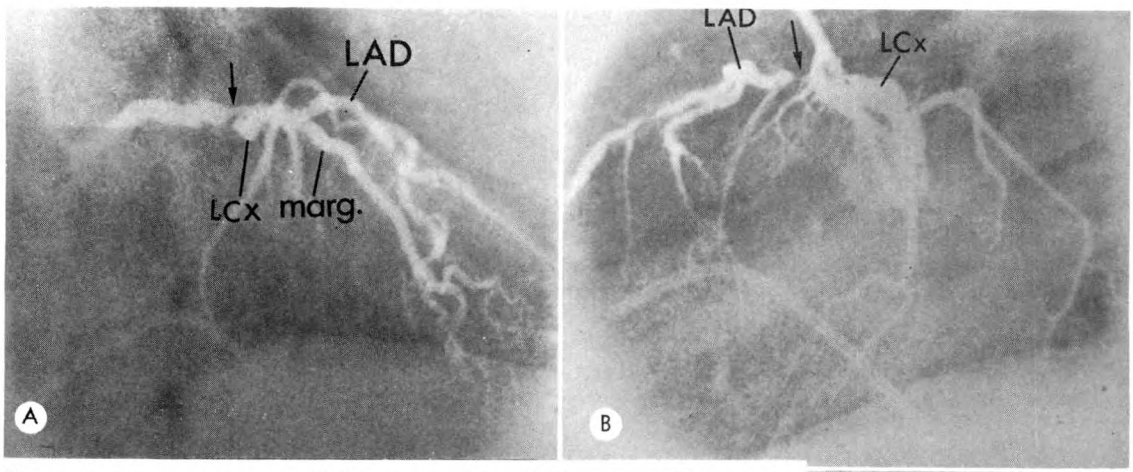
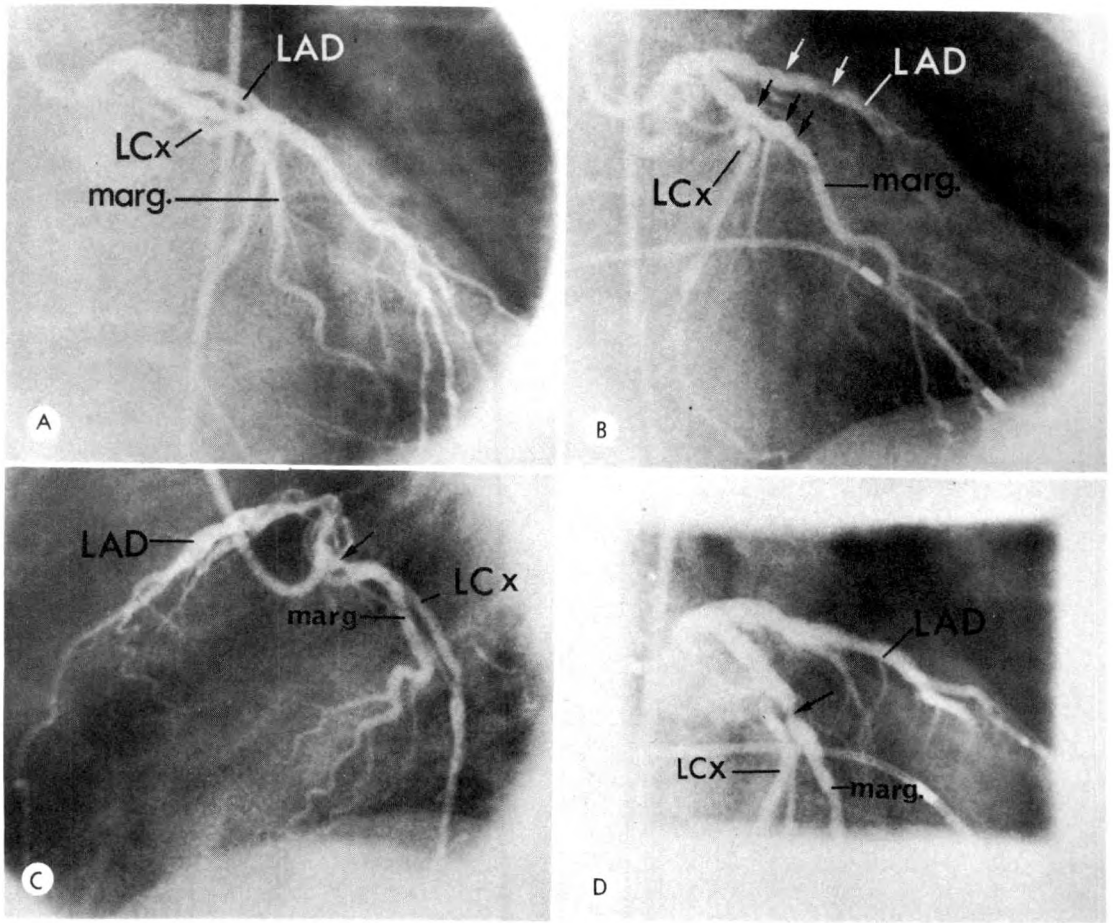
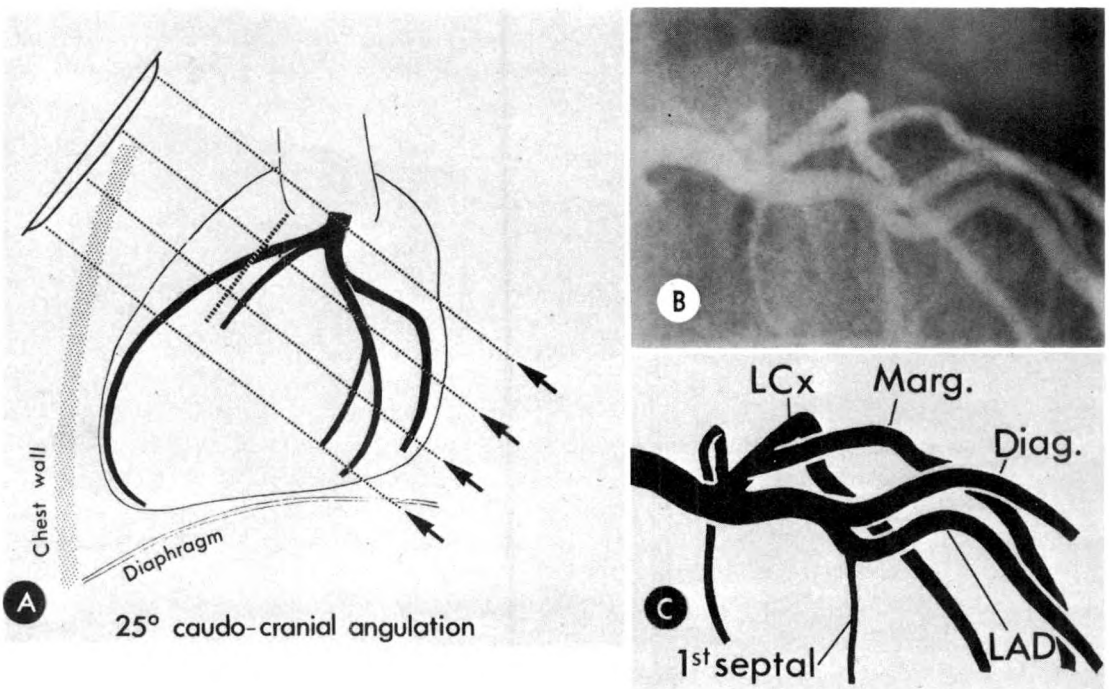


FIG. 7. Accurate assessment of the extent of proximal stenoses of the left coronary artery using a cranio-caudal angulation. The frontal and 60° LAO projections did not supply additional information and are not shown. (*A*) The 30° RAO projection shows a severe (75 per cent) stenosis of the main left coronary artery (→). The LCx and marginal (marg.) branches are superimposed on the proximal LAD artery. (*B*) The lateral view demonstrates a 90 per cent stenosis (→) of the proximal LAD artery, but there is again poor visualization of the proximal LCx artery. (*C*) The 20° cranial angulation clearly separates the proximal left coronary artery branches and demonstrates that the stenosis of the main left coronary artery (→) extends not only into the proximal segment of the LAD (↔), but also into the origin of the LCx artery (↠). This projection also separates the origin of the left marginal (marg.) branch from the distal LCx artery

FIG. 9. Special 25° caudo-cranial sagittal angulation. (*A*) Direction of the x-ray beam in this angulation. The proximal segment of the LAD and diagonal arteries are almost perpendicular to the orientation of the x-ray beam. (*B*) Cineangiographic frame and (*C*) corresponding diagram in this projection (same patient as in Fig. 3). The proximal LAD artery is displaced towards the center of the film. The LAD and diagonal artery are also separated from each other and are viewed lengthwise, along their longitudinal axis. Note, however, that in this caudo-cranial angulation, the proximal segment of the LCx artery is again seen tangentially. Thus, both cranial and caudal angulations may be required for correct assessment of all proximal segments of the left coronary artery.


LAD
LCx
marg.
A
LAD
LCx
marg.
B
LAD
LCx
marg
C
LAD
LCx
marg.
D


Chest wall
Diaphragm
A
25° caudo-cranial angulation
B
LCx
Marg.
Diag.
C
1st septal
LAD

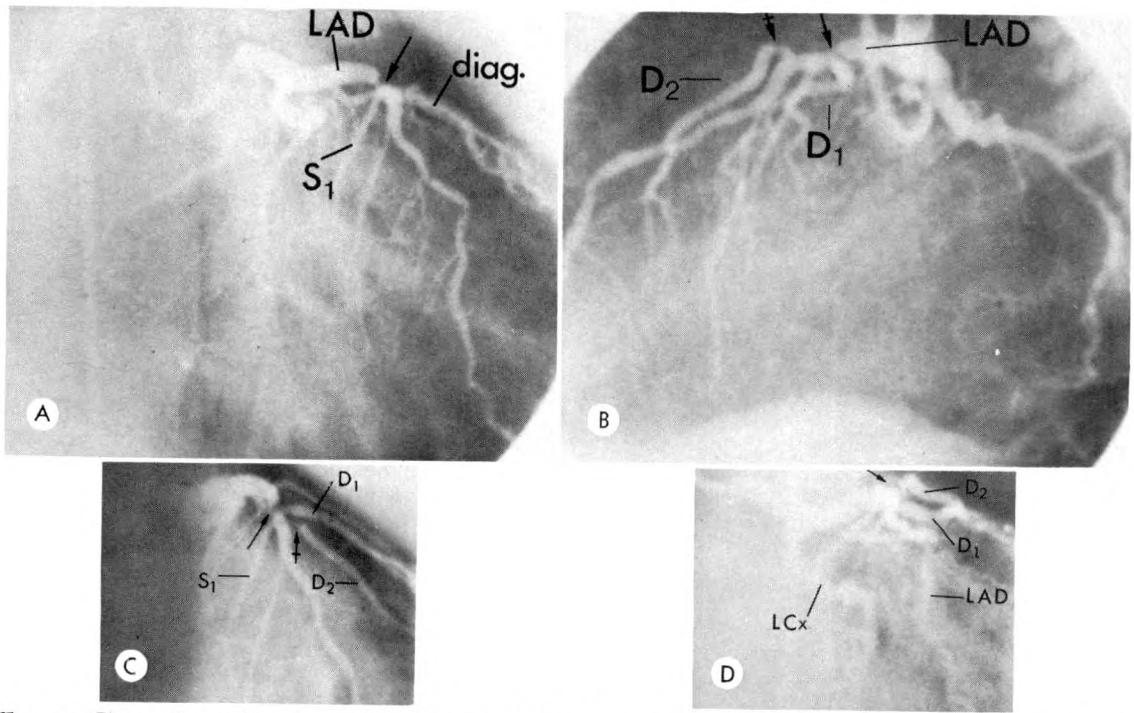


FIG. 10. Cineangiographic views of the left coronary artery illustrating the usefulness of special angulated projections for visualization of severe stenosis on the diagonal artery. The frontal view does not supply any additional information and is not shown. (*A*) The 30° RAO view shows a severe narrowing of the LAD artery (→), proximal to the first septal (S_1) and diagonal arteries (diag.). There is no demonstration of stenosis at the origin of these 2 superimposed diagonal branches. (*B*) The lateral view gives more information: the marked stenosis of the proximal LAD is again visualized and one sees, in addition, a moderate (50 per cent) narrowing of the second diagonal artery (↔). The 60° LAO projection is not contributory. (*C*) The 25° caudo-cranial sagittal angulation which is more perpendicular to the proximal LAD artery permits a good assessment of the severe stenosis of the proximal LAD artery (→) with slight involvement of the origin of the first diagonal artery. This view also demonstrates a short severe (90 per cent) stenosis situated very proximally, at the origin of the second diagonal (D_2) artery (↔). (*D*) The 25° cranio-caudal sagittal angulation again shows the severe stenosis at the origin of D_2 (→). In this view, D_2 is projected more superiorly than D_1.

small vessel coronary artery disease.[11] Myocardial infarction with normal coronary arteries can also be explained by recanalization of a coronary embolus or thrombus.[4] These different pathologic conditions, however, are rare and probably do not account for more than 5 per cent of all patients with angina pectoris. Before one concludes to the presence of normal coronary arteries in these patients, all measures must be taken to exclude potential errors of interpretation.[1,10,12,15]

This is important not only for accurate diagnosis but also for definite assessment of coronary artery obstructions when myocardial revascularization is contemplated Whenever possible, all significant lesions in vessels with an acceptable distal vascular bed should be corrected surgically. Failure to recognize and to bypass a severe lesion of the diagonal or the circumflex artery may be responsible for residual anginal pain after operation.

Underestimation and pitfalls in the interpretation of coronary arteriograms can be frequent. Common sources of error are: poor visualization or lack of visualization of a stenosis because of superimposition of vessels; short, diaphragmatic proximal narrowings; eccentric or slit-like stenosis; er-

roneous identification of coronary artery branches; absence of opacification of collateral or terminal branches; and diffuse cylindrical narrowings. The percentage of these errors is related to the observer's experience as well as to the technical quality of the radiologic document and number of projections used. Indeed, one must reemphasize the importance of obtaining multiple views of the coronary arteries, especially views at right angle from each other, such as the frontal and true lateral projections. The routine use of a true lateral projection is essential to uncover short, diaphragmatic stenoses, to better assess eccentric or slit-like stenosis and to avoid errors in identification of coronary artery branches. When only oblique views are used, large diagonal or first septal branches are sometimes mistaken for the main left anterior descending artery.

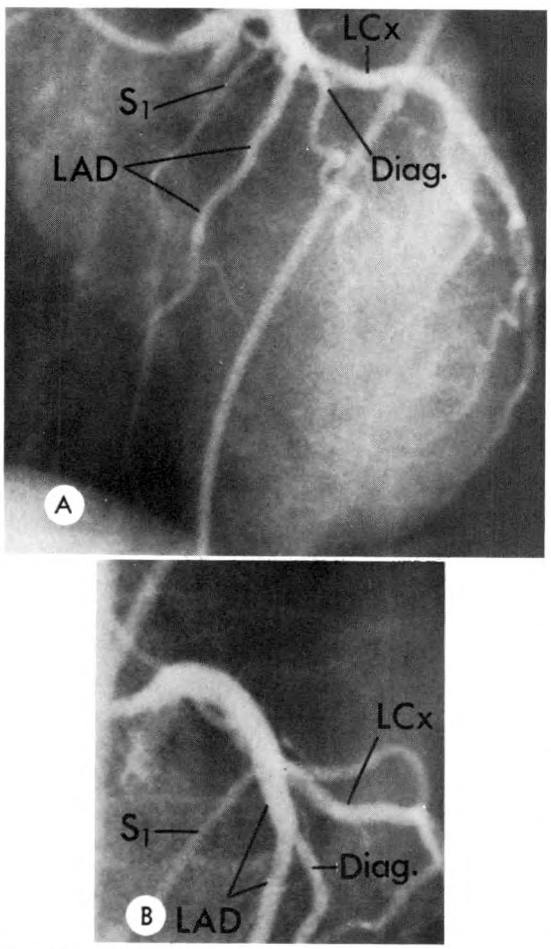


FIG. 11. (*A*) Routine 45° LAO projection of a near normal left coronary arteriogram. Note that the origin of the diagonal artery (diag.) is overlapped by the proximal segment of the circumflex artery (LCx). Also the proximal segment of the LAD artery is seen foreshortened. (*B*) 25° caudo-cranial sagittal angulation (image intensifier inclined towards the head of the patient). In addition, a 15° LAO rotation was used in order to separate the proximal LAD segment from the proximal circumflex artery (LCx). In this view the proximal LAD and the origin of the diagonal artery (diag.) are well visualized and separated from each other and are viewed lengthwise along their longitudinal axis. S_1 = first septal branch.

Even with the routine use of a true lateral projection, one is often confronted with constant superimpositions of proximal branches of the left coronary artery. We have attempted to overcome these difficulties by the systematic use, during the last 18 months, of specially angulated projections in a sagittal plane, either in a cranial or caudal direction or both. These additional views greatly improve visualization of the proximal segments of the left coronary artery. Annoying superimpositions can be clearly separated and the proximal branches of the left coronary artery are also seen lengthwise, along their longitudinal axis, avoiding the tangential or cross-sectional view often obtained in the transverse plane. Stenoses in these areas can be adequately visualized and accurately assessed.

SUMMARY

It has been a standard practice during selective coronary angiography to obtain multiple angulated views of the coronary vessels in a single transverse plane.

These views usually provide an excellent definition of the longitudinally oriented right coronary artery and middle and distal segments of the left coronary artery. However, the proximal branches of the left coronary artery are more transversely oriented and can be parallel to the axis of the x-ray beam in this plane of rotation. This results not infrequently in superimpositions and incomplete tangential views of these

segments. Therefore, stenoses in these areas can be overlooked or underestimated.

We have found that these difficulties can be overcome, in most instances, by the systematic use of additional views in a sagittal plane, angulated approximately 25° either in a cranial or caudal direction or both.

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REFERENCES

1. ADAMS, D. F., ABRAMS, H. L., and RUTTLEY, M. Roentgen pathology of coronary artery disease. *Seminars in Roentgenol.*, 1972, *7*, 319–351.
2. ARBOGAST, R., and BOURASSA, M. G. Myocardial function during atrial pacing in patients with angina and normal coronary arteriograms. *Am. J. Cardiol.*, 1973, *32*, 257–263.
3. BOURASSA, M. G., LESPÉRANCE, J., and CAMPEAU, L. Selective coronary arteriography by percutaneous femoral artery approach. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *107*, 377–383.
4. CAMPEAU, L., LESPÉRANCE, J., BOURASSA, M. G., and ASHEKIAN, P. B. Myocardial infarction without obstructive disease at coronary arteriography. *Canad. M. A. J.*, 1968, *99*, 837–843.
5. ELIOT, R. S., and BRATT, G. Paradox of myocardial ischemia and necrosis in young women with normal coronary arteriograms: relation to abnormal hemoglobin-oxygen dissociation. *Am. J. Cardiol.*, 1969, *23*, 633–638.
6. EUSTERMAN, J. H., ACHOR, R. W. P., KINCAID, O. W., and BROWN, A. L., JR. Atherosclerotic disease of coronary arteries: pathologic-radiologic correlative study. *Circulation*, 1962, *26*, 1288–1295.
7. FRIEDBERG, C. K., and HORN, H. Acute myocardial infarction not due to coronary artery occlusion. *J.A.M.A.*, 1939, *112*, 1675–1682.
8. GRAY, C. R., HOFFMAN, H. A., HAMMOND, W. S., MILLER, K. L., and OSEASOHN, R. O. Correlation of arteriographic and pathologic findings in coronary arteries in man. *Circulation*, 1962, *26*, 494–499.
9. GRONDIN, C. M., LESPÉRANCE, J., BOURASSA, M. G., CASTONGUAY, Y. R., and GRONDIN, P. Preoperative cineangiography and post-mortem findings in patients with aortocoronary vein grafts: cause for some concern. (Abstract.) *Circulation*, 1972, *45–46*, Suppl. II, 11–161.
10. JAMES, T. N. Editorial: Angina without coronary disease. *Circulation*, 1970, *42*, 189–191.
11. JAMES, T. N. Pathology of small coronary arteries. *Am. J. Cardiol.*, 1967, *20*, 679–691.
12. KEMP, H. G., EVANS, H., ELLIOT, W. C., and GORLIN, R. Diagnosis accuracy of selective coronary cinearteriography. *Circulation*, 1967, *36*, 526–533.
13. NEILL, W. A., KASSEBAUM, D. G., and JUDKINS, M. P. Myocardial hypoxia as basis for angina pectoris in patient with normal coronary arteriograms. *New England J. Med.*, 1968, *279*, 789–792.
14. OLIVA, P. B., POTTS, D. E., and PLUSS, R. G. Coronary arterial spasm in Prinzmetal angina: documentation by coronary arteriography. *New England J. Med.*, 1973, *288*, 745–751.
15. SHIREY, E. K., HAWK, W. A., MUKERJI, D., and EFFLER, D. B. Percutaneous myocardial biopsy of left ventricle. *Circulation*, 1972, *46*, 112–122.
16. VLODAVER, Z., FRECH, R., VAN TASSEL, R. A., and EDWARDS, J. E. Correlation of antemortem coronary arteriogram and postmortem specimen. *Circulation*, 1973, *47*, 162–169.

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NEW LORDOTIC PROJECTION FOR IMPROVED VISUALIZATION OF THE LEFT CORONARY ARTERY AND ITS BRANCHES*

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CORONARY arteriography has become an accurate and well established method of demonstrating the anatomy and pathology of the coronary arteries. However, some lesions are missed even with meticulous technique. This may be due to overlapping of multiple arteries, or eccentricity of the lesion. In order to overcome these problems, many authors[1,2,3] have suggested taking multiple oblique and lateral views, especially of the left coronary artery. Nevertheless, the main left coronary artery and its proximal branches are often not well seen in spite of these multiple views. The main left coronary artery is often foreshortened and telescoped upon itself in routine oblique views, especially in the left anterior oblique view (LAO), and the proximal left anterior descending coronary artery (LAD) is often obscured by overlying diagonal branches especially in the right anterior oblique (RAO) view.

A new technique,* utilizing lordotic RAO and LAO projections, allows better visualization of the left coronary artery and its branches.

METHOD

Selective catheterization of the right and left coronary arteries is performed using the technique described by Judkins.[1] Cine filming and rapid serial roentgenograms are obtained in the RAO and the LAO projections. The rapid serial roentgenograms are obtained utilizing a horizontal beam with the patient lying in direct contact with the lateral changer. The lordotic views are incorporated into the routine serial roentgenographic filming sequence of the left coronary artery. The positioning used in the lordotic oblique views is shown in Figures 1, *A* and *B* and 2, *A* and *B*.

The center of the heart is marked on the skin under fluoroscopy with the patient in the supine position. This is used as the reference point for centering the angulated beam in both the lordotic LAO and RAO views. When the left coronary artery is selectively catheterized, serial roentgenograms are obtained in the lordotic RAO and LAO views as well as the routine oblique views. For the lordotic views, the patient is positioned slightly caudally with respect to the changer (to allow the angulated central beam to hit the center of the changer). The roentgenographic tube is angled 45° in a cephalad direction and positioned so that the central ray passes through both the reference point on the patient and the center of the serial film changer. In our laboratory, the roentgenographic factors are 1,000 ma., 0.02 sec., 95 to 105 kv., and 100 cm. target-film distance. In order to maintain the 100 cm. target-film distance with the oblique beam, the tube has to be moved closer to the patient than in the routine views. The grid is positioned with the lines horizontally to avoid grid cutoff. Certain technical problems arise in obtaining the lordotic views. Due to the angulation and the overlapping of the diaphragm, the beam must traverse a greater thickness of tissue in the lordotic view. Thus, to obtain adequate penetra-

* This manuscript was completed but not submitted prior to the publication of a paper by Ivan L. Bunnell *et al.*, Half-axial projection: new look at proximal left coronary artery. *Circulation*, 1973, *48*, 1151–1156, describing a similar LAO projection.

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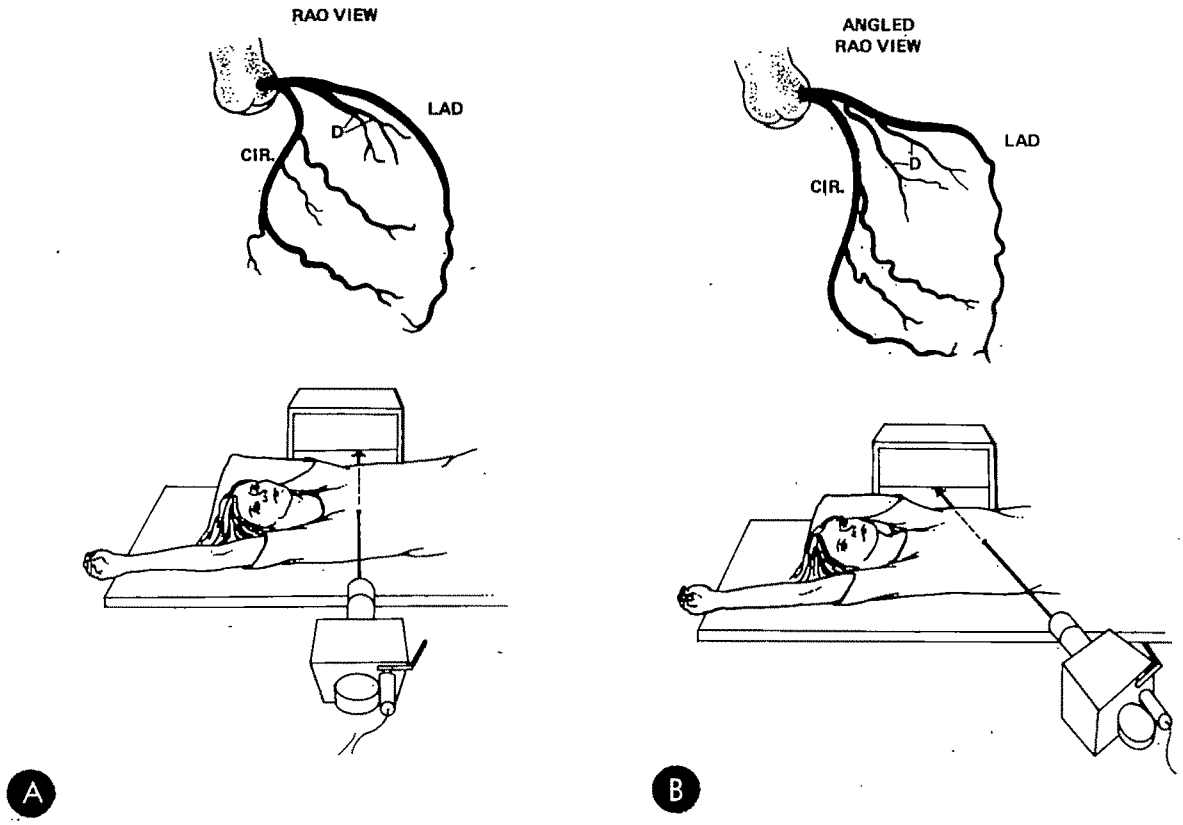


FIG. 1. (*A* and *B*) Positioning for serial filming in the routine and lordotic right anterior oblique (RAO) views of the left coronary artery using the lateral changer. The drawing of the angiogram has been rotated 90° clockwise into the conventional viewing position from its projection on the changer. In order to position the heart as close to the film as possible to obtain the RAO projection, the patient must be in a left anterior oblique (LAO) position with regard to the lateral changer.

tion, a high kv. technique must be used. Increased heart-film distance and longer exposure times are required; thus, the image quality of the lordotic views tends to be less sharp than the routine views. The increased scatter and higher kv. used diminish the roentgenographic contrast. Geometric distortion is also present (lengthening of the coronary artery in the direction of the beam), but this has not hampered the evaluation of the roentgenograms. The resulting roentgenogram is of somewhat inferior quality when compared with routine oblique views using a perpendicular horizontal beam, but the new diagnostic information gained is often invaluable.

MATERIAL

Forty coronary arteriographies were performed, using routine and lordotic oblique projections. The usefulness of the new projection in unmasking or better defining lesions is illustrated by the following cases. In the illustrations for all the projections presented within a given case, the identically numbered arrows denote the same lesion or segment of artery.

REPORT OF CASES

CASE 1. *Lesion in the LAD masked by an overlying diagonal branch. Foreshortening of the main left coronary artery.*

The routine RAO (Fig. 3*A*) and LAO (Fig. 3*B*) views of the left coronary artery show a severe narrowing and diffuse irregularity of the circumflex coronary artery (arrow 1). Minimal irregularity of the proximal LAD (arrow 2) is suspected, but not well defined due to almost complete overlap of the LAD by diagonal

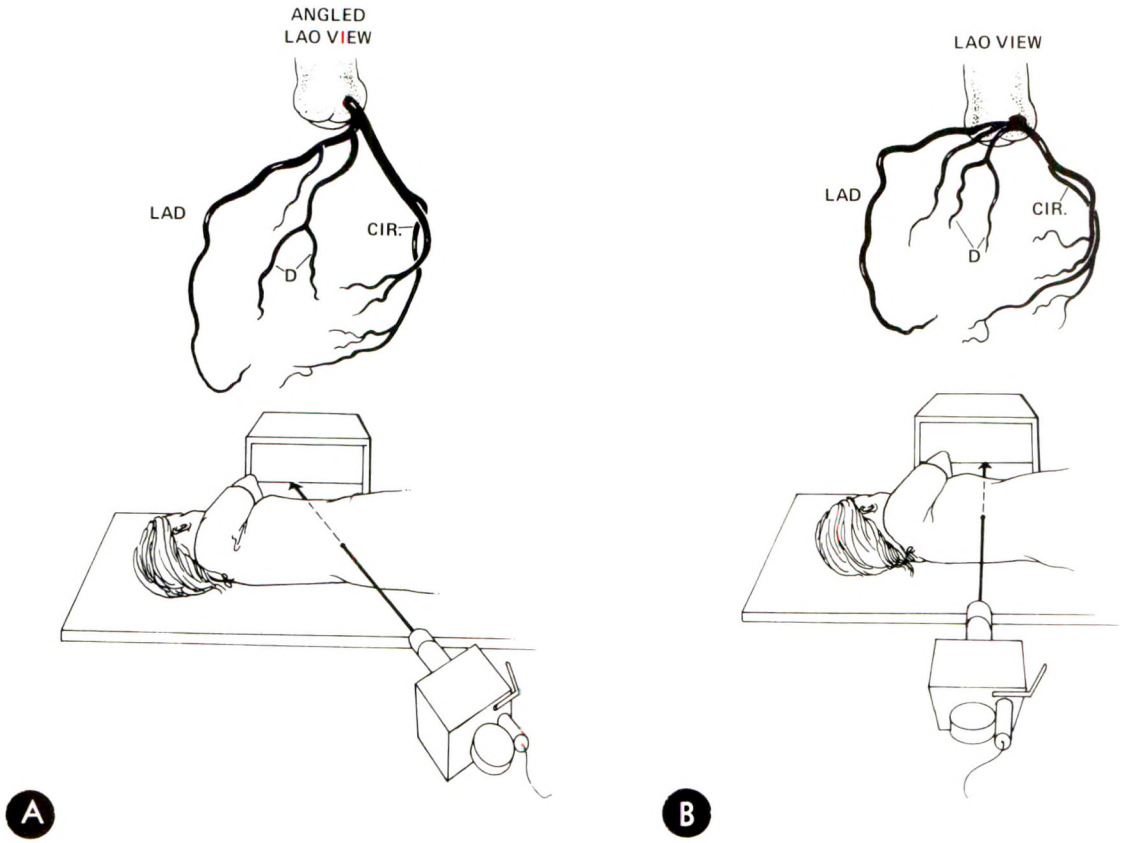


Fig. 2. (*A* and *B*) Positioning for serial filming in the routine and lordotic left anterior oblique (LAO) views of the left coronary artery using the lateral changer. The drawing of the angiogram has been rotated 90° clockwise into the conventional viewing position from its projection on the changer.

branches in the RAO and partial overlap in the LAO projection. The main left coronary artery (ML) is telescoped upon itself and a lesion in it could be missed.

The lordotic RAO view (Fig. 3*C*) completely separates those vessels and demonstrates a severe narrowing of the LAD just distal to the origin of a large diagonal branch (arrow 2). In addition, the main left coronary artery (ML) which appears markedly foreshortened in both the routine LAO and RAO views is now seen in its entirety and even the circumflex lesions (arrow 1) are optimally defined.

Case II. *Lesions in the origins of the LAD and a major diagonal branch obscured by overlap and foreshortening.*

The routine LAO view (Fig. 4*A*) appears entirely normal with considerable overlap and foreshortening of the main left coronary artery and of the proximal portion of its branches (arrows 1 and 2). The routine RAO view (Fig. 4*B*) demonstrates only very slight irregularity at the origin of the major diagonal branch (arrow 1) in this patient with angina pectoris documented by electrocardiographic changes.

The RAO lordotic projection (Fig. 4*C*) separates these vessels and demonstrates a 60–70 per cent irregular narrowing of the proximal 1 cm. of the major diagonal branch (arrow 1). In addition, the main left coronary artery is better seen and a smooth 30–40 per cent narrowing of the origin of the LAD (arrow 2) is demonstrated.

Case III. *Superimposition of the main left, proximal LAD and circumflex coronary arteries masking the presence and severity of multiple LAD lesions.*

The routine LAO view (Fig. 5*A*) demonstrates severe disease of the circumflex coronary artery (arrow 1) with segmental occlusion. A

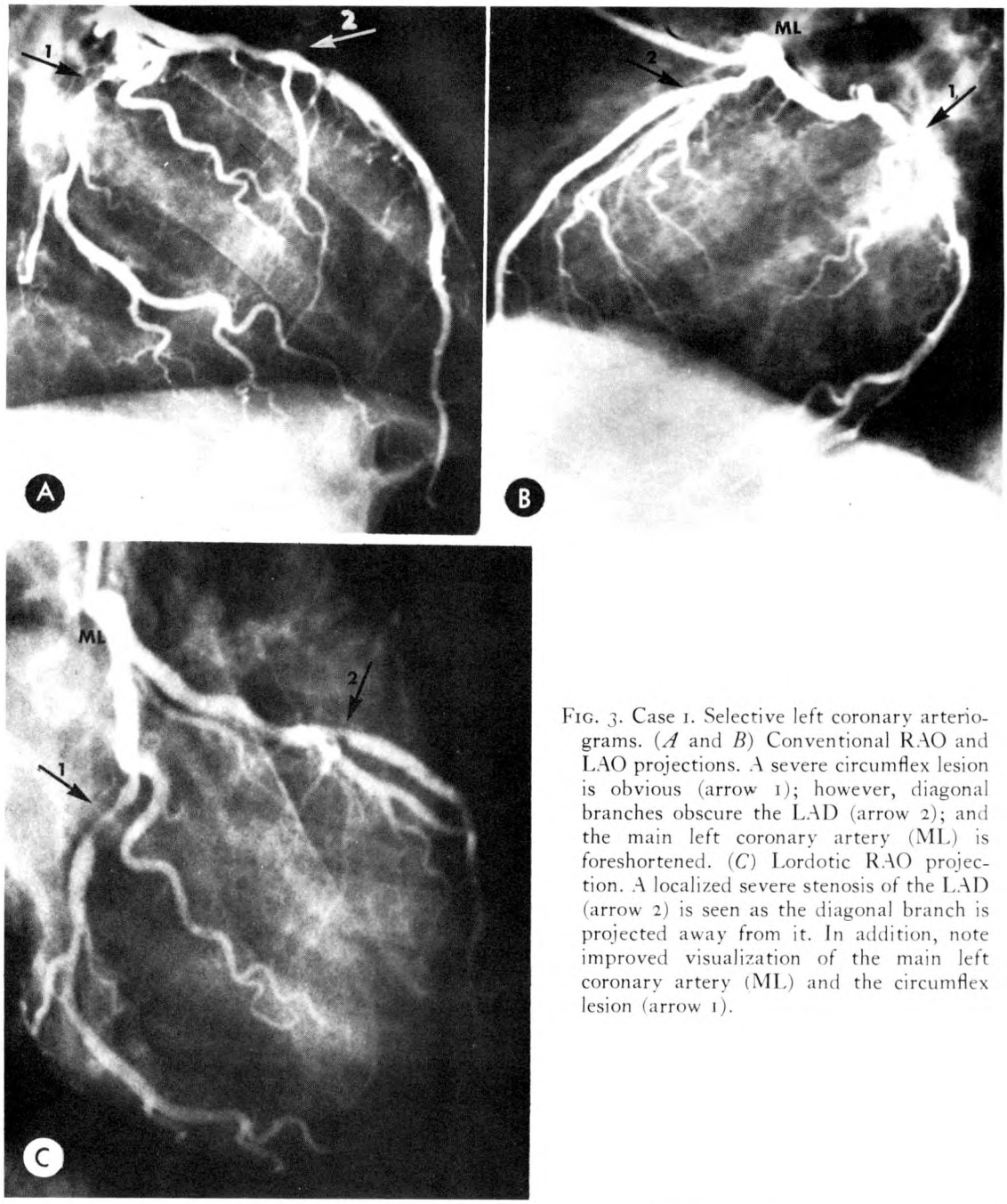


FIG. 3. Case 1. Selective left coronary arteriograms. (*A* and *B*) Conventional RAO and LAO projections. A severe circumflex lesion is obvious (arrow 1); however, diagonal branches obscure the LAD (arrow 2); and the main left coronary artery (ML) is foreshortened. (*C*) Lordotic RAO projection. A localized severe stenosis of the LAD (arrow 2) is seen as the diagonal branch is projected away from it. In addition, note improved visualization of the main left coronary artery (ML) and the circumflex lesion (arrow 1).

mild plaque in the proximal LAD (arrow 2) is seen. The main left, proximal LAD and proximal circumflex coronary arteries are completely superimposed (arrow 3). The routine RAO view (Fig. 5*B*) also demonstrates the circumflex lesions (arrow 1); however, the main left, proximal LAD and proximal circumflex are again almost completely obscured by each other (arrow 3). A severe lesion of the LAD just distal to its large diagonal branch is suspected but not clearly demonstrated (arrow 4).

The lordotic RAO view (Fig. 5*C*) clearly demonstrates the short main left coronary artery. The proximal circumflex coronary artery and its first small obtuse marginal branch are now separated from the proximal LAD and re-

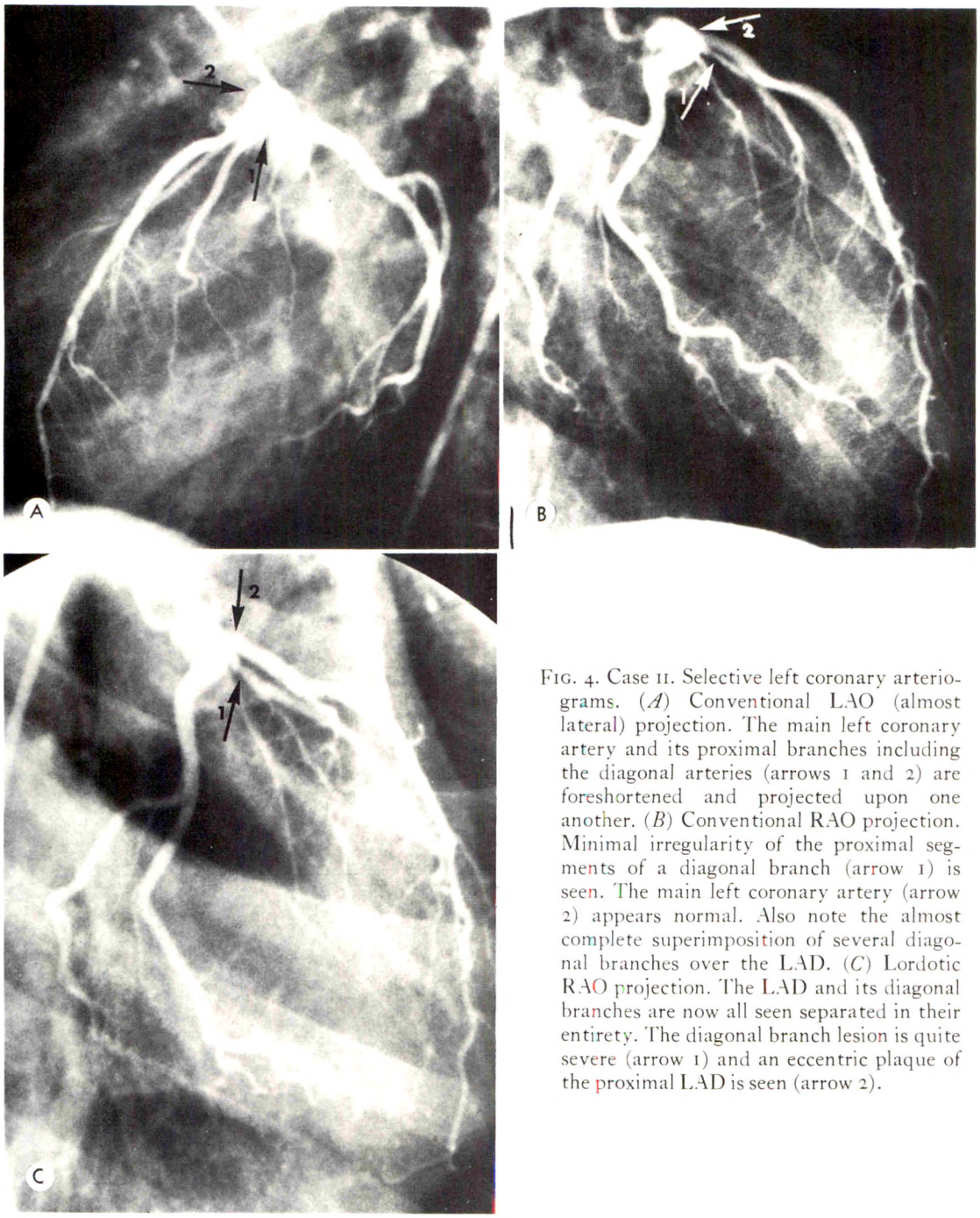


Fig. 4. Case 11. Selective left coronary arteriograms. (*A*) Conventional LAO (almost lateral) projection. The main left coronary artery and its proximal branches including the diagonal arteries (arrows 1 and 2) are foreshortened and projected upon one another. (*B*) Conventional RAO projection. Minimal irregularity of the proximal segments of a diagonal branch (arrow 1) is seen. The main left coronary artery (arrow 2) appears normal. Also note the almost complete superimposition of several diagonal branches over the LAD. (*C*) Lordotic RAO projection. The LAD and its diagonal branches are now all seen separated in their entirety. The diagonal branch lesion is quite severe (arrow 1) and an eccentric plaque of the proximal LAD is seen (arrow 2).

veal an unsuspected long severe eccentric plaque (arrow 3) demonstrated by irregular and decreased opacification of the lumen of this segment. Just distal to the large diagonal branch, the diagonal and LAD branches are separated and a 1 cm. segment of complete occlusion of the LAD (arrow 4) followed by a 1 cm. narrowed segment (arrow 2) is shown, whereas only a narrowing was suspected on routine views (Fig. 5, *A* and *B*; arrows 2 and 4).

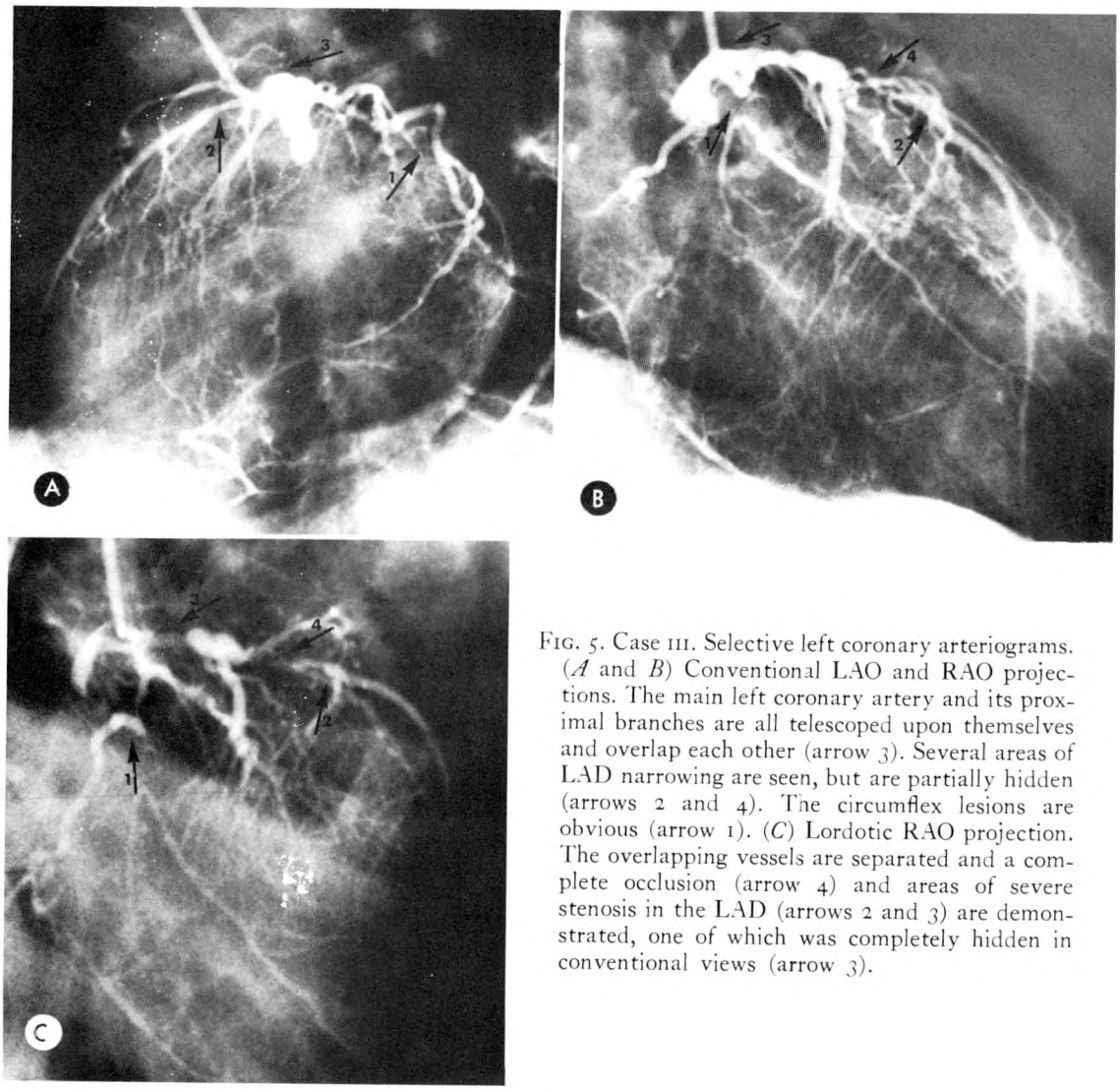


FIG. 5. Case III. Selective left coronary arteriograms. (*A* and *B*) Conventional LAO and RAO projections. The main left coronary artery and its proximal branches are all telescoped upon themselves and overlap each other (arrow 3). Several areas of LAD narrowing are seen, but are partially hidden (arrows 2 and 4). The circumflex lesions are obvious (arrow 1). (*C*) Lordotic RAO projection. The overlapping vessels are separated and a complete occlusion (arrow 4) and areas of severe stenosis in the LAD (arrows 2 and 3) are demonstrated, one of which was completely hidden in conventional views (arrow 3).

CASE IV. *Severe lesions in the LAD and diagonal coronary arteries obscured by their overlap.*

A single severe lesion in the LAD (arrow 1) and mild diffuse plaques in the LAD and circumflex coronary arteries (arrows 2, 3 and 4) are suggested by the routine RAO view (Fig. 6*A*). The routine LAO view (Fig. 6*B*) demonstrates a severe single lesion in the LAD (arrow 1) and suggests the presence of another area of stenosis (arrow 2) partially overlain by a large diagonal branch. The main left and proximal LAD are markedly foreshortened (arrow 3) and cannot be adequately evaluated.

The lordotic LAO view (Fig. 6*C*) optimally displays and separates from each other the main left, LAD and diagonal coronary arteries demonstrating the above lesions (arrows 1, 2 and 4) and some stenoses hidden on routine views. There are moderately severe diffuse plaques in the main left and proximal LAD coronary arteries and a severe stenosis at the origin of the first major diagonal artery is also shown (arrow 3).

DISCUSSION

The above cases show how the lordotic LAO and RAO views have been helpful in detecting and clarifying focal atherosclerotic lesions in the main left coronary artery

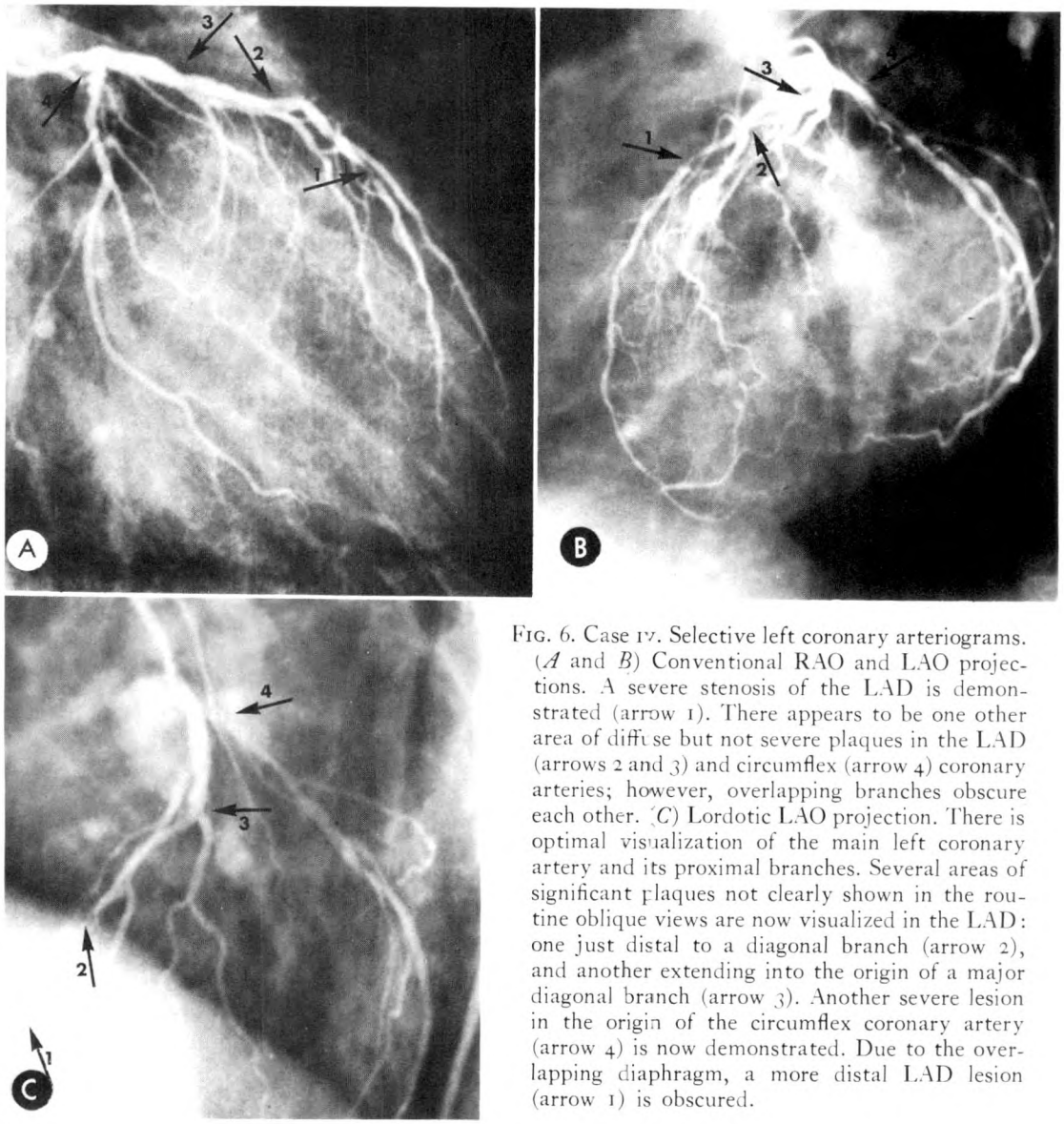


FIG. 6. Case IV. Selective left coronary arteriograms. (*A* and *B*) Conventional RAO and LAO projections. A severe stenosis of the LAD is demonstrated (arrow 1). There appears to be one other area of diffuse but not severe plaques in the LAD (arrows 2 and 3) and circumflex (arrow 4) coronary arteries; however, overlapping branches obscure each other. (*C*) Lordotic LAO projection. There is optimal visualization of the main left coronary artery and its proximal branches. Several areas of significant plaques not clearly shown in the routine oblique views are now visualized in the LAD: one just distal to a diagonal branch (arrow 2), and another extending into the origin of a major diagonal branch (arrow 3). Another severe lesion in the origin of the circumflex coronary artery (arrow 4) is now demonstrated. Due to the overlapping diaphragm, a more distal LAD lesion (arrow 1) is obscured.

and its proximal branches. This technique has been particularly helpful in cases in which there is foreshortening of the main left coronary artery or overlapping of the LAD and its diagonal branches in the routine oblique projections. Foreshortening often occurs in patients with a horizontal heart due to a high diaphragm or in patients with left ventricular dilatation.

At this time, it is not feasible to use the lordotic views performing conventional cinefluorography. When isocentric units become available, it will be possible to obtain the lordotic views using cine or 105 mm. spot filming. The isocentric unit is one in which the roentgenographic tube and the image intensifier are mounted in a rigid C or U arm, with provisions for moving the arm in a horizontal as well as vertical axis. With such equipment lordotic views in any projection can easily be obtained without moving the patient. In

addition, geometric distortion will not be present since the roentgenographic beam will be perpendicular to the intensifier screen at all times.

SUMMARY

Two new lordotic oblique projections of the left coronary artery are described.

When the LAO view of the left coronary artery is obtained with the horizontal beam angled 45° cephalad, the main left coronary artery, the proximal left anterior descending coronary artery, and the diagonal branches may be optimally seen.

When the angulated horizontal beam is used to obtain the RAO view of the left coronary artery, the proximal LAD is seen with the diagonal branches projected away from it.

The roentgenographic technique of the lordotic views is discussed. Coronary arteriograms are presented to demonstrate the usefulness of these new views.

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REFERENCES

1. JUDKINS, M. P. Percutaneous transfemoral selective coronary arteriography. *Radiol. Clin. North America*, 1968, *6*, 467–492.
2. SONES, F. M. Cine coronary arteriography. In: The Heart. Second edition. Edited by J. W. Hurst and R. B. Logue. McGraw-Hill Book Company, Inc., New York, 1970, pp. 377–385.
3. VLODAVER, Z., FRECH, R., VAN TASSEL, R. A., and EDWARDS, J. E. Correlation of the antemortem coronary arteriogram and the postmortem specimen. *Circulation*, 1973, *48*, 162–169.

CORONARY ARTERIOGRAPHY IN LEFT MAIN CORONARY ARTERY DISEASE*

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TWO basic questions arise in examining patients with left main coronary artery disease (LMCAD): What special risk does selective coronary arteriography impose in the presence of LMCAD? Is either the Sones or the Judkins method for selective coronary visualization* intrinsically safer and therefore preferable in LMCAD?

In 2 recent series, an average mortality of 13 per cent was experienced in relation to coronary arteriography in 62 patients with LMCAD.[3,8] Of 30 patients examined by Sones' method by Lavine and co-workers,[8] 2 died of acute heart failure during catheterization, and 1 of cardiac arrest 5 hours after an uncomplicated study. Two of 24 patients Cohen and co-workers[3] examined by Sones' method died of evolving myocardial infarction, while 3 of 8 they examined by Judkins' method had fatal cardiac arrest on introduction of the catheter into the left main coronary artery (LMCA). Cohen and co-workers believe that the Sones method is preferable for patients with LMCAD.[3] Bruschke and co-workers'[2] review of 37 medically treated patients with LMCAD who were uneventfully studied in Sones' laboratories seems to support this belief. While we agree that the Sones method can be done with reasonable safety, we do not consider it either safer or preferable to the Judkins method in patients with LMCAD. We have performed 67 coronary arteriographies with the Judkins method on 51 patients with significant LMCAD. No complication occurred during arteriography. One patient with advanced 4 vessel disease and established congestive heart failure developed ventricular fibrillation 6 hours after an uneventful study and died 3 days later.

* *Sones' method* uses right brachial arteriotomy, the same pliable catheter for left and right coronary catheterization and cine coronary arteriography.[14]

Judkins' method uses a percutaneous transfemoral approach, specifically preshaped, torque-controlled catheters, different for left and right coronary catheterization and both cine and serial filming.[5,6]

CASE MATERIAL AND CLINICAL DATA

Fifty-one patients with significant obstruction (more than 50 per cent) of the LMCA represent 2.4 per cent of approximately 2,100 patients examined from 1966 to 1973 at the University of Oregon Medical School. There were 41 men and 10 women; their ages averaged 54 years (range of 42 to 71). Nineteen patients had angina at rest, 24 patients had disabling angina, and 4 had less than 1 episode of angina a day. The remaining 4 patients with valvular disease examined prior to surgery had no angina, but were limited by valvular symptoms. Eleven patients had a history of myocardial infarction, 11 had hypertension, 3 diabetes, and 16 of 30 examined patients had lipid abnormalities. Electrocardiograms showed prior myocardial infarction in 12 patients, ischemic ST depression in 5, nonspecific ST-T abnormalities in 15, left ventricular hypertrophy in 10, and were normal in 8 patients. Stress electrocardiograms done in 13 patients were positive in 9. None of the patients had positive histories or tests for syphilis.

METHOD

Thirty-six patients had 1 study; 14 had 2 studies, both before and after myocardial

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revascularization surgery. One patient had 3 studies. All studies were done by the Judkins percutaneous technique, using the transfemoral approach for 65 examinations and the left transaxillary approach twice.[5,6,10] Secobarbital (Seconal, Lilly), (100 to 200 mg.) and atropine (0.6 mg.) were used intramuscularly as premedication. Continuous electrocardiographic monitoring was done routinely; for possible later use, an intravenous tube was inserted at the outset of the procedure in most patients. Preshaped polyurethane Ducor† or polyethylene Torcon‡ catheters, usually 8F, were used with 7F for small patients or axillary approach. Catheter tip pressures were continuously monitored, except for the moment of angiographic injection.[4–6]

Ordinarily, a pigtail catheter was first introduced for pressure studies in the aorta and left ventricle. Left ventriculography with cine filming in the right anterior oblique (RAO) projection followed, except in patients whose left ventricle end-diastolic pressure exceeded 25 mm. Hg. In such patients, it was done when possible after coronary studies.

Left coronary arteriography was the next step in the procedure. Prophylactic sublingual nitroglycerin (1/150th gr.) was given, a left coronary catheter introduced and placed in the aortic arch. With the patient in a slight left anterior oblique (LAO) position, the catheter was then advanced slowly until its tip reached the level of the LMCA. Its origin was then visualized and recorded on cine through a nonselective injection of 6 to 8 cc. of contrast medium.§ Unless there was evidence of serious ostial stenosis, the catheter was then slowly advanced into the artery's ostium, while the catheter tip pressure was closely observed. Four to 6 cc. of contrast medium was then injected (RAO projection, cine recording) to visualize the whole LMCA. If diffuse LMCA involvement or localized stenosis was shown within reach of the catheter tip, the catheter was left in the ostium of the LMCA. In peripheral stenosis of a long LMCA, it was advanced into a more stable position, again with strict attention to catheter tip pressure. Selective injections were made only when the catheter tip was completely free in the LMCA, as evidenced by an undamped pressure. Depending on the apparent capacity of the left coronary system, 6 to 9 cc. of contrast medium were injected by hand. Filming depended on the type and degree of stenosis. In 4 patients with ostial stenosis exceeding 70 per cent, the examination was limited to cineangiography in the LAO, RAO and lateral projections. The other 47 patients had 62 complete coronary studies, which consisted of cineangiography in LAO and RAO projections and of serial roentgenography, usually in lateral and both oblique projections.

After left coronary arteriography was completed, selective right coronary arteriography, and, in patients after bypass graft surgery, selective graft visualization were done.[13]

ANGIOGRAPHIC FINDINGS

There were calcifications in the LMCA area (seen best on fluoroscopy) in 11 patients (22 per cent). Restrictive luminal disease of the LMCA was of variable location and degree (Table 1). It was localized to its origin in 10 patients (Fig. 1), the distal portion in 27 (Fig. 2–4), and involved the LMCA diffusely in 14 patients (Fig. 5–7). In 12 of 27 patients with stenosis of the distal LMCA, the length of the LMCA was less than 12 mm. (Fig. 2); thus the lesion was within possible reach of the catheter tip. In the other 15 patients the LMCA length was 12 to 23 mm. (Fig. 3; and 4). Obstruction of the LMCA was measured as a percentage decrease in diameter and ranged from 50–70 per cent in 23 patients, from 70–90 per cent in 24, and exceeded 90 per cent in 4 patients. Stenosis of the origin or distal part of the LMCA was mostly concentric with only slightly irregular outlines (30 patients, 81 per

† Cordis Corporation, Miami, Florida 33137.
‡ Cook, Incorporated, Bloomington, Indiana 47401.
§ Meglumine diatrizoate (Renografin 76%), Squibb and Sons, New Jersey 08540.

Table I

DISTRIBUTION OF LEFT MAIN CORONARY ARTERY DISEASE (LMCAD) IN 51 PATIENTS

Location of LMCAD	Percentage decrease in left main coronary artery (LMCA) diameter		
	50%–70%	70%–90%	>90%
Origin of LMCA	6	4	
Distal part of LMCA	11	13	3
Diffuse involvement of LMCA	6	7	1
Total	23	24	4

cent), (Fig. 1–4). Diffuse LMCAD usually presented as irregular tubular narrowing (Fig. 5–7), and in 3 cases atherosclerotic ulcerations were also present (Fig. 7).

Significant coronary disease was restricted to the LMCA in only 3 of the 51 patients in this series; the other 48 also had significant involvement of other coronary arteries (Fig. 2–7). Both coronary systems were involved in 36 patients, the left coronary artery system in 7, and the right system in 5 patients. Twenty-four patients had complete occlusion of the main right coronary artery. Significant collaterals were

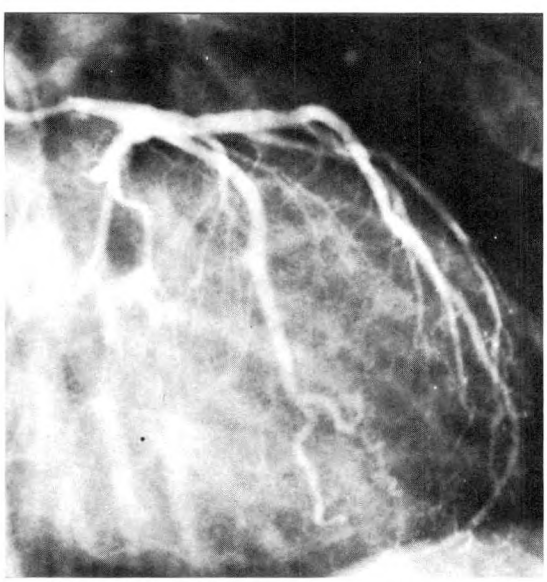


Fig. 1. An approximately 50 per cent narrowing and calcification of the LMCA (left main coronary artery) origin (58 year old man, selective left coronary arteriogram, RAO [right anterior oblique] projection).

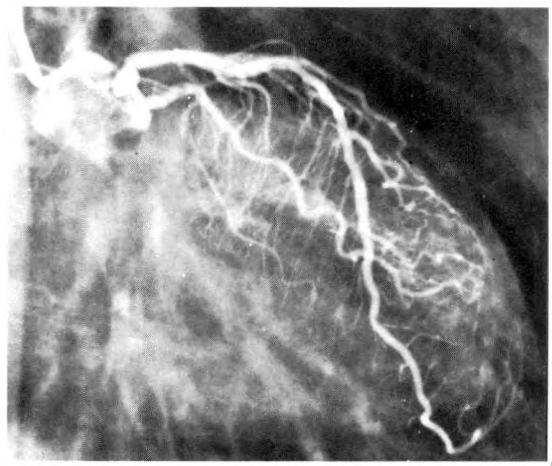


Fig. 2. An approximately 95 per cent narrowing of the peripheral portion of a short (11 mm. long) LMCA and occlusive disease of the circumflex artery (55 year old man, selective left coronary arteriogram, RAO projection).

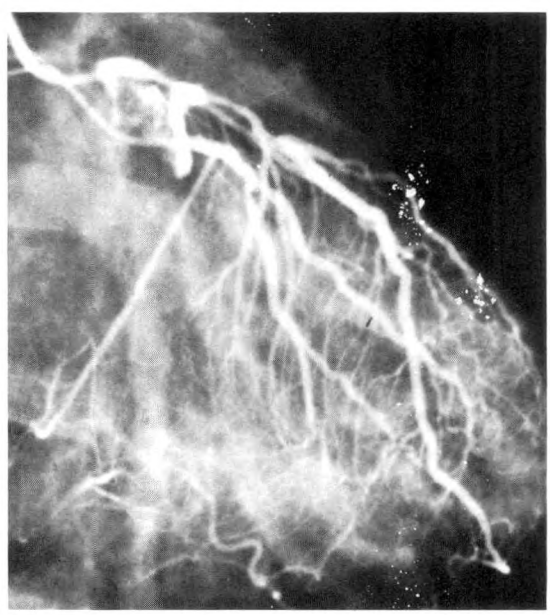


Fig. 3. An approximately 75 per cent narrowing of the peripheral portion of a 15 mm. long LMCA and obstructive disease of the left anterior descending, circumflex and right coronary arteries (55 year old man, selective left coronary arteriogram, RAO projection).

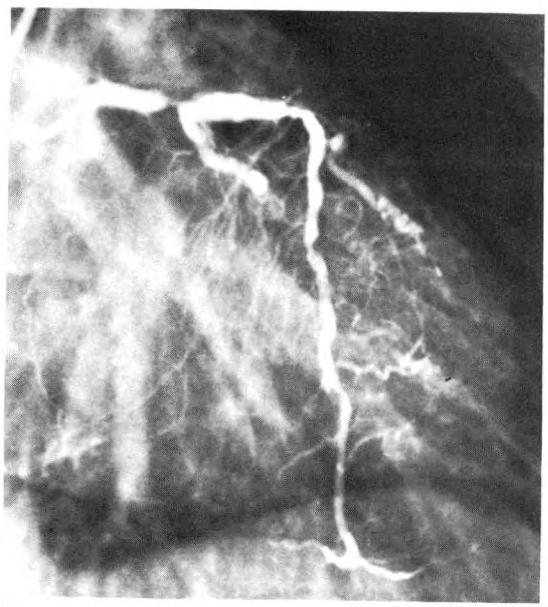


Fig. 4. An approximately 80 per cent narrowing of the peripheral portion of a 20 mm. long LMCA and advanced obstructive disease of the circumflex, left anterior descending, diagonal and right coronary arteries (50 year old man, selective left coronary arteriogram, RAO projection).

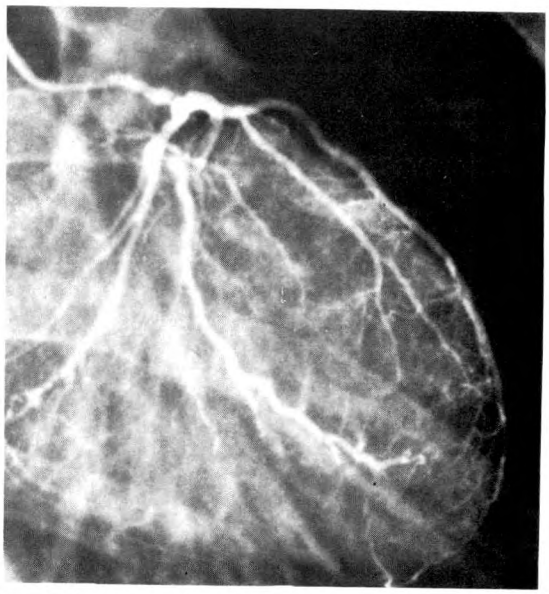


Fig. 6. An approximately 50 per cent diffuse and 95 per cent distal narrowing of the LMCA and occlusive disease of the left anterior descending and right coronary arteries (42 year old man, selective left coronary arteriogram, RAO projection).

seen in 40 patients. Their direction was from right to left in 15, left to right in 18, and left to left in 7 patients. The left system was dominant in 5, the right in 40 and both systems were balanced in 6 patients. Left ventriculography showed asynergic contraction in 32 patients. Left ventricular

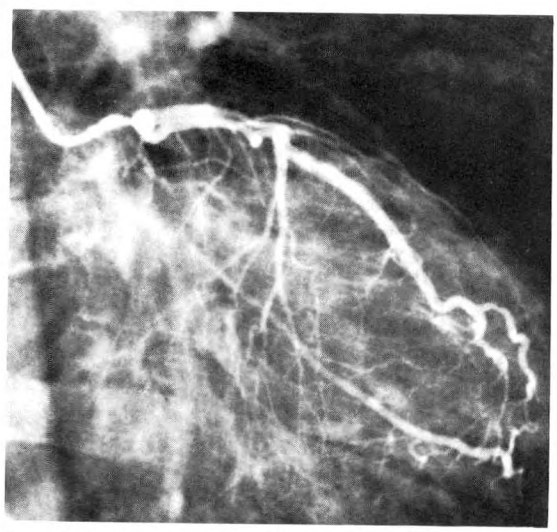


Fig. 5. An approximately 60 per cent diffuse narrowing of the LMCA and obstructive disease of the left anterior descending and circumflex arteries (55 year old man, selective left coronary arteriogram, RAO projection).

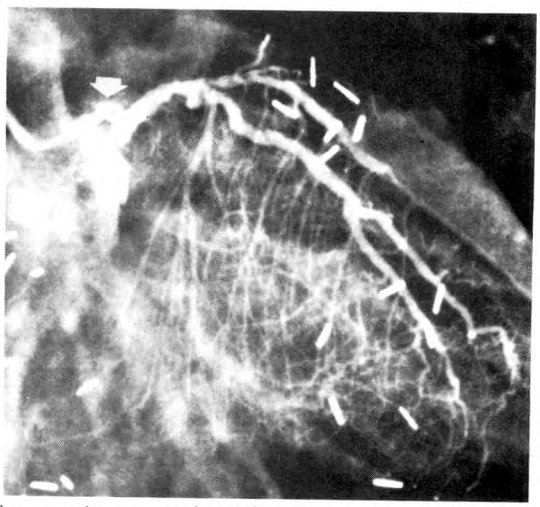


Fig. 7. An approximately 70 per cent narrowing and atherosclerotic ulceration (arrowhead) of the LMCA and occlusive disease of the left anterior descending and circumflex arteries (60 year old man, selective left coronary arteriogram, RAO projection).

end-diastolic pressures ranged from 8 to 35, with a mean of 16 mm. Hg.

COMPLICATIONS

Patients occasionally experienced chest pain during examination, in which case the catheter was withdrawn from the LMCA and additional nitroglycerin given. After the disappearance of pain the examination was resumed. Occasionally pronounced bradycardia developed after coronary injection, in which case the catheter was withdrawn from the LMCA, 0.4–0.6 mg. of atropine injected intravenously, and the procedure was then continued, without major rhythm disturbances. No major complication occurred during angiography in this series. In particular, neither subintimal injection nor dissection of the LMCA occurred, nor was there serious hypotension, ventricular fibrillation, myocardial infarction, cardiac failure, or cardiac arrest.

One patient died 3 days after an uneventful study. He was a 62 year old man with diabetes, an 8 year history of angina and a 3 year history of mild heart failure. Two months before the study, a myocardial infarct led to an increase in his angina and heart failure. The prestudy ECG showed atrial fibrillation, frequent premature ventricular contractions and nonspecific ST-T abnormalities. Coronary arteriography demonstrated advanced four vessel coronary disease with 50 per cent stenosis of the middle part of the LMCA, proximal occlusions of the left anterior descending, circumflex and right coronary arteries and multiple stenotic lesions in the distal vessels. Left ventricular end-diastolic pressure was 35 mm. Hg. Left ventriculography was not done. There were no complications during or immediately after angiography. About 3 hours later he started to complain of nausea and epigastric pain, the frequency of premature ventricular contractions increased, and 6 hours after the procedure, ventricular fibrillation developed. Defibrillation was initially successful, but recurrent fibrillations with cardiopulmonary arrests led to his death 3 days after angiography. Autopsy revealed advanced coronary artery disease with scattered myocardial fibrosis and a resolving 2 to 4 month old myocardial infarct. There was no evidence of a fresh infarct.

DISCUSSION

Obstructive disease of the LMCA, which supplies most of the left ventricle by its branches—the left anterior descending and circumflex arteries—carries a poor prognosis. Only half of patients with nontreated LMCAD survive 2 to 3 years and many die suddenly.[2,3] Aortocoronary bypass graft surgery is apparently the treatment of choice in LMCAD.[3,8] To select patients for surgery, accurate information about the extent of LMCAD, the involvement of peripheral coronary vessels, their suitability for bypass grafting and the status of left ventricular function is essential. Such information can be obtained only by high quality coronary arteriography and left ventriculography.

The presence of LMCAD heightens the risk of selective coronary arteriography and requires consideration of several factors. Since LMCAD often reflects severe generalized coronary disease with impaired myocardial function, arteriography and left ventriculography with multiple injections of contrast medium, causing volume overload, can lead to acute heart failure. Local damage to the LMCA by a catheter is another important factor. Catheter manipulation in the diseased artery can acutely aggravate left ventricular ischemia by causing local spasm, intimal dissection, the dislodgement of atheromatous plaques or thrombi, or through its physical presence, a complete obstruction of flow through a stenosis.

It has been said that local damage is due to the type of catheter and that torque-controlled, preshaped catheters are dangerous.[3] Cohen and co-workers[3] had a 37 per cent mortality with the Judkins method and an 8 per cent mortality with the Sones method, and concluded that the latter

method was safer. However, the complication and mortality rate reflect more than just the methods or catheters used. Bruschke and co-workers[2] reported 37 medically treated patients with LMCAD, who were studied by the Sones method without immediate complications. Our series, including 67 immediately uncomplicated studies in 51 patients, shows that coronary arteriography can be done also with high safety in the presence of LMCAD by the Judkins method. There were later fatalities in both series, related probably to the severity of disease. One of our patients died 3 days after the procedure and 3 of the patients of Bruschke *et al.*[2] within a week.

Clearly, the choice of method for studying LMCAD will continue to reflect the individual angiographer's preference and experience. However, whatever technique is used, clinical judgment and technical competence are of particular importance when coronary arteriography is done in the presence of LMCAD.

The possibility of LMCAD has to be kept in mind when examining patients with severe angina (46 per cent of our patients, 53 per cent of Lavine *et al.*[8]), angina at rest, or in a crescendo pattern (38 per cent of our patients, 36.6 per cent of Lavine *et al.*[8]), and coexisting angina and dyspnea (38 per cent of the patients of Cohen *et al.*[3]). LMCAD is likely when angina and ventricular failure are easily induced by atrial pacing[8] and in the presence of a highly positive exercise electrocardiogram with an R-ST depression exceeding 2 mm. (present in 70 per cent of the patients of Cohen *et al.*[3]). Calcification of the LMCA, best seen on fluoroscopy, is an important and reliable indication of possible LMCAD. However, it appears in less than one-fourth of the instances (22 per cent in our series, 23 per cent in the series of Lavine *et al.*[8]).

Precautionary measures include the placement of an intravenous catheter for possible emergency drug administration, the use of intramuscular atropine for premedication and its additional intravenous use during the procedure, if bradycardia occurs. The placement of a temporary transvenous pacemaker is an important preventive adjunct to the study of patients with high grade block, intrafascicular disease, or bradyarrhythmias not responding to intravenous atropine. Other safety measures include the use of nitroglycerin prior to selective coronary catheterization and whenever catheter manipulation seems likely to lead to coronary artery spasm.

Meticulous technique is imperative and includes continuous monitoring of catheter tip pressure; skilled, cautious catheter manipulation; and selective injections of contrast medium only while the catheter tip lies free within the LMCA. Vigilant, continuous catheter tip pressure monitoring is crucial, particularly when the catheter is advanced or the patient's position changed. Should there be damping or a fall in pressure indicating impingement, the catheter must be withdrawn immediately into a free position, usually into the aorta. Depending on the patient's status and symptoms, nitroglycerin is then used, the catheter reinserted and the procedure continued.

Cautious catheter manipulation is important, especially with the use of a preshaped, torque-controlled catheter. All torque must be removed in the aortic arch, and further advance down the ascending aorta done slowly without rotation.

A logical plan of examination serves safety. The study should progress through steps chosen on the basis of the findings. The ostium of the LMCA is evaluated first by a nonselective injection of contrast medium in slight (10°–20°) LAO projection, with coned down cine filming. Should there be seen fluoroscopically a stenosis likely to be completely blocked by a catheter tip, selective studies should be omitted (a rare situation not encountered in our series). This excluded, the catheter is carefully advanced into the ostium and the whole LMCA evaluated by an injection of contrast medium made in slight RAO projection (10° to 20°). Tightly coned down cineangiography is again done. We consider the slight RAO projection as most in-

formative about lesions of the LMCA (Fig. 1–7). If diffuse, irregular involvement of the LMCA or localized proximal LMCA stenosis within reach of the catheter tip are seen on fluoroscopy, then the catheter is not advanced beyond the ostium of the LMCA. If there is a stenosis in the peripheral part of a long LMCA (more than 12 mm.), the catheter can usually be advanced into a more stable position. In either case, injections are made and the whole left system visualized. Injections into the ostium of the LMCA should start slowly to prevent catheter recoil from this relatively less stable position.

The selection of filming technique (*i.e.*, whether to employ cineangiography alone, or in combination with large film serial roentgenography) should be influenced by both the initial anatomic findings and the efficiency of the technical personnel. We have done both in most patients. We restricted the examination to cineangiography in only 4 patients with severe ostial stenosis. With less experienced personnel, however, it might be necessary to proceed without large film studies in order to minimize the time the catheter is in the LMCA. Cineangiography plus rapid serial, 70 mm. or 105 mm. fluorography, by eliminating the need to move the patient to a film changer, may simplify matters without the significant sacrifice of anatomic information represented by cine alone.

Postangiographic care is as important as that given during the procedure. Hypotension in particular should be avoided or promptly treated, if it occurs. Patients with LMCAD and advanced diffuse coronary disease may become hypotensive easily. Coronary arteriography and left ventriculography, by further depressing an already impaired left ventricular function,[1,11,12] plus volume overloading due to the hyperosmolar effect of contrast medium,[7] may cause acute heart failure during or shortly after the procedure. Subsequently, increased diuresis may cause volume depletion,[9] a mechanism we suspect led to the loss of our patient after an uneventful study. All patients must therefore have frequent blood pressure checks after the procedure and be given fluids orally or intravenously in sufficient amount to avoid dehydration. Although we have not kept our patients in a coronary care unit, this would presumably be useful.

SUMMARY

1. The presence of left main coronary artery disease (LMCAD) increases both the risk and the importance of coronary arteriography, an essential in the selection of patients for aortocoronary bypass surgery.

2. Our experience in the performance of 67 coronary arteriographies in 51 patients with significant LMCAD contradicts published views and shows that the Judkins method can be used with safety in the presence of LMCAD.

3. Clinical judgment and technical competence are of particular importance when coronary arteriography is done in the presence of LMCAD.

4. Careful patient evaluation with precautionary measures—skilled, meticulous catheterization technique, vigilant catheter tip pressure monitoring, a logical progressive plan of examination and adequate postangiographic care—help to minimize complications in examination of patients with LMCAD.

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REFERENCES

1. Brown, R., Rahimtoola, S. H., Davis, G. D., and Swan, H. J. C. Effects of angiocardiographic contrast medium on circulatory dynamics in man: cardiac output during angiocardiography. *Circulation*, 1964, *31*, 234–240.
2. Bruschke, A. V. G., Proudfit, W. L., and Sones, F. M., Jr. Progress study of 590 consecutive nonsurgical cases of coronary disease followed 5–9 years. I. Arteriographic correlations. *Circulation*, 1973, *47*, 1147–1153.
3. Cohen, M. V., Cohn, P. F., Herman, M. V., and Gorlin, R. Diagnosis and prognosis of main left coronary artery obstruction. *Circulation*, 1972, Suppl. 1, *45* and *46*, 1–57–65.

4. Green, G. S., McKinnon, C. M., Rösch, J., and Judkins, M. P. Complications of selective percutaneous transfemoral coronary arteriography and their prevention: review of 445 consecutive examinations. *Circulation*, 1972, *45*, 552–557.
5. Judkins, M. P. Selective coronary arteriography. I. Percutaneous transfemoral technic. *Radiology*, 1967, *89*, 815–824.
6. Judkins, M. P. Percutaneous transfemoral selective coronary arteriography. *Radiol. Clin. North America*, 1968, *6*, 467–492.
7. Kloster, F. E., Bristow, J. D., Jacobs, W. R., Porter, G. A., and Griswold, H. E. Hemodynamic effects of angiocardiography. *Invest. Radiol.*, 1966, *1*, 398–406.
8. Lavine, P., Kimbiris, D., Sega., B. L., and Linhart, J. W. Left main coronary artery disease: clinical, arteriographic and hemodynamic appraisal. *Am. J. Cardiol.*, 1972, *30*, 791–796.
9. Porter, G. A., Kloster, F. E., and Bristow, J. D. Sequential effect of angiographic contrast agent on canine renal and systemic hemodynamics. *Am. Heart J.*, 1971, *81*, 80–92.
10. Price, J. E., Jr., and Rösch, J. Selective coronary arteriography by percutaneous left transaxillary approach using preshaped torque control catheters. *Circulation*, 1973, *48*, 1321–1323.
11. Rahimtoola, S. H., Duffy, J. P., and Swan, H. J. C. Ventricular performance after angiocardiography. *Circulation*, 1967, *35*, 70–78.
12. Rahimtoola, S. H., Gau, G. T., and Raphael, M. J. Cardiac performance after diagnostic coronary arteriography. *Circulation*, 1970, *41*, 537–544.
13. Rösch, J., Judkins, M. P., Green, G. S., and Kidd, H. Aortocoronary venous bypass grafts. *Radiology*, 1972, *102*, 567–573.
14. Sones, F. M., Jr., and Shirey, E. K. Cine coronary arteriography. *Mod. Concepts Cardiovasc. Dis.*, 1962, *31*, 735–738.

ANGIOGRAPHIC DEMONSTRATION OF THE ABSENCE OF AN ATRIAL SEPTAL DEFECT IN THE PRESENCE OF PARTIAL ANOMALOUS PULMONARY VENOUS CONNECTION*

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SHUNTS of oxygenated blood to the systemic venous circulation (left-to-right shunts) due to partial anomalous pulmonary venous connection (PAPVC) comprise 0.5 per cent of all cases of congenital heart disease[5] and are diagnosed and surgically corrected with increasing frequency.

In 1964, Schumacker and Judd[6] summarized their own experience and those of others in patients with PAPVC where the state of the atrial septum was known. They found an associated atrial septal defect (ASD) in 90 per cent of patients with PAPVC to the superior vena cava or right atrium whereas in PAPVC to the inferior vena cava, a much rarer site of drainage, only 15 per cent of patients had an ASD.

In cases of total anomalous pulmonary venous connection, an ASD along with a frequently present patent ductus arteriosus is an important avenue of mixing of oxygenated and deoxygenated blood and keeps the patient alive. In most cases of PAPVC, however, an associated ASD serves only to complicate the diagnostician's task. Surgical correction of PAPVC, ASD, or their combined presence is now possible, although the technical approach for each is somewhat different.[5] The preoperative determination whether one, the other, or both are responsible for the overcirculated lung fields is therefore of more than academic interest.

Clinical, electrocardiographic, plain film chest roentgenographic examination as well as indicator dilution and oxygen saturation data, and even conventional pulmonary angiography do not reliably establish whether an ASD is present in addition to PAPVC in such patients. The presence of PAPVC with or without an associated ASD, however, can be established roentgenographically by main pulmonary arteriography followed by selective right, or left pulmonary arteriography.

MATERIAL, METHOD AND RESULTS

In the past 5 years at the New York Hospital 22 patients with clinically suspected left-to-right shunts and subsequently demonstrated by cardiac catheterization were included in this study. When an anomalous pulmonary vein (APV) connecting either to the right atrium or one of its systemic venous tributaries was identified by main pulmonary arteriography, selective right, or left pulmonary arteriograms were obtained to rule in or out an associated ASD.

The above technique demonstrated in 8 patients with PAPVC an intact atrial septum—confirmed at surgery in 5 cases.

In all 8 of the above patients with an intact atrial septum, the connection of the APV was supradiaphragmatic.

Only 3 out of 22 patients had infradiaphragmatic PAPVC and in none of these was the atrial septum intact (Table 1).

DISCUSSION

Oxygenated blood normally drains via the pulmonary veins into the left atrium. Anomalous pulmonary veins connect directly to the right atrium or indirectly via

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TABLE I

INCIDENCE OF ASD ASSOCIATED WITH VARIOUS TYPES OF PAPVC

	Supradiaphragmatic Connection				Infradiaphragmatic Connection	
	SVC	Left SVC	Left Innominate Vein	Right Atrium	IVC	Hepatic Vein
ASD Present	6*	0	1	4	2	1*
ASD Not Present	6	1	0	2	0	0

* This patient had both right upper lobe APVC to the SVC, and right middle and lower lobe APVC to a hepatic vein.

ASD = atrial septal defect; PAPVC = partial anomalous pulmonary venous connection; SVC = superior vena cava; IVC = inferior vena cava; APVC = anomalous pulmonary venous connection.

the systemic venous circulation (superior vena cava, left superior vena cava, inferior vena cava, innominate vein, portal vein, hepatic veins or the coronary sinus), resulting in an extracardiac left-to-right shunt.

A short summary of the techniques used to diagnose PAPVC follows: although each is useful, they cannot accurately differentiate the presence of PAPVC *vs.* an ASD or their frequent coexistence. This is because of the physiologic and hemodynamic similarities present in both conditions.

A. PHYSICAL FINDINGS AND SYMPTOMS

Most patients are asymptomatic. The symptomatic may show shortness of breath, upper respiratory tract infections, cyanosis on exertion, retarded physical development and, rarely, congestive heart failure. Physical examination reveals a systolic ejection pulmonic murmur due to increased blood flow. Patients with an intact atrial septum have normal, or wide fixed splitting of the second heart sound. Those with an associated ASD have wide splitting of the second heart sound with accentuation of its pulmonic component.[4,5] Thus, symptoms and physical findings offer little differential clue.

B. ELECTROCARDIOGRAPHY

All of the findings are due to left-to-right shunting effectively at the atrial level; thus, not surprisingly, the electrocardiographic abnormalities of PAPVC and ASD are identical. They are most frequently right axis deviation, incomplete right bundle branch block, right ventricular hypertrophy and, rarely, atrial fibrillation.[4,5]

C. ROENTGENOLOGIC DIAGNOSIS

Increased and prominent pulmonary vascularity and right heart enlargement are the cardinal features of both PAPVC and ASD. In cases associated with sinus venosus ASDs the right APV may drain into and dilate the inferior ampullary portion of the superior vena cava, which produces a rounded density.[3] Even if an APV such as in the "Scimitar syndrome" were demonstrated, the presence or absence of an associated ASD could not be ruled out.

D. CARDIAC CATHETERIZATION AND ANGIOCARDIOGRAPHY

1. *Catheterization and selective injection of the APV.* If the catheter enters a pulmonary vein from the right atrium, an APV is demonstrated. Unfortunately, it is often difficult to be sure that this pulmonary vein is entered directly from the right atrium or whether it is entered from the left atrium through an ASD.[7]

2. *Oxygen saturation.* Oxygen saturation in the right atrium in excess of that in the high superior vena cava or inferior vena cava suggests the presence of a left-to-right shunt.[7] It does not, however, separate

PAPVC associated with an ASD from PAPVC without an ASD, if the anomalous vein enters directly into the right atrium or into the superior vena cava, inferior vena cava or a systemic vein very close to the right atrium. In these instances an oxygen saturation step-up at the right atrial level in addition to a step-up in the superior vena cava would still not prove the associated presence of an ASD because inferior vena cava blood usually has a higher oxygen saturation than superior vena cava blood due to higher oxygen extraction of the upper part of the body (especially cerebral circulation), as compared to that in the lower part of the body (renal circulation).

3. *Indicator dilution methods.* The appearance time and concentration of green dye* injected into the pulmonary circulation can be measured in the systemic arterial circulation. Left-to-right shunts are demonstrated by extended appearance and decreased concentration of the indicator, but the exact location of the shunt is not identified. Selective left and right pulmonary artery dye injections will give differential dye curves, if one side has an APV in the absence of an ASD,[7] similar to selective left and right pulmonary angiography.

4. *Gas inhalation.* The early appearance in the right atrium of inhaled gas will demonstrate a left-to-right shunt without identifying its location.

5. *Angiocardiography.* Injection of contrast medium into the right ventricle or pulmonary artery will first opacify the pulmonary arteries and later during the so-called "levo phase" the pulmonary veins which normally drain into and opacify the left atrium, then the left ventricle. At the time of opacification of the left atrium, no contrast medium should be seen in the right atrium. If contrast medium is detected in the left atrium and right atrium almost simultaneously, a left-to-right shunt must be present proximal to the left ventricle. This may be due to an APV, to an ASD, or a combination of both. If the APV enters the right atrium directly, or a systemic vein near the right atrium, the exclusion of an ASD is impossible by this technique.

There is, however, an angiocardiographic method to identify, or rule out an ASD in the presence of PAPVC. Assuming that unilateral anomalous pulmonary venous connection (APVC) is demonstrated, or suspected on a main pulmonary arteriogram (Fig. 1, *A* and *B*), it should be followed by selective pulmonary angiography. If necessary, injection of the pulmonary artery ipsilateral to the APV will more clearly demonstrate the APV (Fig. 3, *A* and *B*). Selective injection of the pulmonary artery contralateral to the APV will opacify the normally draining pulmonary veins which opacify the left atrium during the venous ("levo") phase. If at this time contrast medium is detected almost simultaneously in the right atrium, the presence of an ASD has been shown. On the other hand, if this injection does not opacify the right atrium, an ASD has been excluded (Fig. 2), and opacification of the right atrium following the main pulmonary artery injection was due to blood being shunted via the APV.

There are a few limitations and pitfalls to the above technique:

(a) If only a very small ASD is present, the amount of opacified blood shunted across it may not be detectable in the right atrium.

(b) Small sinus venosus ASDs may be easily missed, or are detected in an unusual manner as indicated below. These ASDs are located in the high posterior portion of the atrial septum near the entrance of the superior vena cava, and are in 86 per cent of cases accompanied by right PAPVC[2] which drains into the right atrium or into the often dilated ampullary portion of the superior vena cava in close proximity to the ASD. Thus, theoretically the normal left pulmonary veins may not contribute significant amounts of blood to the shunt[1] and therefore the ASD could be missed.

Several of our patients, however, with

* Cardio-Green, Hynson, Wescott and Dunning, Inc.

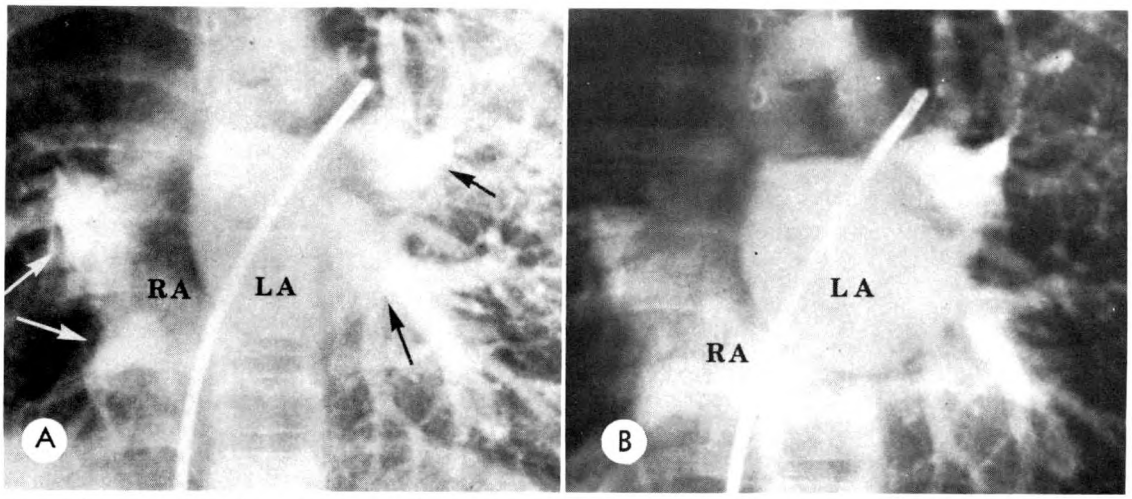


FIG. 1. Patient with right partial anomalous pulmonary venous connection and an intact atrial septum. Main pulmonary arteriograms, venous ("levo") phase.

(*A*) The normal left pulmonary veins (black arrows) connect to and opacify the left atrium (LA). The anomalous pulmonary veins on the right (white arrows) connect to the right atrium (RA). (*B*) Slightly later both atria (RA and LA) are opacified.

right PAPVC had small sinus venosus ASDs which were clearly demonstrated by a selective left pulmonary artery injection. In each of these cases, the ASD was demonstrated by reflux of contrast material into the superior vena cava through the ASD during atrial systole without appreciable opacification of the right atrium (Fig. 4, *A* and *B*). This is easily explained by the anatomic location of the sinus venosus ASD in close proximity to the superior vena cava and by the small size of the shunt. The lower portion of the right atrium is filled by unopacified blood from the inferior vena cava and thus reduces the concentration of contrast in the main body of the right atrium to below visible levels.

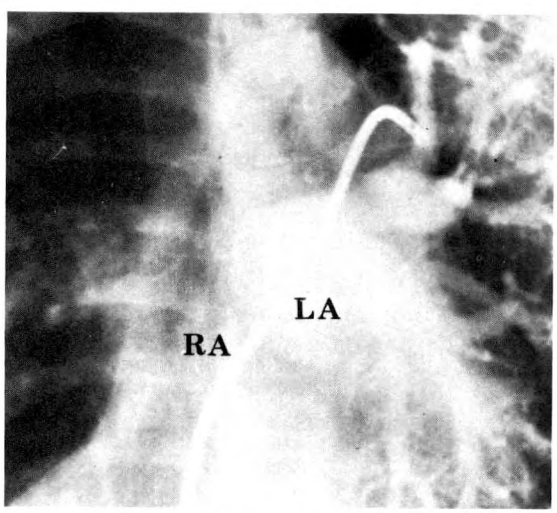


FIG. 2. Same patient as in Figure 1. Selective left pulmonary arteriogram. Venous ("levo") phase. The left pulmonary veins normally opacify the left atrium (LA); however, no contrast material is seen in the area of the right atrium (RA). This demonstrates the absence of an atrial septal defect.

(c) The angiographer must scrupulously observe good technique during selective pulmonary artery injections. Unless the catheter has been advanced well beyond the bifurcation of the main pulmonary artery, the catheter may recoil upon injection or contrast medium may reflux into the opposite pulmonary artery which results in spurious opacification of the right atrium.

Selective right, or left pulmonary angiography requires little additional time and manipulation and can be accomplished by using moderate amounts of additional contrast medium without compromising the patient's safety. It yields graphic and reliable demonstration of the nature of the anomalies, which were not available using nonselective methods.

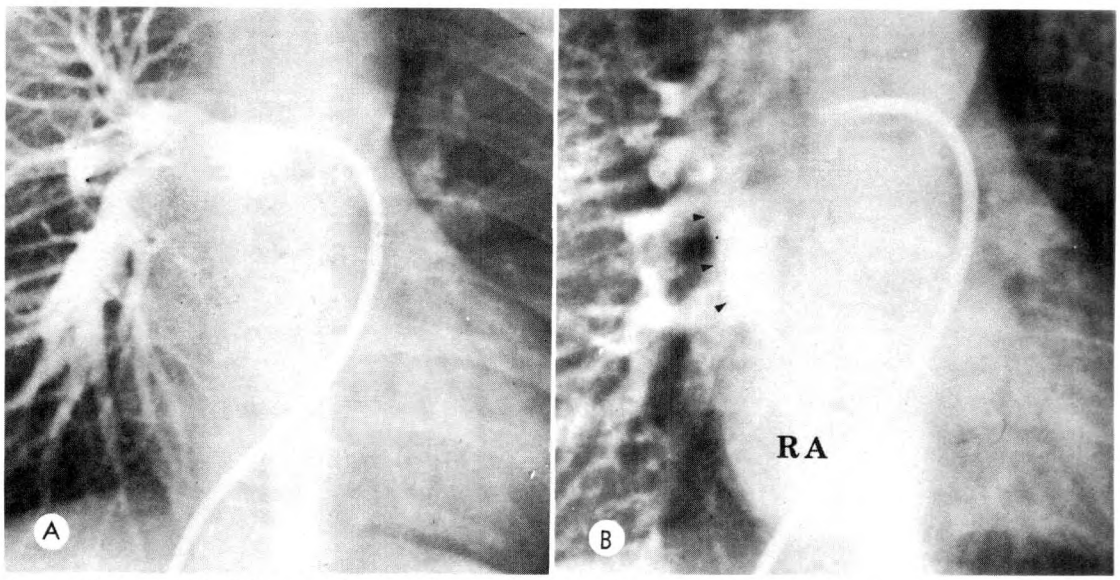


FIG. 3. Patient with right partial anomalous pulmonary venous connection and an associated small sinus venosus ASD. Selective right pulmonary arteriograms.

(*A*) Arterial phase. (*B*) The venous ("levo") phase demonstrates the anomalous right pulmonary veins connecting to and opacifying first the dilated ampulla of the superior vena cava (black arrowheads) and then the right atrium (RA).

SUMMARY

Twenty-two patients with partial anomalous pulmonary venous connection (PAPVC) were studied. In 8 patients, the absence of an associated atrial septal defect was established by a roentgenographic method not previously described.

If an anomalous pulmonary vein (APV) is identified or suspected by main pulmonary arteriography, selective injection of

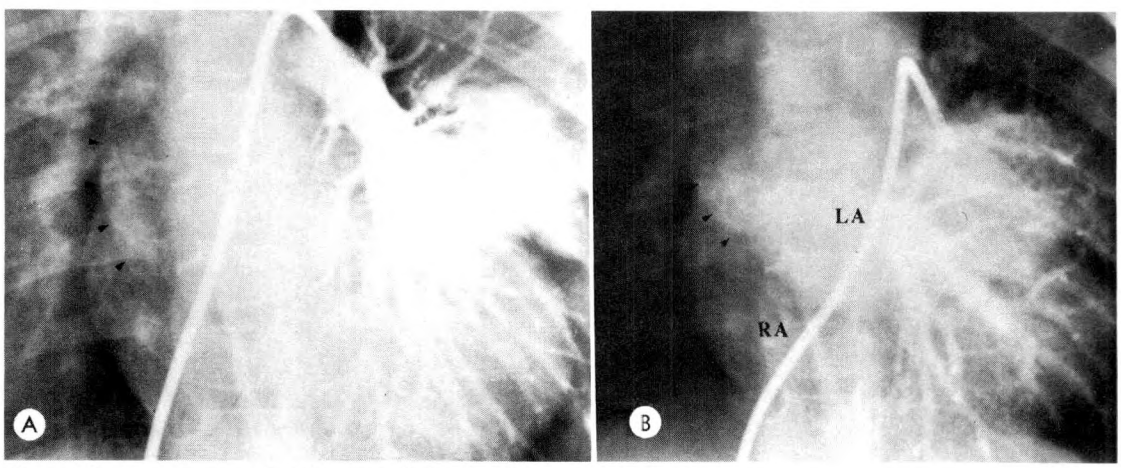


FIG. 4. Same patient as in Figure 3. Selective left pulmonary arteriograms.

(*A*) Arterial phase. Note the rounded shadow of the dilated ampullary portion of the superior vena cava (black arrowheads) which is visible on chest roentgenograms. (*B*) Venous ("levo") phase. The left atrium (LA) is opacified by the normally connecting left pulmonary veins. Note the dense opacification of the dilated ampulla of the superior vena cava (black arrowheads). This is the only indication of the presence of a small sinus venosus ASD in the absence of contrast medium in the right atrium (RA).

the ipsilateral pulmonary artery will more clearly demonstrate it, while selective injection of the pulmonary artery contralateral to the APV will establish whether or not an associated ASD is present.

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REFERENCES

1. Braunwald, E., Lombardo, C. R., and Morrow, A. G. Drainage pathways of pulmonary veins in atrial septal defect. *Brit. Heart J.*, 1960, *22*, 385–390.
2. Daria, J. E., Cheitlin, M. D., and Bedynek, J. L. Sinus venosus atrial septal defect: analysis of fifty cases. *Am. Heart J.*, 1973, *85*, 177–185.
3. Dow, J. D. Radiologic diagnosis of sinus venosus type of atrial septal defect. *Guy's Hosp. Rep.*, 1959, *108*, 305–313.
4. Hickie, J. B., Gimlette, T. M. D., and Bacon, A. P. C. Anomalous pulmonary venous drainage. *Brit. Heart J.*, 1956, *18*, 365–377.
5. Kalke, B. R., Carlson, R. G., Ferlic, R. M., Sellers, R. D., and Lillehei, C. W. Partial anomalous pulmonary venous connections. *Am. J. Cardiol.*, 1967, *20*, 91–101.
6. Schumacker, H. B., and Judd, D. Partial anomalous pulmonary venous return with reference to drainage into inferior vena cava and to intact atrial septum. *J. Cardiovasc. Surg.*, 1964, *5*, 271–278.
7. Swan, H. J. C., Burchell, H. B., and Wood, E. H. Differential diagnosis at cardiac catheterization of anomalous pulmonary venous drainage related to atrial septal defects or abnormal venous connections. *Proc. Staff Meet. Mayo Clin.*, 1953, *28*, 452–462.

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LEFT VENTRICULAR ANGIOGRAPHIC ANATOMY OF OSTIUM PRIMUM DEFECT IN THE ADULT*

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OSTIUM primum defect, a form of endocardial cushion defect also known as incomplete atrioventricular canal, is an uncommon congenital lesion in the adult patient.[1,4,6,8,10,11] The anatomic defects present include a low-lying atrial septal defect associated, in the most common form, with a cleft in the anterior leaflet of the mitral valve. Occasionally, a cleft or deformed tricuspid valve is also present. The preoperative clinical diagnosis is made on the basis of typical clinical findings of an atrial septal defect, leftward or superior axis deviation in the frontal plane of the electrocardiogram, and signs of mitral insufficiency. While these clinical findings are helpful in evaluating the patient, a definitive preoperative diagnosis requires left ventricular angiography. In the frontal projection the left ventriculogram reveals varying degrees of the typical "goosenecking" of the outflow tract of the left ventricle—a finding specific for endocardial cushion defects.[1,2] In addition, the angiogram will reveal the abnormal and cleft mitral valve. In order to better understand the origin and dynamics of these angiocardiographic findings, we performed postoperative studies on 8 adult patients with an ostium primum defect 5 to 90 months after successful surgical repair. Left ventriculography allowed assessment of the presence and degree of persistent "goosenecking," the extent to which surgery altered the appearance of the irregular cleft mitral valve, and the presence and degree of residual mitral insufficiency after repair.

MATERIAL AND METHOD

Eight adult patients who previously had undergone repair of an ostium primum defect at Stanford University Hospital were re-evaluated at from 5 to 90 months (mean 34 months), postoperatively. There were 4 females and 4 males, ranging in age at the time of surgery from 15 to 33 years, (mean 24 years). Preoperatively, 5 patients had undergone left ventriculography, 3 by retrograde arterial catheterization and 2 by venous catheterization, crossing the atrial septal defect and mitral valve into the left ventricle.

Postoperatively, all patients underwent right and left heart catheterization with left ventriculography by the retrograde arterial approach. After hemodynamic measurements, left ventriculography was carried out in either the anteroposterior (AP), the right anterior oblique (RAO), or both AP and RAO projections. In all 8 patients cine angiograms were recorded, while in 1 case biplane rapid films at 6 per second were recorded also. Where possible, the postoperative studies were performed using the same technique and projections as the preoperative studies.

Surgical repair of the congenital lesion had been performed at Stanford University Hospital under the direction of Dr. Norman Shumway. When present, the cleft mitral valve was repaired using horizontal mattress sutures with 3–0 Tevdec. The mitral valve attachments and chordae tendineae were not altered at surgery. In 1 patient with a typical ostium primum

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atrial septal defect, the mitral valve was very redundant but had no obvious cleft, and therefore no primary repair on the mitral valve was carried out. The atrial septal defect was closed in 2 patients using a Teflon patch and in the other 6 patients using a pericardial patch. In all cases, the atrial septal defect was anatomically large.

RESULTS

A. PREOPERATIVE STUDIES

The preoperative clinical evaluation included history, physical findings, chest roentgenograms and electrocardiography. Only 4 of 8 patients were asymptomatic at the time of preoperative evaluation. The chest roentgenogram in each case was compatible with a large left-to-right shunt at the atrial level (Fig. 1*A*). No chest roentgenogram showed left atrial enlargement. The electrocardiogram in each case revealed the typical leftward or superior axis deviation, and in some cases showed other findings commonly noted in these patients, including first degree A–V block, right ventricular hypertrophy and right ventricular conduction disturbances.

Preoperative cardiac catheterization revealed a pulmonary to systemic flow ratio that varied from 1.5:1 to 6:1. The pulmonary vascular resistance and pulmonary artery pressures were normal in 7 of 8 patients. The phasic left atrial or pulmonary artery wedge pressure did not reveal a significantly elevated V wave which might represent mitral insufficiency in any patient studied (Table 1).

Preoperative left ventricular angiograms were recorded in 5 patients and in each case showed the typical findings of this form of endocardial cushion defect. The position of the mitral valve was invariably abnormal, being displaced inferiorly and medially to varying degrees. During systole, the medial border of the left ventricle, which is formed in these patients by the abnormal mitral valve, was irregular and scalloped due to the thickened valve tissue. In 4 of the 5 patients, the classic radiolucent notch as described by Baron[2] was demonstrated. This is formed by the coapted and thickened margins of the cleft anterior mitral leaflet (Fig. 2*A*; and 3, *A* and *C*). In diastole, the left ventricular outflow tract of all

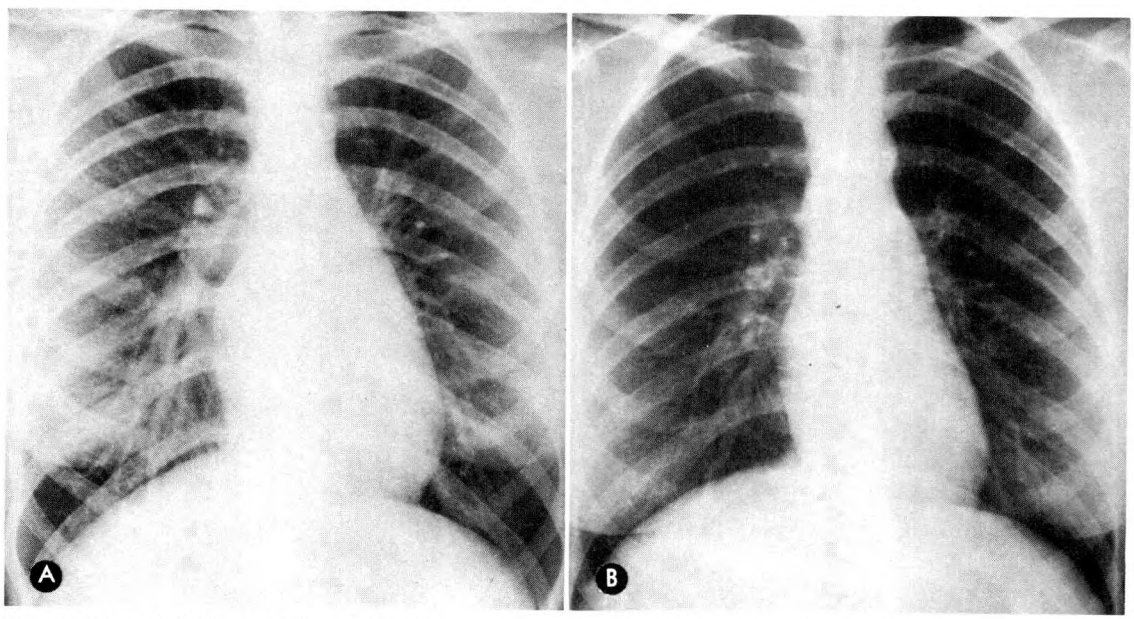


FIG. 1. Case 1. (*A*) Preoperative chest roentgenogram. Note increased pulmonary vascular markings without obvious cardiomegaly. (*B*) Same patient postoperatively. Note decrease in heart size and normal pulmonary vascular markings

TABLE I

CARDIAC CATHETERIZATION RESULTS

Case No.	Age at Surgery (yr.)		Months of Follow-up	Cardiac Catheterization Pressure (mm./Hg)			Q_S^P	C.O. (l./min.)
				PA	$\overline{PAW}$	$\overline{RA}$		
I.	15	Pre		15/4	5	3	3.1:1	3.7
		Post	30	14/6	7	4	1:1	4.2
II.	19	Pre		25/9	8	5	4.1:1	4.5
		Post	96	18/9	6	3	1:1	6.4
III.	20	Pre		20/7	7	5	2.7:1	4.5
		Post	24	12/4	3	2	1:1	5.1
IV.	24	Pre		28/8	6	4	6:1	2.8
		Post	64	16/5	4	2	1:1	4.0
V.	25	Pre		78/33	4	4	1.7:1	3.9
		Post	5	45/22	5	4	1:1	4.2
VI.	26	Pre		19/5	6	5	5.4:1	3.3
		Post	42	18/7	8	2	1:1	5.1
VII.	24	Pre		24/12	3	2	2.8:1	4.1
		Post	8	17/6	7	1	1:1	6.4
VIII.	33	Pre		25/8	6	1	1.5:1	5.5
		Post	5	18/5	8	1	1:1	6.4

PA = Pulmonary artery pressure, systolic/diastolic; $\overline{PAW}$ = Pulmonary artery wedge mean pressure; $\overline{RA}$ = Right atrial mean pressure; Q_S^P = Ratio of pulmonary to systemic blood flow; C.O. = Systemic cardiac output.

5 patients showed varying degrees of narrowing, representing the morphologic picture of the "gooseneck" deformity (Fig. 2*B*; and 3, *B* and *D*). The left ventriculogram also permitted preoperative estimations of mitral valve competence. Two LV angiograms, done by means of venous catheterization crossing the ASD and mitral valve, showed mild mitral insufficiency. In 2 of the 3 patients who had retrograde left ventriculography, mitral insufficiency was demonstrated also (Table II).

B. POSTOPERATIVE FINDINGS

Postoperatively historical review, physical findings, chest roentgenograms and electrocardiograms were obtained. Seven patients were asymptomatic, while 1 patient still complained of mild palpitations. The chest roentgenogram in each case showed a normal peripheral pulmonary vasculature. The large main pulmonary arteries present in each of the 8 patients preoperatively had decreased somewhat in size but were still prominent (Fig. 1*B*).

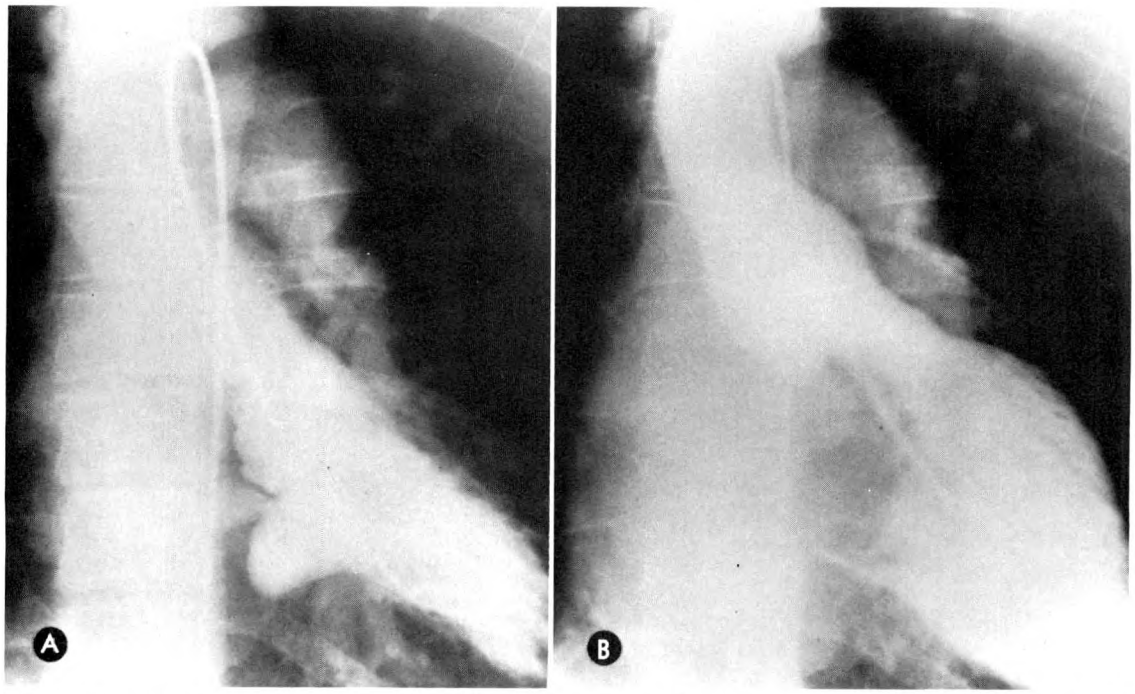


FIG. 2. Case VIII. Preoperative left ventricular angiograms in the anteroposterior projection. The arterial catheter has been passed retrograde across the aortic valve into the ventricle. (*A*) During systole the mitral valve is seen in tangent due to its abnormal position and forms the right border of the left ventricle. The irregular scalloping of this border is due to the redundant, thickened nature of the mitral anterior leaflet. Note the radiolucent notch, representing the cleft, through which a jet of contrast material is seen regurgitating. (*B*) During diastole there is "goosenecking" of the left ventricular outflow tract, occurring as the anterior mitral valve swings open.

Heart size decreased in patients even in cases where it was not grossly enlarged preoperatively. The electrocardiogram continued to show the leftward or superior axis deviation.

Postoperative cardiac catheterization showed the shunt to be closed in every case (Table I). Pulmonary artery pressures were slightly lower in 4 of 7 patients in whom the pressure was normal preoperatively, and had decreased markedly in 1 patient who had moderately severe pulmonary hypertension. The pulmonary artery wedge phasic pressure did not suggest mitral insufficiency in any case. However, left ventricular angiography demonstrated mitral insufficiency in 3 of the 8 postoperative patients (Table II). The postoperative anatomy of the left ventricular outflow tract was only minimally changed by surgery. The systolic abnormalities were still present in all 8 patients. These included the irregular medial border of the left ventricle and the frequent radiolucent notch representing the cleft in the mitral valve (Fig. 4*A*). The typical "goosenecking" with narrowing of the left ventricular outflow tract during diastole also persisted

FIG. 3. Case V. Preoperative left ventricular angiograms in another patient in the anteroposterior projection. (*A*) During systole the radiolucent notch, representing the cleft, is obvious. (*B*) During diastole there is marked mitral regurgitation and massive left-to-right shunting through the ostium primum septal defect which partially obscures the "gooseneck" deformity of the left ventricular outflow tract. (*C* and *D*) Diagrammatic representations of *A* and *B*, respectively. Striped area represents regurgitated contrast medium shunting to the right side of the circulation.

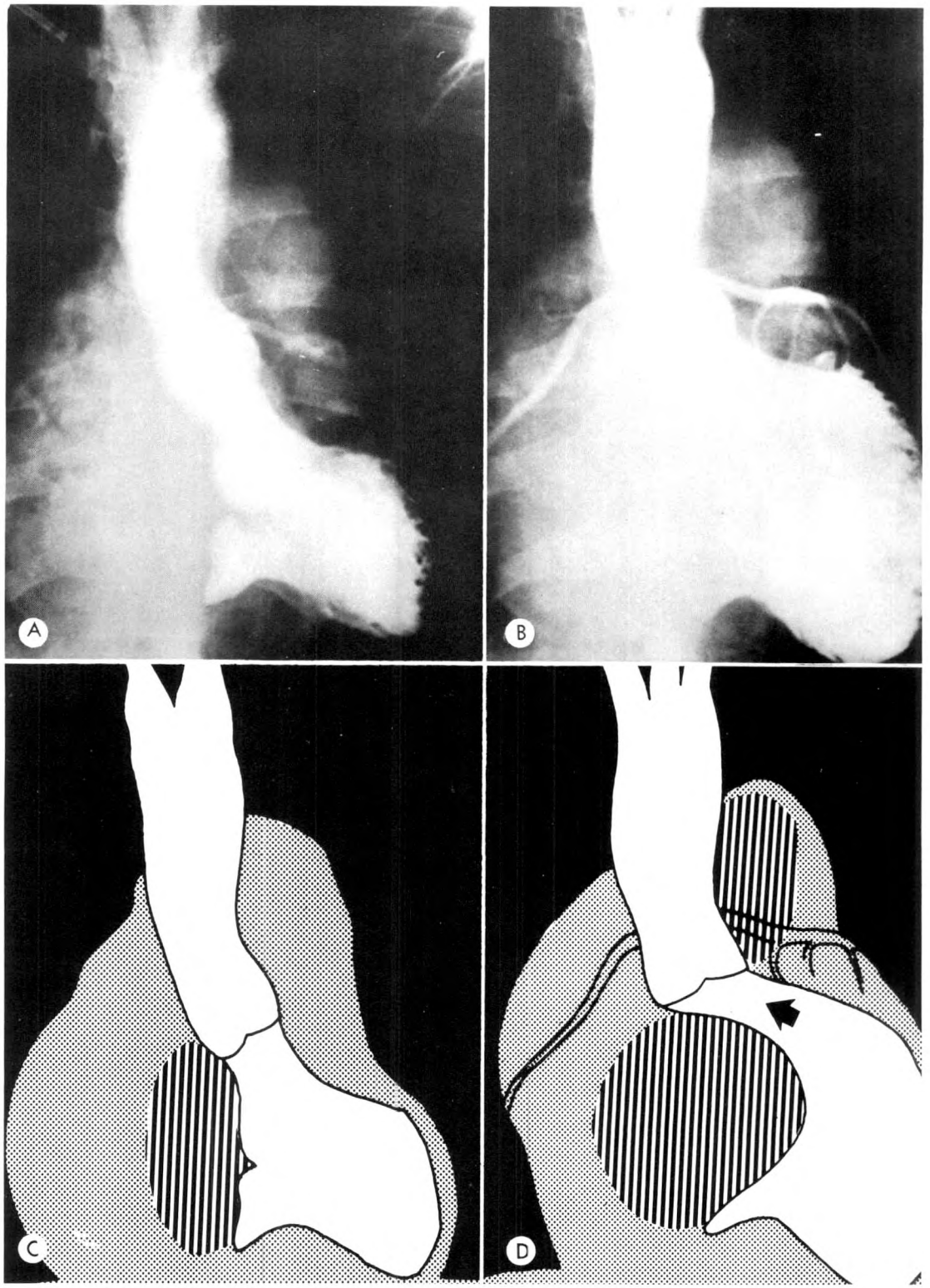

A
B
C
D

TABLE II

SUMMARY OF ANGIOGRAPHIC FINDINGS: LEFT VENTRICULOGRAMS

Case No.		Catheter Approach	Projection	Cleft Mitral Valve	Mitral Regurgitation	Appearance of LV Outflow Tract
I.	Pre	Venous	AP+Lat	+	Trace	GD—moderate
	Post	Arterial	RAO	+	Trace	GD—unchanged
II.	Pre	Venous[1]	—	—	—	—
	Post	Arterial	AP, RAO	+	o	GD—marked
III.	Pre	Venous[1]				
	Post	Arterial	AP, RAO	+	Trace	GD—mild
IV.	Pre	Venous[1]				
	Post	Arterial	AP, RAO	+	o	GD—moderate
V.	Pre	Arterial	AP+Lat LAO, RAO	+	4+	GD—moderate
	Post	Arterial	AP+Lat RAO	+	2+	GD—slightly less
VI.[3]	Pre	Venous	Multiple[3]			
	Post	Arterial	RAO	+	o	GD—mild
VII.	Pre	Arterial	RAO	o	o	GD—moderate
	Post	Arterial	RAO	o	o	GD—unchanged
VIII.	Pre	Arterial	AP+Lat RAO+LAO	+	2+	GD—moderate
	Post	Arterial	AP, RAO	+	o	GD—slightly less

1 = No angiography performed; 2 = Cardiac malposition; 3 = Unable to determine anatomy; GD = "Gooseneck" deformity.
Mild
Moderate: degrees of left ventricular outflow tract narrowing.
Marked
Note: Case VII—Mitral valve thickened and irregular but no cleft.

in each patient, but occasionally appeared to be somewhat less striking compared to the preoperative study (Fig. 4*B*; and Table II).

DISCUSSION

The anatomic findings in preoperative patients with endocardial cushion defect as demonstrated by the left ventriculogram, were reviewed by Baron *et al.*[1] During ventricular systole, the right border of the left ventricle is formed by the abnormal cleft anterior mitral leaflet. This margin appears irregular and scalloped because of the multiple abnormal chordae incorporated into it. The nonopaque notch frequently seen marks the location of the cleft. It is produced when the thickened, free edges of the cleft leaflet coapt. When the edges do not coapt completely, regurgitation of contrast material into the left atrium is seen at this point. In diastole, the left ventricular outflow tract is narrowed and more horizontal than is normal. This appearance also is due to the displacement, inferiorly and

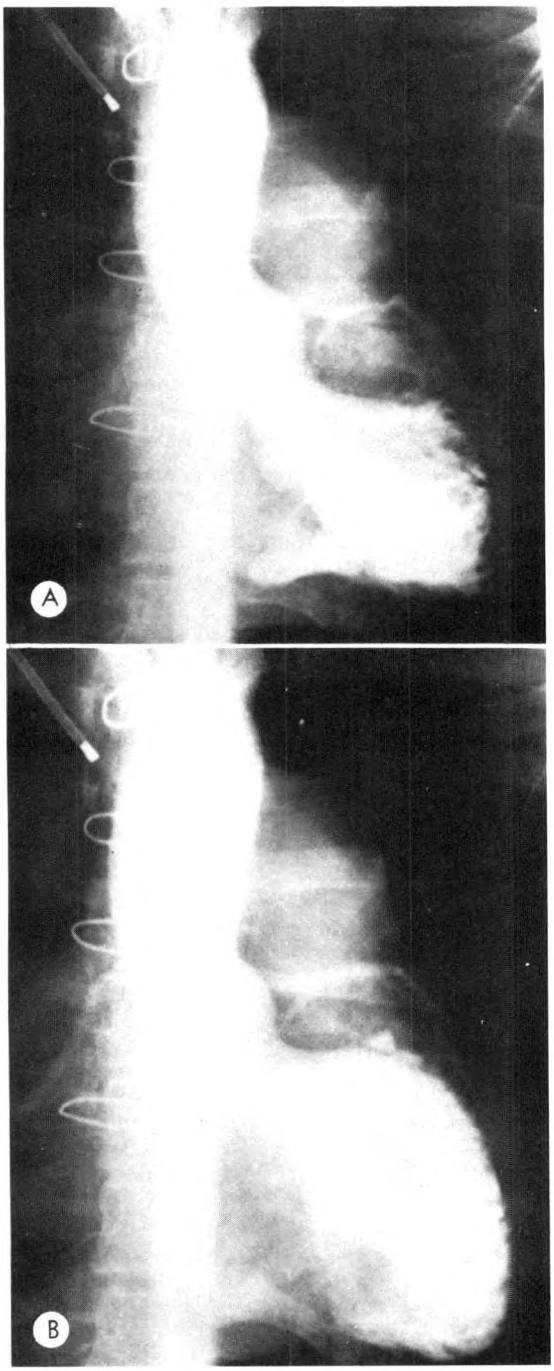


Fig. 4. Case v. Postoperative left ventricular angiograms in the anteroposterior projection (same patient as in Figure 3, *A–D*). (*A*) During systole persistence of abnormal position of mitral valve with its irregular, scalloped margin and cleft. (*B*) During diastole the "gooseneck" deformity of left ventricular outflow tract persists and is better seen because there is less mitral regurgitation and no left-to-right shunting.

medially, of the mitral valve and has been called the "gooseneck deformity" of the left ventricular outflow tract.

These same findings were demonstrated in our 5 adult patients with a form of endocardial cushion defect (*i.e.*, ostium primum defect) who had preoperative ventriculography performed. We were interested in the effect, if any, of surgery on these anatomic findings. Surgical repair of the large cleft in the anterior leaflet of the mitral valve might be expected to result in abolition of the mitral regurgitation and change the appearance of the abnormal anterior leaflet on left ventriculography. Postoperative angiocardiograms have been obtained in previously reported cases of the ostium primum form of endocardial cushion defect with varied results. Rastelli *et al.*[9] stated that the characteristic appearance of the left ventricle, including the nonopaque notch seen in systole, persisted after surgical repair of the cleft mitral leaflet in 2 cases of the partial form of atrioventricular canal in which left ventriculography was performed postoperatively. They did not mention any change or persistence of any diastolic abnormalities in these 2 cases. Griffiths *et al.*[6] performed postoperative left ventricular angiography in 8 patients with ostium primum defect. Each patient showed persistence of the characteristic preoperative deformity of the medial border of the left ventricular outflow tract seen in systole. However, they felt that the left ventriculograms frequently showed a change in the contour and amount of narrowing of the outflow tract as seen in diastole. They stated that the "gooseneck" tended to be less narrowed and in some cases the region returned to a near normal appearance.

The persistence of the "gooseneck" would not be surprising when one considers its anatomic basis. Blieden *et al.*[3] presented 2 cases of the complete form of endocardial cushion defect where this deformity, as demonstrated by left ventriculography, persisted after surgical repair in which the ventricular septum was built up with syn-

thetic material and the natural mitral valve was replaced by a prosthesis. This group of investigators undertook anatomic studies of specimens with endocardial cushion defect. Their findings agreed with those of Goor *et al.*[5] and indicated that the diaphragmatic (inflow) aspect of the left ventricle and the posterior ventricular septum are short relative to the outflow length of the ventricular septum. Also, there is a concave deficiency of the subaortic aspect of the septum. In diastole the mitral mechanism swings into the left ventricular outflow tract when the mitral valve opens. The anterior mitral leaflet tends to lie against the superior aspect of the ventricular septum and serves to outline the extent and shape of the septum and of the left ventricular cavity. This gives the appearance of varying degrees of subaortic narrowing of the outflow track—commonly known as the "gooseneck" deformity. Persistence of the "gooseneck" following surgical correction of the deficiency of the ventricular septum appears to be related to the short nature of the inflow part of the left ventricle at its diaphragmatic aspect, since this part of the heart is not altered by the surgical procedures described.

In the partial type of endocardial cushion defect (*i.e.*, ostium primum defect) the same deficiencies of the inflow length and ventricular septum as in the complete form are seen but usually to a lesser extent. There is no ventricular communication, but the abnormal anterior mitral leaflet still will outline the deficient ventricular septum in diastole.

The left ventricular angiogram in all 8 of our patients demonstrated persistence of the irregular medial border in systole due to the nature of the anterior mitral leaflet. In the 7 patients who had a cleft anterior mitral leaflet, the radiolucent notch representing the cleft was still demonstrated. In diastole, the appearance of the "gooseneck" deformity also persisted, but occasionally to a lesser degree when compared to preoperative studies. Repair of the cleft in the mitral valve may have caused some restriction of the movement of the anterior leaflet into the outflow tract thus causing the "neck" to appear slightly wider in some cases. Postoperative changes in the appearance of the left ventricle during systole or diastole are therefore minimal.

Our study is comparable to that presented by Griffiths *et al.*[6] except that their patients were younger with a mean age of 10 years. Only 1 of their 8 patients showed postoperative mitral insufficiency on biplane (AP and lateral) serial rapid films (6/sec.). In 3 of our 8 patients, single plane cineangiography in the RAO projection demonstrated mild degrees of mitral regurgitation. This difference in filming technique may account for the higher detection of mitral regurgitation in our series.

SUMMARY

Comparison of the left ventricular angiographic anatomy pre- and postoperatively has demonstrated that the surgical repair of both the atrial septal defect and cleft mitral valve in adult patients with an ostium primum defect causes very little change in the characteristic angiographic picture of the left ventricle, the left ventricular outflow tract or the mitral valve.

Of 8 patients studied pre- and postoperatively, all had complete closure of the defect. Postoperatively 3 had persistent mild mitral insufficiency. The radiolucent notch representing the cleft in the anterior leaflet was seen in 6 of 8 cases postoperatively despite suture repair.

The medial border of the left ventricle in systole, which is formed by the displaced anterior leaflet of the mitral valve, was markedly irregular due to the abnormal leaflet in all 8 cases. During diastole the left ventricular outflow tract had a similar appearance in both pre- and postoperative angiograms, with persistence of the typical "gooseneck" deformity.

Left ventricular angiography by retrograde arterial catheterization utilizing cineangiography is the optimal technique for

anatomic confirmation of the ostium primum defect and for assessing the presence and severity of mitral regurgitation.

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REFERENCES

1. Baron, M. G., Wolf, B. S., Steinfeld, L., and Van Mierop., L. H. S. Endocardial cushion defects: specific diagnosis by angiocardiography. *Am. J. Cardiol.*, 1964, *13*, 162–175.
2. Baron, M. G. Abnormalities of mitral valve in endocardial cushion defects. *Circulation*, 1972, *45*, 672–680.
3. Blieden, L. C., Randall, P. A., Castaneda, A. R., Lucas, R. V., Jr., and Edwards, J. E. "Gooseneck" of endocardial cushion defect: anatomic basis. *Chest*, 1974, *65*, 13–17.
4. Braunwald, N. S., and Morrow, A. G. Incomplete persistent atrioventricular canal: operative methods and results of pre- and postoperative hemodynamic assessments. *J. Thoracic & Cardiovasc. Surg.*, 1966, *51*, 71–80.
5. Goor, D., Lillehei, C. W., and Edwards, J. E. Further observations on pathology of atrioventricular canal malformation. *A.M.A. Arch. Surg.*, 1968, *97*, 954–962.
6. Griffiths, S. P., Ellis, K., Burris, J. O., Bumenthal., S., Bowman, F. O., Jr., and Malm, J. R. Postoperative evaluation of mitral valve function in ostium primum defect with cleft mitral valve: partial form of atrioventricular canal. *Circulation*, 1969, *40*, 21–29.
7. Lillehei, C. W., Anderson, R. C., Ferlic, R. M., and Bonnabeau, R. C., Jr. Persistent common atrioventricular canal: recatheterization results in 37 patients following intracardiac repair. *J. Thoracic & Cardiovasc. Surg.*, 1969, *57*, 83–94.
8. Rastelli, G. C., Weidman, W. H., and Kirklin, J. W. Surgical repair of partial form of persistent common atrioventricular canal with special reference to problem of mitral valve incompetence. *Circulation*, 1965, Suppl. II, *31*, *32*, 31–35.
9. Rastelli, G. C., Kirklin, J. W., and Kincaid, O. W. Angiocardiography of persistent common atrioventricular canal. *Mayo Clin. Staff Proc.*, 1967, *42*, 200–209.
10. Scott, L. P., Hauek, A. J., Nadas, A. S., and Gross, R. E. Endocardial cushion defect: preoperative and postoperative surgery. *Circulation*, 1962, *26*, 218–227.
11. Weyn, A. S., Bartle, S. H., Nolan, T. B., and Dammann, J. F., Jr. Arterial septal defect, primum type. *Circulation*, 1965, Suppl. II, *31*, *32*, 13–23.

THE IMPORTANCE OF SELECTIVE ANGIOGRAPHY OF SYSTEMIC ARTERIAL SUPPLY TO THE LUNGS IN PLANNING SURGICAL CORRECTION OF PSEUDOTRUNCUS ARTERIOSUS*

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THE term "pseudotruncus arteriosus" has been applied to the severe form of tetralogy of Fallot in which atresia of the pulmonic valve and varying lengths of the main pulmonary trunk are present.[3-5] The associated intracardiac abnormalities include ventricular septal defect and a stenotic or atretic infundibular portion of the right ventricle. In the commonest form, right and left pulmonary arteries are present and join together in a confluence at the distal end of the atretic main pulmonary artery segment. Blood passes from the aorta to the pulmonary circulation by way of systemic arteries which arise from the aortic arch, brachiocephalic arteries or descending thoracic aorta and anastomose with the pulmonary arterial tree near the hilum of the lung.[7] It is often not clear whether these systemic arteries represent a patent ductus arteriosus (PDA), bronchial arteries or remnants of the primitive intersegmental arteries.[9] Pseudotruncus arteriosus is diagrammatically represented in Figure 1.

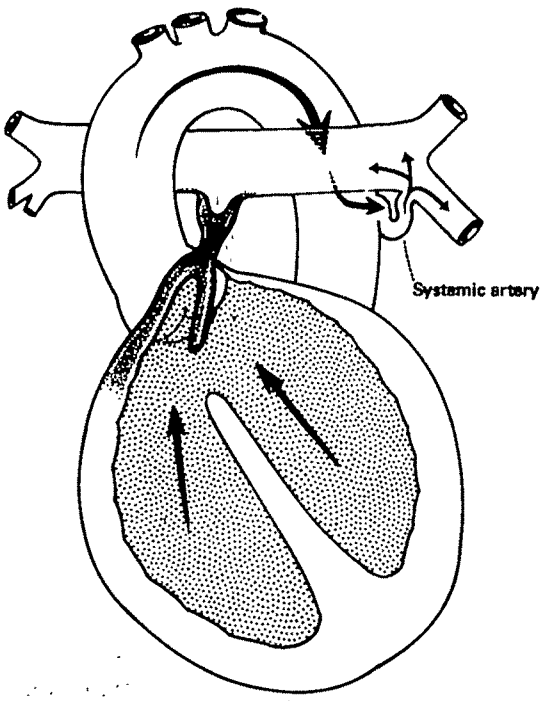


FIG. 1. Diagram of the blood flow pattern in pseudotruncus arteriosus. The ventricular portion of the heart is shown with a septal defect, along with an atretic pulmonic valve and main pulmonary artery. The systemic-pulmonary connection is depicted here as a single vessel arising from the descending thoracic aorta. In actuality, there may be several connecting vessels arising from the arch, descending aorta or the brachiocephalic arteries.

Type IV truncus arteriosus is a related malformation characterized by complete absence of pulmonary arteries due to failure of development of the sixth aortic arches.[2,4,12] Blood passes from the aorta to the lungs directly by way of one or more enlarged systemic arteries.

The difference between pseudotruncus and Type IV truncus arteriosus is therefore relatively clear-cut from a theoretic viewpoint, pulmonary arteries being present in the former and absent in the latter. The 2 malformations are quite similar, however, from clinical and electrocardiographic view-

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points[1,4,12] and numerous authors have had difficulty distinguishing them by angiocardiographic methods.[4,6,8-10] In earlier years, this distinction was purely of academic interest since both were considered inoperable lesions. Recently, however, effective techniques have been devised for surgical correction of pseudotruncus and certain cases of Type IV truncus arteriosus.[3,11,13] Decisions as to the feasibility of surgery and the exact type of operation to be performed rely to a large extent upon precise angiographic demonstration of pulmonary vascular anatomy.

SURGICAL CONSIDERATIONS

The basic principles of surgical repair of pseudotruncus arteriosus have been described by McGoon *et al.*,[11] and Doty *et al.*[3] The systemic-pulmonary communicating vessels are divided at their origins. The ventricular septal defect is closed in such a way as to direct left ventricular blood into the aorta. Finally, direct communication is established between the right ventricle and the post-atretic confluence of the pulmonary arteries by means of an aortic homograft[11] or a valved conduit.[3] Venous blood is thus directed from the right ventricle through the aortic homograft or conduit to the pulmonary arteries and then through the lungs. After returning to the heart, oxygenated blood passes out the aorta. The procedure restores an entirely normal flow pattern and has so far proven effective for this previously inoperable lesion. The outlook is most favorable when the systemic-pulmonary communications contain narrowed segments which protect the lungs against systemic arterial pressure prior to surgery.

A similar but somewhat riskier type of repair can be carried out in certain cases of Type IV truncus arteriosus in which each lung is supplied entirely by 1 or 2 large systemic arteries, preferably containing proximal segmental stenoses. These vessels are divided and then reimplanted into the distal end of the homograft or conduit extending from the right ventricle.[11,13] In those patients with Type IV truncus arteriosus having multiple smaller bronchial or systemic arteries instead of 1 or 2 large arteries, surgical correction is not possible at this time.

ANGIOGRAPHIC CONSIDERATIONS

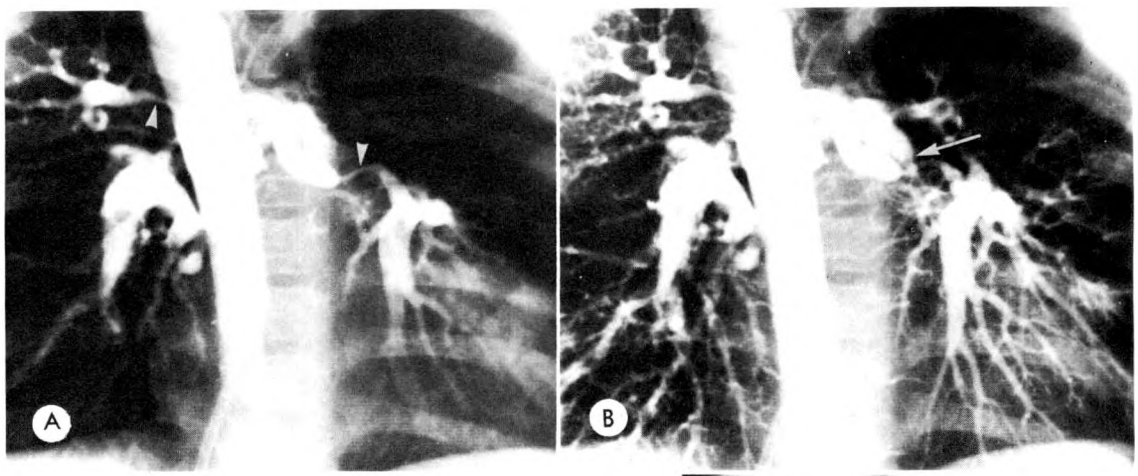
Right ventricular angiocardiography in pseudotruncus arteriosus demonstrates the ventricular septal defect, a large overriding aorta, and the absence of antegrade filling of a main pulmonary artery. The narrowed infundibular portion of the right ventricle ending blindly at the atretic pulmonic valve may be seen. Injection of contrast medium into the thoracic aorta sometimes, but not always, shows that pulmonary arteries are present. The enlarged aorta receives the entire output of both sides of the heart and the flow rate is very rapid. Adequate aortic opacification is thus difficult to achieve. Adequate opacification of the pulmonary arteries is even less likely to occur, since they receive blood indirectly through the systemic arteries. It is for this reason that difficulties are often encountered in evaluating the pulmonary circulation in patients with this type of malformation.

Even if the aortogram succeeds in demonstrating the presence of pulmonary arteries, it is unlikely to reveal the detailed anatomic information necessary for planning operative repair. The surgeon must know, for example, whether a central confluence of the pulmonary arteries exists, into which the homograft or conduit can be inserted. The adequacy of development of the more distal pulmonary arterial tree must also be determined. In some cases, the pulmonary arteries may be hypoplastic and therefore incapable of transporting the entire output of the right heart to the lungs.[1] Other patients have confluent pulmonary arteries which receive blood from a large systemic artery, but then supply only *parts* of both lungs. The remaining lung tissue is perfused by other bronchial or systemic arteries which arise directly from the aorta and which are apparently

not connected to the pulmonary circulation. Cases of this sort seem to exhibit features of both pseudotruncus and Type IV truncus arteriosus malformations. The homograft or conduit operation may be inadvisable if the pulmonary arteries perfuse only small portions of the lungs.

Because it is necessary to map the pulmonary artery anatomy so precisely, we have recently been performing selective angiography of the systemic arteries supplying the lungs in cases in which a diagnosis of pseudotruncus has been entertained. This approach was initially described by Chesler *et al.*[1] Scrutiny of the thoracic aortogram should indicate the location of these systemic arteries. The largest ones are catheterized selectively utilizing precurved polyethylene or woven dacron catheters. Volume and flow rate of injected contrast material are chosen according to the size of the patient, the catheter, and the vessel being injected. No individual injection exceeds 1 cc. of contrast material per kilogram of body weight. Rapid serial roentgenograms are simultaneously exposed in the frontal and lateral projections. The rapid injection of a relatively large bolus of contrast material directly into the systemic arteries will result in retrograde filling of the pulmonary arteries, if any are present, and determination of their degree of development.

The accompanying illustrations clearly indicate the importance of using this approach in evaluating patients with pseudotruncus arteriosus. Figure 2, *A–C*, is a study performed 6 years ago before the



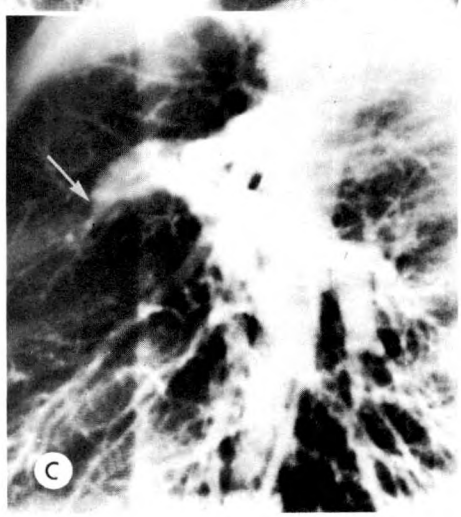


FIG. 2. (*A*) Frontal projection of a descending thoracic aortogram in a patient with pseudotruncus arteriosus. There appear to be at least 3 and possibly more systemic arteries supplying peripheral vessels within the lungs which have the configuration of pulmonary arteries. Note the stenoses within the systemic-pulmonary communications (white arrowheads). (*B* and *C*) Simultaneous frontal and lateral roentgenograms later in the sequence. Retrograde filling of right and left pulmonary arteries has occurred *via* the systemic arteries. The pulmonary arteries join together in a central confluence (white arrows) distal to the atretic segment. These central pulmonary arteries appear somewhat hypoplastic and it is not possible to determine whether they connect with all the peripheral segments of both lungs or only some of them.

present technique was adopted. The diagnosis of pseudotruncus was established, but since surgical repair was not yet available, additional information was not required. There appear to be at least 3 large systemic arteries arising from the descending aorta and perfusing the lungs. A confluence of right and left pulmonary arteries can be seen, having filled in retrograde fashion via the systemic vessels. The pulmonary arteries are rather small, however, and it is questionable whether they connect with all the peripheral segments of both lungs. By current standards, this represents an inadequate study, since it does not establish whether the patient is a favorable candidate for surgery.

Figure 3, *A–J*, is a study of a patient with pseudotruncus who also has 3 large systemic arteries arising from the descending aorta. Individual selective angiograms show that one perfuses most of the right upper, middle and lower lobes, but does not appear to connect with a central pulmonary artery. Another perfuses the anterior and lingular segments of the left upper lobe and the left lower lobe. This vessel also fails to connect with a pulmonary artery. The third appears to directly fill a confluence of right and left pulmonary arteries which in turn perfuse the right middle and right lower lobes and the apical-posterior segment of the left upper lobe. The initial thoracic aortogram had demonstrated the presence of the confluence of right and left pulmonary arteries and could have erroneously suggested that the patient was therefore an optimal surgical candidate. However, the selective systemic arteriograms showed that, although pulmonary arteries are present, they perfuse only parts of both lungs. While surgery is feasible in such a case, the anatomic arrangement is not ideal. Because of persistent limitation of activity and cyanosis, a homograft operation was performed and the patient has done quite well during the ensuing 6 months. Postoperative right ventriculography shows that the graft is functioning normally, but that, as predicted, it only perfuses the right middle and right lower lobes and the apical-posterior segment of the left upper lobe.

Figure 4, *A* and *B*, is a study of a patient with pseudotruncus in whom selective angiography of a very large systemic artery fills a confluence of right and left pulmonary

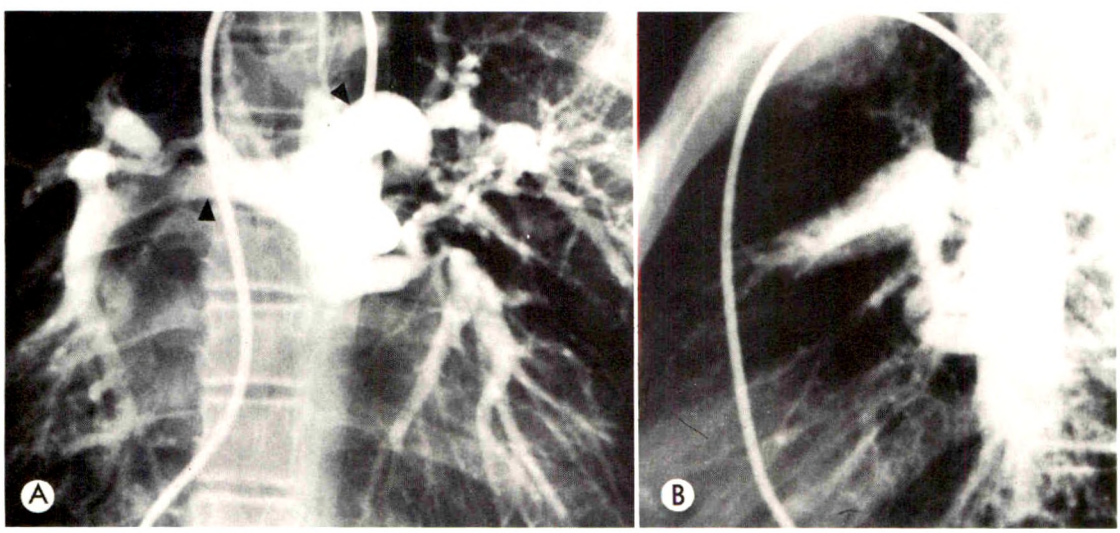


Fig. 3. (*A* and *B*) Simultaneous frontal and lateral views of a descending thoracic aortogram in a second patient with pseudotruncus arteriosus. Large systemic arteries supply peripheral vessels within the lungs having the configuration of pulmonary arteries. There is retrograde filling of what appear to be right and left pulmonary arteries (black arrowheads). It is not clear whether these central pulmonary arteries are connected with all peripheral segments of both lungs.

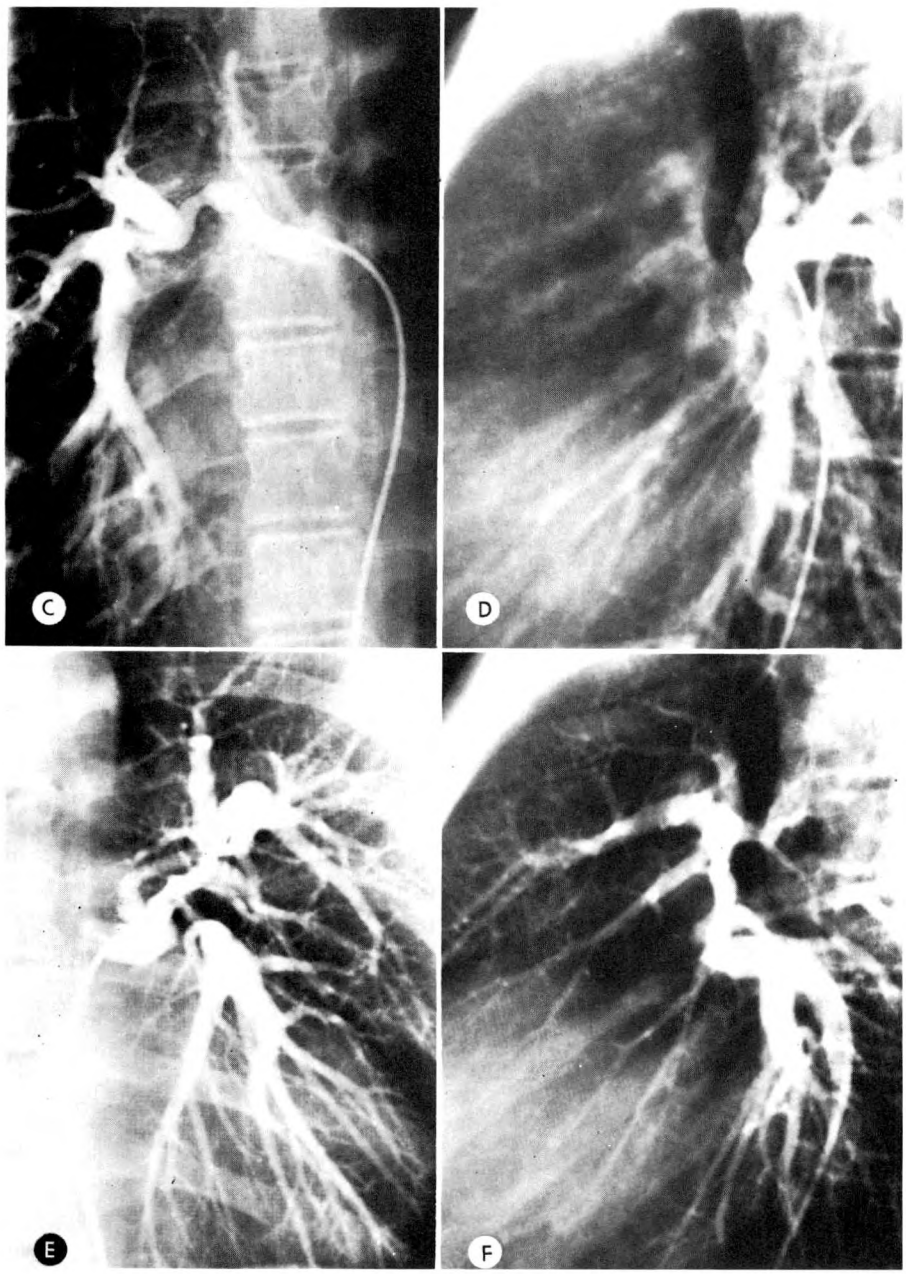


FIG. 3. (*C* and *D*) Selective angiography of one systemic artery supplying most of the right upper, middle and lower lobes. There is no apparent connection with the right pulmonary artery. (*E* and *F*) Selective angiography of a second systemic artery supplying the left lower lobe and the lingular and anterior segments of the left upper lobe, but not its apical-posterior segment. There is no apparent connection with the left pulmonary artery.

arteries. These in turn perfuse the entire right lung and most of the left lung. A separate small bronchial artery perfuses the lingular region. Since almost all pulmonary tissue is supplied by the pulmonary arterial tree, this patient would be a better surgical candidate than the patient shown in Figure 3, *A–J*. The operation had not yet been performed at the time this paper was written.

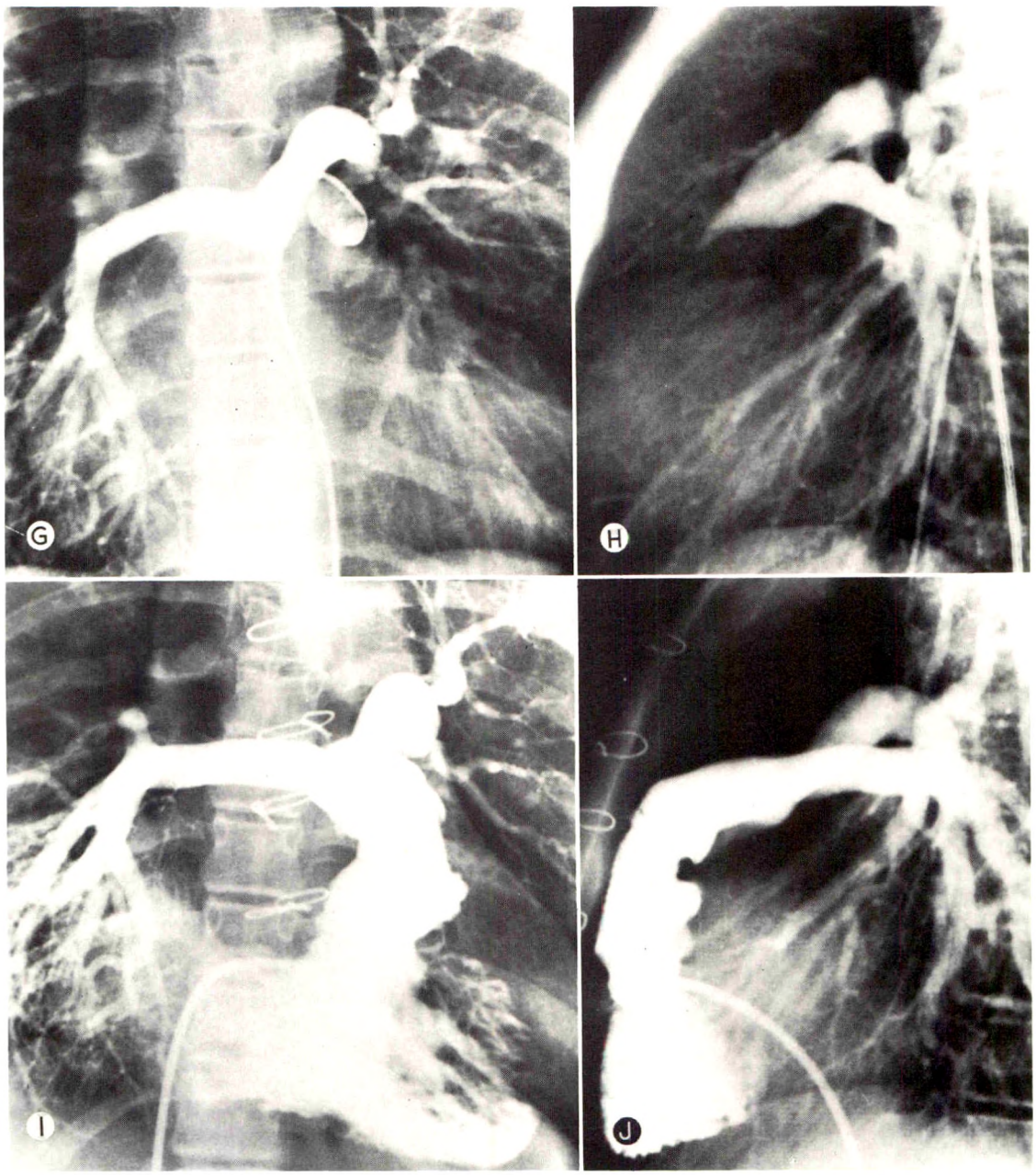


FIG. 3. (*G* and *H*) Selective angiography of a third systemic artery fills the left and right pulmonary arteries directly. Their confluence at the distal end of the atretic segment is clearly seen. The left pulmonary artery supplies the apical-posterior segment of the left upper lobe and the right pulmonary artery supplies the right middle and right lower lobes. Note that the right middle and right lower lobes also were supplied by the first systemic artery. Although well-developed confluent right and left pulmonary arteries are therefore demonstrated, they directly perfuse only approximately half of the lung tissue. (*I* and *J*) Postoperative right ventriculography. An aortic homograft connects the right ventricle and the central confluence of the right and left pulmonary arteries. The graft is functioning well, but flow passes only to those segments which were perfused directly by right and left pulmonary arteries on the preoperative study.

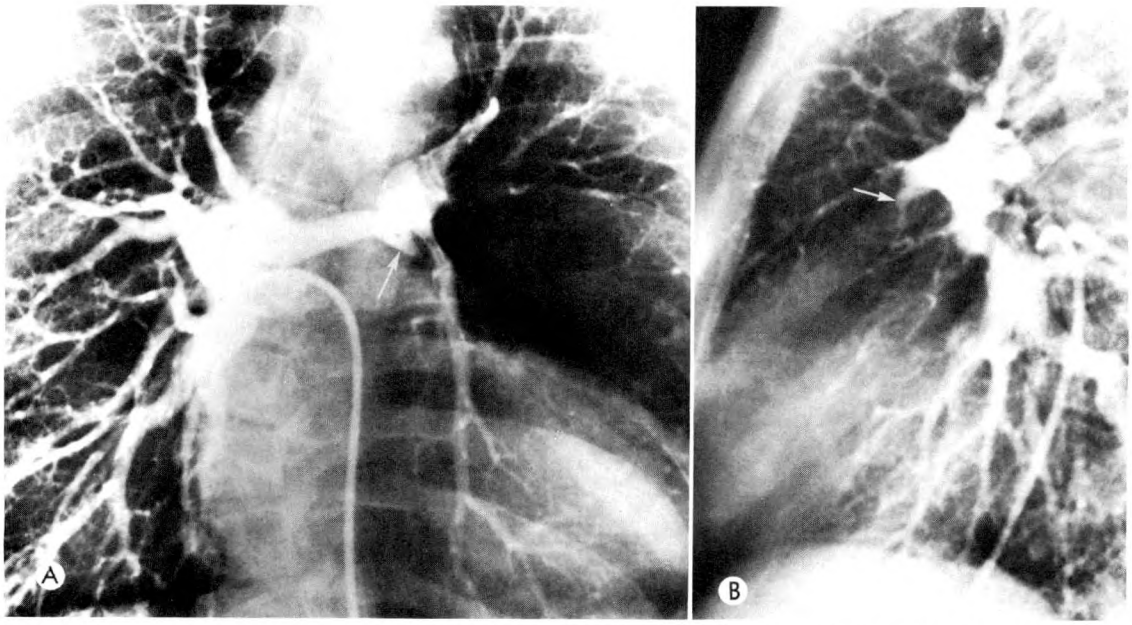


FIG. 4. (*A* and *B*) Selective angiography of a large systemic artery arising from the right side of the descending thoracic aorta in a third patient with pseudotruncus arteriosus. The right and left pulmonary arteries and their confluence (white arrows) fill rapidly in retrograde fashion. Note that the right pulmonary artery is in direct continuity with all peripheral segments of the right lung and that the left pulmonary artery is in direct continuity with most segments of the left lung. Filling of the left lower lobe arteries is somewhat delayed due to the retrograde filling pattern of the central pulmonary vessels. A thoracic aortogram (not shown) suggested that the lingular area was supplied by a separate systemic artery originating from the descending thoracic aorta.

The patient shown in Figure 5 has a relatively normal left pulmonary artery which receives blood through a systemic vessel resembling a PDA. No pulmonary arteries were visualized on the right. The right lung appeared to be supplied entirely by small bronchial arteries. A graft connecting the right ventricle with the left pulmonary artery would be feasible, but there is no way of restoring a physiologic flow pattern on the right.

In addition to the 4 cases discussed above, our experience has included 6 earlier cases with similar malformations. All 6 were studied by angiocardiography and thoracic aortography, but none had selective angiography. In 3 cases it was not possible to definitely conclude whether the lesion was pseudotruncus or Type IV truncus arteriosus. The other 3 were felt to have pseudotruncus arteriosus, but in each

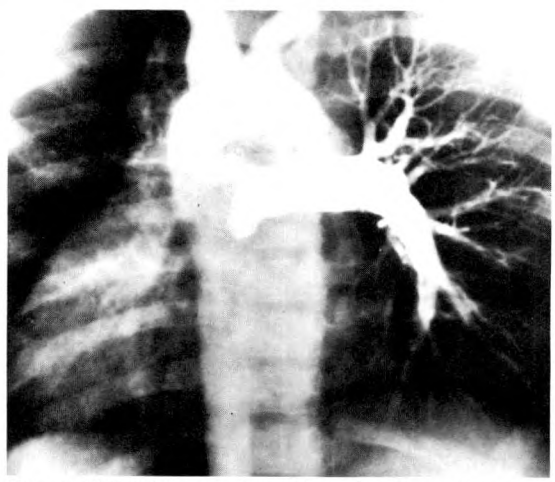


FIG. 5. Selective arteriography of a systemic artery resembling a patent ductus arteriosus in a patient with pulmonary atresia and dextrocardia. A normal left pulmonary artery is visualized, but the entire right pulmonary arterial tree is absent. Thoracic aortography (not shown) indicated that the right lung was supplied by numerous small bronchial arteries.

instance the degree of definition of pulmonary artery anatomy was suboptimal.

SUMMARY

Selective angiography of large systemic arteries which appear to supply blood to the lungs should be performed in patients with suspected pseudotruncus arteriosus (tetralogy of Fallot with pulmonary atresia) in an attempt to accurately define the post-atretic pulmonary artery anatomy.

If pulmonary arteries are in fact present, this technique greatly enhances the possibility of visualizing them, thereby establishing the diagnosis of pseudotruncus arteriosus, while ruling out Type IV truncus arteriosus. It will also show whether a confluence of right and left pulmonary arteries exists, whether the peripheral pulmonary branches are adequately developed, and whether they supply blood to all or only parts of both lungs.

Surgical correction of this congenital lesion is now considered advisable in many cases and this information is of great importance in proper planning of the operative procedure.

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REFERENCES

1. Chesler, E., Beck, W., and Schrire, V. Selective catheterization of pulmonary or bronchial arteries in preoperative assessment of pseudotruncus arteriosus and truncus arteriosus: Type IV. *Am. J. Cardiol.*, 1970, *26*, 20–24.
2. Collett, R. W., and Edwards, J. E. Persistent truncus arteriosus: classification according to anatomic types. *S. Clin. North America*, 1949, *29*, 1245–1270.
3. Doty, D. B., Kouchoukos, N. T., Kirklin, J. W., Barcia, A., and Bargeron, L. M. Surgery for pseudotruncus arteriosus with pulmonary blood flow originating from upper descending thoracic aorta. *Circulation*, 1972, Suppl. 1, *46*, 121–129.
4. Edwards, J. E., Carey, L. S., Neufeld, H. N., and Lester, R. G. Congenital Heart Disease: Correlation of Pathologic Anatomy and Angiocardiography. Volume II. W. B. Saunders Company, Philadelphia, 1965.
5. Edwards, J. E., and McGoon, D. C. Absence of anatomic origin from heart of pulmonary arterial supply. *Circulation*, 1973, *47*, 393–398.
6. Garcia, R., Cargill, J. W., and Drake, E. H. Pseudotruncus arteriosus: report of oldest surviving patient. *Am. Heart J.*, 1969, *78*, 537–540.
7. Guntheroth, W. G., Arcasoy, M. M., Phillips, L. A., and Figley, M. M. Demonstration of collateral circulation to lungs with angiocardiographic studies in congenital heart disease. *Am. Heart J.*, 1962, *64*, 293–300.
8. Hallermann, F. J., Kincaid, O. W., Tsakiris, A. G., Ritter, D. G., and Titus, J. L. Persistent truncus arteriosus: radiographic and angiocardiographic study. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 827–834.
9. Jefferson, K., Rees, S., and Somerville, J. Systemic arterial supply to lungs in pulmonary atresia and its relation to pulmonary artery development. *Brit. Heart J.*, 1972, *34*, 418–427.
10. Lafargue, R. T., Vogel, J. H. K., Pryor, R., and Blount, S. G. Pseudotruncus arteriosus: review of 21 cases with observations on oldest reported case. *Am. J. Cardiol.*, 1967, *19*, 239–246.
11. McGoon, D. C., Rastelli, G. C., and Wallace, R. B. Discontinuity between right ventricle and pulmonary artery: surgical treatment. *Ann. Surg.*, 1970, *172*, 680–689.
12. Stuckey, D., Bowdler, J. D., and Reye, R. D. K. Absent sixth aortic arch: form of pulmonary atresia. *Brit. Heart J.*, 1968, *30*, 258–264.
13. Wallace, R. B., Rastelli, G. C., Ongley, P. A., Titus, J. L., and McGoon, D. C. Complete repair of truncus arteriosus defects. *J. Thoracic & Cardiovasc. Surg.*, 1969, *57*, 95–105.

BRONCHOCUTANEOUS FISTULA AS A LATE COMPLICATION OF PERMANENT EPICARDIAL PACING*

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SURGICAL implantation of a permanent pacemaker to stimulate the heart is a well-established procedure of great benefit. Once a pacemaker has been installed, the physician's attention is directed not only to the patient's illness but also to the unique complications of permanent pacing. Since the implanted pacemaker system represents a large foreign body, infection is not a rare complication.

The following case illustrates a bronchocutaneous fistula as a late complication of permanent epicardial pacing.

REPORT OF A CASE

M.J., a 62 year old mild diabetic male, was readmitted to the University of Virginia Hospital in January 1973 for replacement of a pulse generator and evaluation of a chronic draining sinus in the left upper abdominal wall.

The patient had been seen initially in 1960 for evaluation of syncope. He subsequently developed complete heart block, and in 1965 a Medtronic Model 5870 pacemaker was inserted. The epicardial leads were implanted transthoracically, and the generator was placed in the left anterior abdominal wall.

In August 1967, the patient received prophylactic antibiotics and the power pack was replaced under local anesthesia. The patient's postoperative course was uneventful. He did well until April 1968 when a suture abscess developed in the upper portion of the battery pouch. The patient received systemic antibiotics without improvement. Consequently, the suture abscess was excised, and the upper portion of the generator pouch was revised on April 15, 1968. An abscess with a persistent draining fistula developed in the generator pouch and a partial herniation of the pulse generator ensued.

On August 5, 1968, a skin flap was rotated to cover the generator. Despite irrigation with hydrogen peroxide and antibiotic solutions, the fistula continued to drain purulent material. Cultures revealed growths of *Staphylococcus aureus* and *Pseudomonas aeruginosa*.

In September 1968, a transvenous pacemaker was inserted, and the generator was implanted in the right anterior chest wall. The epicardial pacemaker was removed, and the fistulous tract excised. However, the transthoracic leads were divided and left in place, anchored to the rectus fascia. The abdominal pouch was drained and irrigated. The wound closed, but the draining fistula reappeared and persisted despite local irrigations and systemic antibiotics.

The patient remained in satisfactory condition in spite of the draining fistula until the winter of 1972 when he developed a chronic productive cough, anorexia, weakness and a 10 pound weight loss. The patient was readmitted to change the transvenous generator and to evaluate the fistula.

Physical examination revealed a fistula in the left upper abdominal wall which drained purulent material. The old epicardial leads protruded from the sinus tract when the patient hyperextended his spine.

A roentgenogram of the chest revealed an infiltrate in the lingula (Fig. 1). A sinogram was obtained by injecting the fistulous tract. The fistula was found to communicate with the pericardium and the lingular division of the left upper lobe bronchus (Fig. 2; and 3).

The bronchocutaneous fistula was confirmed at thoracotomy. The fistula was ligated, and the old epicardial leads were removed.

DISCUSSION

The placement of foreign material inside the body carries with it a certain risk of infection. Schowengerdt and Masangkay[9]

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reviewed the literature from 1965 to 1970 on transvenous pacing and found the rate of infection to be 5.2 per cent. The rate of infection in epicardial pacing has been variously reported from 0 per cent[12] to 42 per cent.[3] Harris *et al.*[7] found a significantly lower incidence of sepsis and rejection when using the endocardial system as compared to the epicardial system. However, a study by Gadboys *et al.*[6] of 190 patients indicates a similar rate of infection for the 2 types of pacemakers. In their report, 91 patients received an epicardial pacemaker resulting in 2 cases of infection, and 99 patients received a transvenous pacemaker resulting in 3 cases of infection. Firor *et al.*[4] reported 48 cases of pacemaker implantation with epicardial leads and 48 cases with transvenous leads. There were 2 infections following thoracotomy and 5 infections associated with transvenous insertion. Thus, infection is not an uncommon complication of permanent pacing.

When the infection is localized, it presents as pain over the generator site. The pain tends to disappear when a sinus develops, draining the infected pocket. If the infection is not controlled, the generator may be extruded. The infection may travel along the wires to the myocardium resulting in an increase in the stimulation threshold,[3] and septicemia may develop. Endo-

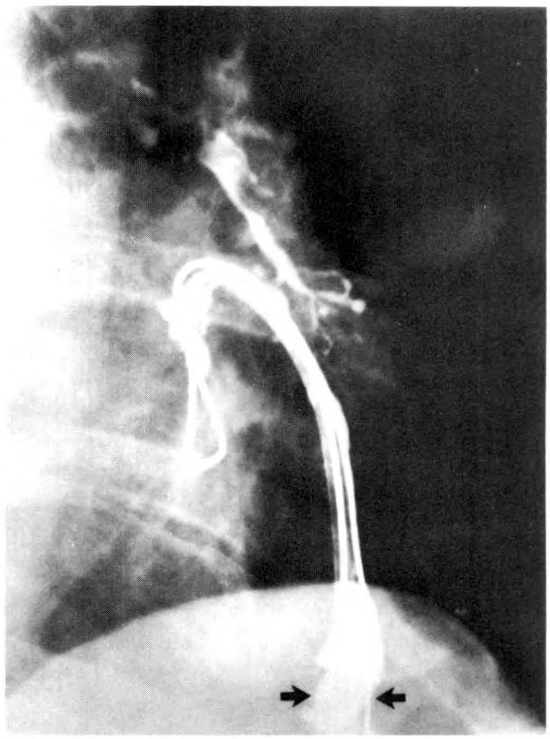


FIG. 2. A sinogram obtained by injecting contrast material into the cutaneous fistula (arrows). The connection between the fistulous tract which extends along the epicardial leads, the pericardium and the lingular bronchus, is demonstrated.

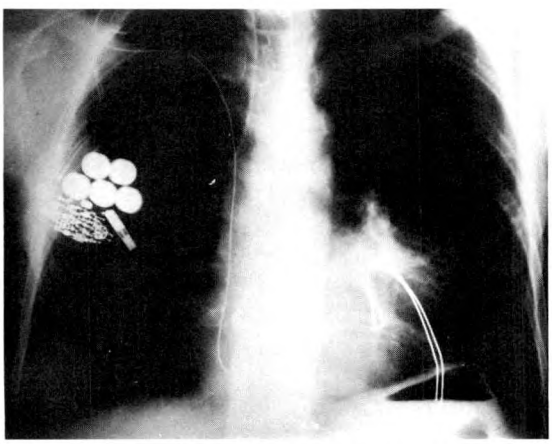


FIG. 1. Posteroanterior chest roentgenogram reveals an infiltrate in the lingula. The old epicardial leads remain in the left thorax. The functioning transvenous pacemaker is seen in good position.

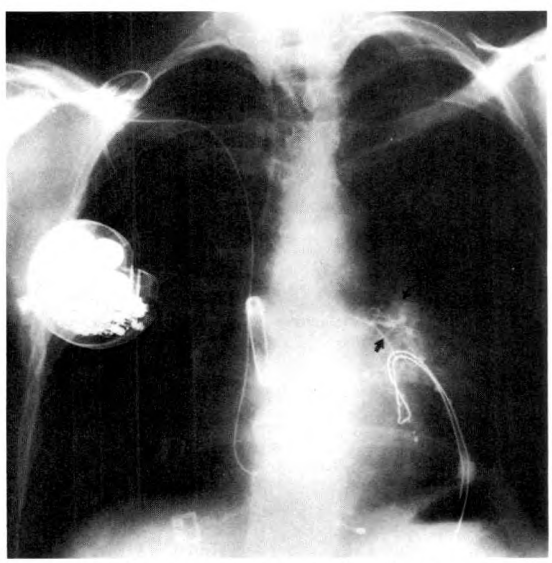


FIG. 3. A follow-up posteroanterior chest roentgenogram demonstrates the bronchocutaneous fistula and the lingular infiltrate (arrow).

carditis has been reported.[7] Septic pulmonary emboli have also occurred.[11,13] Death may result from these complications.

The guiding principle in the management of infected pacemakers has been the removal of all foreign material including the generator and the electrodes.[3,4,7,8,10] A new pacemaker is then implanted at a different site. More recently, however, local debridement and irrigation with antibiotic solutions have been advocated in the treatment of infected pulse generators.[2,5]

The patient in the present report was treated by irrigation of the pouch and when this failed the generator was removed. However, the leads were left in place, and the infection traveled along the leads to the heart and to the adherent lung resulting in a bronchocutaneous fistula.

This case suggests that the management of infected pulse generators with local debridement and irrigation should be cautiously re-evaluated.

SUMMARY

Although local debridement and irrigation may control some pacemaker infections, the present case illustrates the need for careful monitoring of infected pacemakers. The value of the sinogram in the evaluation of infected pacemakers is demonstrated.

If the infection persists despite vigorous therapy, a sinogram should be obtained to ascertain the extent of the fistulous tract. The infected pacemaker is not an ordinary wound infection since the pacing wires lead inward to the heart.

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REFERENCES

1. BEREGOVICH, J., and FENIG, S. Complications with permanent transvenous pacemakers. *New York J. Med.*, 1970, *70*, 761–766.
2. DARGAN, E. L., and NORMAN, J. C. Conservative management of infected pacemaker pulse generator sites. *Ann. Thoracic Surg.*, 1971, *12*, 297–299.
3. DAVIES, J. G., and SIDDONS, H. Experience with implanted pacemakers: technical considerations. *Thorax*, 1965, *20*, 128–134.
4. FIROR, W. B., LOPEZ, J. F., NANSON, E. M., and MORI, M. Clinical management of infected pacemaker. *Ann. Thoracic Surg.*, 1968, *6*, 431–436.
5. FURMAN, R. W., HILLER, A. J., PLAYFORTH, R. H., BRYANT, L. R., and TRINKLE, J. K. Infected permanent cardiac pacemaker. *Ann. Thoracic Surg.*, 1972, *14*, 54–58.
6. GADBOYS, H. L., LUKBAN, S., and LITWAK, R. S. Long-term follow-up of patients with cardiac pacemakers. *J. Cardiol.*, 1968, *21*, 55–59.
7. HARRIS, A., BLUESTONE, R., BUSBY, E., DAVIES, G., LEATHAM, A., SIDDONS, H., and SOWTON, E. Management of heart block. *Brit. Heart J.*, 1965, *27*, 469–482.
8. RAINER, W. G., NEWBY, J. P., FEILER, E. M., and MANIATIS, W. Management of implantable pacemaker complications. *Rocky Mountain M. J.*, 1966, *63*, 38–43.
9. SCHOWENGERDT, C. G., and MASANGKAY, M. P. Implantation of cardiac pacemakers. *Ohio M. J.*, 1972, *68*, 1015–1018.
10. SCHWARTZ, E. L., CRASTNOPOL, P., and HAMBY, R. I. Catheter extrusion with infection complicating permanent endocardial pacing. *Dis. Chest*, 1968, *54*, 28–31.
11. SCHWARTZ, I. S., and PERVEZ, N. Bacterial endocarditis associated with permanent transvenous cardiac pacemaker. *J.A.M.A.*, 1971, *218*, 736–737.
12. SENNING, A. Pacemaker: problems in use of pacemakers. *J. Cardiovasc. Surg.*, 1964, *5*, 651–656.
13. WAISSER, E., KUO, C-S., and KABINS, S. A. Septic pulmonary emboli arising from permanent transvenous cardiac pacemaker. *Chest*, 1972, *61*, 503–505.

ROENTGENOGRAPHIC EVALUATION OF DISSECTING ANEURYSMS OF THE AORTA*

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ACUTE dissecting aneurysms of the aorta carry a very high mortality during the first 48 hours after onset. The diagnosis must be made and therapy instituted promptly if the mortality rate is to be lowered significantly. Although the clinical history and physical findings may be very suggestive of dissecting aneurysms, "clear-cut" cases may still be misdiagnosed.[13] The definitive diagnosis, therefore, must be made by angiography. The angiography must be performed as an emergency procedure as soon as the patient's condition allows.

The proponents of antihypertensive drug therapy claim that 80 per cent of the patients with dissecting aneurysms should be treated medically. Surgical therapy is reserved for those cases which: (1) cannot be controlled with drugs; (2) suggest impending rupture; (3) have massive aortic valve insufficiency; (4) have occlusion of major aortic branches.[12] Some surgeons feel that all cases with the dissection beginning in the ascending aorta should be treated by surgery.[2] It is therefore imperative in the drug treated patients to angiographically establish the diagnosis, and in the surgical patients, outline the exact anatomy of the dissection to determine the surgical approach and type of procedure which should be performed.

PATHOLOGY

Most authors believe that medial degeneration of the aortic wall is the basic lesion predisposing to a dissecting aneurysm.[6] Arteriosclerosis may be present because of the age of most of these patients, but is more likely a coincidental finding than a significant contributing factor in the dissection. The distribution of dissecting aneurysms supports this; *i.e.*, dissecting aneurysms are most common in the ascending aorta where arteriosclerosis is uncommon, and dissections rarely begin in the lower abdominal aorta where arteriosclerosis is very common.

For a more detailed review of the pathogenesis, the exhaustive review by Hirst *et al.*[6] should be consulted. Briefly, their review concludes that arteriosclerosis is an intimal disease which may cause atrophic and hyaline changes in the media, but not the cystic type of medionecrosis which predisposes to dissection. Only 10 of 74 patients with dissecting aneurysms under the age of 40 had more than minimal arteriosclerosis. On the other hand, in a series of 212 cases of dissecting aneurysms of the aorta, cystic medial necrosis was present in 62.5 per cent. Miscellaneous forms of medial degeneration including hyaline changes, elastic and muscle tissue loss, scarring of the media, and nonspecific changes were seen in an additional 17.3 per cent. Arteriosclerosis occurring alone was seen in only 1.4 per cent, and the media was histologically normal in 16 per cent.

The consensus of opinion regarding the initial event in dissection is rupture of the vasa vasorum because the degenerated media fails to provide sufficient support for these vessels. An intimal tear subsequently develops, exerting pulsatile arterial pressure on the medial hematoma, resulting in a longitudinal cleavage of the aortic media. The so-called dissecting aneurysm is really a dissecting hematoma separating layers of degenerated aortic media. The hypertension present in most of these patients accelerates the dissection, and it is the reduc-

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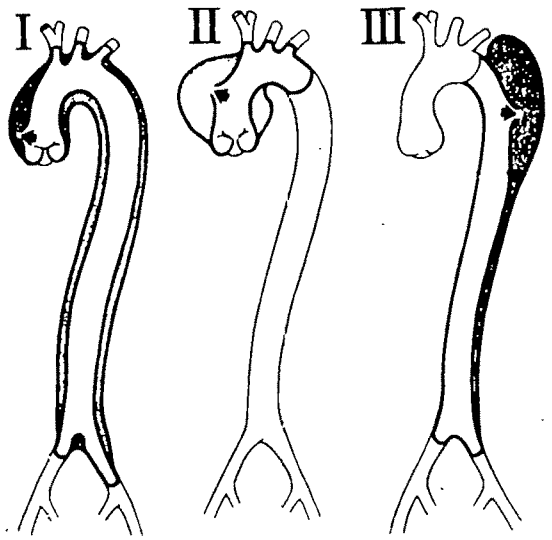


FIG. 1. Classification of dissecting aneurysms (DeBakey's classification).[3]

Type I begins in ascending aorta and extends distal to the aortic arch.

Type II begins in and is confined to the ascending aorta.

Type III begins at or just distal to the left subclavian artery and extends distally from that point.

tion of this driving force which is the goal of antihypertensive medication.[12,13]

CLASSIFICATION

From a diagnostic, prognostic, and therapeutic point of view, DeBakey's classification is the most useful one (Fig. 1).[3] He classified Type I as those which begin just above the aortic valve cusps and extend to the descending thoracic aorta, and frequently to the abdominal and iliac vessels. Type II begins in the ascending aorta and stops at the level of the brachiocephalic vessels, and may dissect into one or more of these vessels. Type III begins at or just distal to the origin of the left subclavian artery and dissects a variable distance down the descending aorta. Daily *et al.*[2] believe that all patients who have dissections involving the ascending aorta should be operated upon. Accordingly, they classify dissecting aneurysms into only 2 groups, Type A, being those involving the ascending aorta (DeBakey's Type I+II), and Type B, which begin at the left subclavian artery (DeBakey's Type III).

Approximately 60 per cent of dissecting aneurysms begin in the ascending aorta, consisting of 46 per cent Type I and 14 per cent Type II. Approximately 35 per cent are Type III, and the remaining 5 per cent begin in the descending thoracic and abdominal aorta.

The most common cause of death is rupture of the dissection into the pericardium, and this occurs in those patients where the dissection begins in the ascending aorta. Therefore, many patients with Type I and II dissections die in the first few hours after onset of the dissection, and are never studied. At aortography, therefore, a greater percentage of patients can be expected to have Type III lesions.[8]

MATERIAL

Table I indicates the material used in compiling this report, and the distribution of patients presented in this table agrees well with those previously reported. Our series, therefore, is a representative sample of all patients with dissecting aneurysms.

ROENTGENOGRAPHIC FINDINGS

CHEST ROENTGENOGRAPHY

Most patients will have suspicious chest roentgenograms. However, a negative chest

TABLE I

CASE MATERIAL

Total No. Cases	49	
	No. of Patients	Per Cent
Sex: Male	37	76
Female	12	24
Type I	24	49
II	9	18
III	16	33
Cystic Medial Necrosis (39 patients with tissue examination)	25	64
Hypertension	34	69
Age Range 11–80 years		
Average Age 55.3 years		

roentgenogram does not rule out a dissecting aneurysm. The most common finding is widening of the superior mediastinum and aorta found in approximately 70 per cent of patients.[5,6] This was the most common plain roentgenographic sign in our series. Other findings include enlargement and haziness of the aortic knob, tracheal displacement to the right and enlargement of the descending aorta (Fig. 2). The tracheal shift may be a very helpful clue to the diagnosis, since frequently the initial roentgenograms are taken with a portable unit, and the heart and mediastinum are both magnified. The trachea normally should remain in the midline, however. Since hypertensive patients often have aortic dilatation, comparison with previous chest roentgenograms is very helpful. The demonstration of progressive widening of the mediastinum is strong evidence of a dissecting aneurysm. Another helpful sign is a distance of greater than 1 cm. between the calcified intima and the outer margin of the aorta. In Type II lesions, disproportionate enlargement of the ascending aorta relative to the arch may be seen. An associated pleural effusion, most commonly on the left, is an ominous sign and indicates that the aneurysm is leaking. Cardiomegaly, although frequently present, is not a helpful sign unless it occurs acutely and indicates aortic insufficiency.

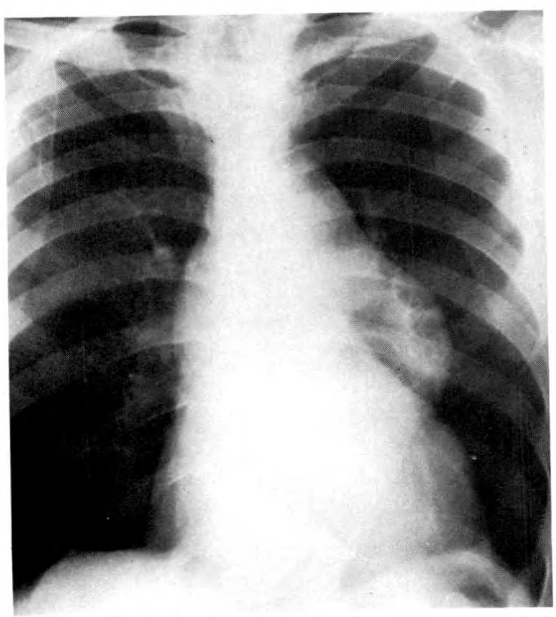


Fig. 2. Upright posteroanterior chest roentgenogram. The descending aorta is abnormally large with a relatively normal ascending aorta and arch. This was a Type III dissection.

ANGIOGRAPHY

Angiography in dissecting aneurysms, in our opinion, must be performed by direct aortography. The visualization of the aorta after venous injection is insufficient in many cases to define the anatomy, particularly in patients with a low cardiac output, congestive failure, and aortic insufficiency. The very important diagnosis of aortic insufficiency cannot be made by a venous injection, and the presence of occlusions of major branch vessels is frequently not seen.

Aortography can be performed by a percutaneous approach from either the axillary or femoral arteries. The right axillary approach may be complicated by involvement of the innominate artery by the dissection, and also has the disadvantage that it is more difficult to do injections into the descending thoracic and abdominal aorta to identify the double channel and appraise the status of major visceral branches. Usually, however, these structures are well seen by injection into the aortic root, and many angiographers routinely use the axillary (or brachial) approach.[5]

The femoral approach is equally satisfactory, but the retrograde advancement of the guidewire and catheter may be more difficult. The percutaneous puncture may enter into the false channel and the catheter may lie entirely within the false channel. Even with a puncture of the true channel, the catheter may enter the false channel at any point through a re-entry intimal tear. The advantage of the femoral approach, however, is that many angiographers are more familiar with this approach, and it also allows injections at any level in the aorta, to better delineate the anatomy.[7]

In practice, our initial approach is determined by the status of the pulses. Oc-

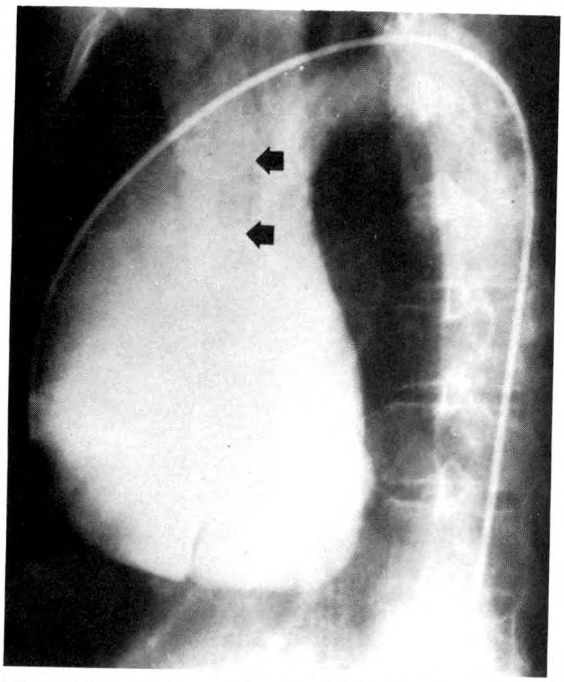


FIG. 3. Retrograde aortogram, right posterior oblique (RPO) projection. The catheter is in the false lumen. Marked enlargement of the ascending aorta is present, and the intimal flap is visible in the root of the aorta, and can also be seen near the origin of the innominate artery (arrows). The aortic valves are not seen because the injection was made into the false lumen.

casionally, because of inability to pass a catheter into the true channel, or a need for a further anatomic definition of the dissection, a second arterial puncture at another site is performed to complete the angiographic examination.

If from the femoral approach, the catheter does not advance beyond the aortic arch, a Type III dissection probably exists and the catheter is in the false channel. If free back flow of blood is present, a small hand test injection should be performed to confirm the position of the catheter. Its presence in the false channel can be confirmed by lack of filling of any branch vessels, slow flow, and the crescentic opacification of only the upper and lateral portions of the arch and descending thoracic aorta. If flow is reasonably satisfactory, angiography can be safely performed with the hand injection of 30 cc. of contrast medium, with filming over a 12 second period. Our routine views are the steep right posterior oblique and anteroposterior views, but frequently only the right posterior oblique is necessary for false channel injections.

Inadvertent pressure injection into the false channel has been reported without complication. Nevertheless, hand injections into false channels are safer, and give satisfactory aortograms.[9,10] Our experience confirms that this is a uniformly safe and valuable procedure. Assuming that there is reasonable flow within the false channel, injection as described should be done without hesitation, and frequently will give essential anatomic information not available from aortography with injection into the true lumen (Fig. 3).

The catheter should then be withdrawn into the abdominal aorta, and re-advanced, since there may be several re-entry points where the true and false channel communicate distally in the aorta. Sometimes a catheter must be withdrawn almost to the arteriotomy site to get it into the true lumen. The previous course of the catheter in the false lumen identified the position of the false lumen, so advancement into the true lumen can usually be determined without test injections. If unsuccessful, a second puncture must be done in the opposite femoral artery or axillary artery, and this will often be successful in catheterization of the true lumen.

The catheter should be positioned in the ascending aorta, and assuming good back flow, a test injection is performed to determine its position. In Type I dissections, the catheter frequently passes into the ascending aorta with ease, yet is in the false lumen. The presence of the catheter in the true lumen is determined at the test injection by visualization of the sinuses of Valsalva, filling of coronary arteries, aortic insufficiency, narrowing of the true lumen, and rapid disappearance of the contrast medium. A catheter in the false lumen is characterized by decreased pulsation of the catheter, nonvisualization of the structures of the normal aortic root, and decreased

flow in the lumen. A hand injection of 30 cc. should be made into the false channel as outlined above, and then the catheter should be withdrawn and re-advanced into the true lumen, if possible.

If the catheter is in the true lumen, a pressure injection should be done in both the steep right posterior oblique and the anteroposterior positions. Except in unusual extenuating circumstances, 2 views of the thoracic aorta should always be obtained. The anteroposterior view of the abdominal aorta is usually sufficient. Occasionally, the abdominal aortogram may give a definitive diagnosis of a dissection which is equivocal on the thoracic aortograms, and essential information regarding visceral branches is obtained. The first abdominal aortogram is obtained by injection into the ascending aorta, since this may be the only entrance by which the false channel in the abdominal aorta can be opacified. The flow may be quite slow, however, and aortograms must sometimes be done for as long as 20 seconds to fully visualize the abdominal aortic false channel. The timing sequence for the abdominal aortogram can easily be determined by observing the flow pattern on the thoracic aortogram. A second injection may be required just above the level of the diaphragm, to better delineate the abdominal aorta and its branches, assuming that the test injection does not show severe constriction of the lumen or other contraindications to injection at this level.

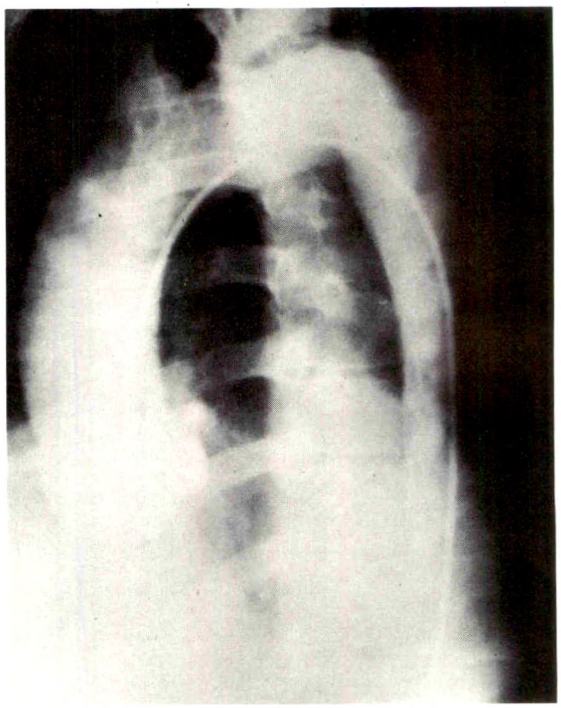


Fig. 4. Retrograde aortogram (RPO). The only indication of a dissecting aneurysm is the marked narrowing of the true aortic lumen in the descending thoracic aorta.

ANGIOGRAPHIC FINDINGS

Of the 49 patients in this report, 59 per cent had aortography performed, and visualization of a double aortic lumen was the single most common finding. The complete aortographic examination should answer the following questions. Where does the dissection begin? How far distally does it extend? What is the magnitude of aortic insufficiency? What branch vessels, including the coronary arteries, show a compromise of their lumens, and are they supplied by the true or false channels? The following angiographic signs are helpful in answering these questions.

(1) *Intimal flap.* A transverse lucent line in the ascending aorta, or longitudinal lucent line in the arch of the aorta, represents the elevated intima and portion of media separating the 2 channels (Fig. 3). Frequently, the point where the intimal flap is visible is the most proximal portion of the dissection. An intimal flap in the ascending aorta indicates a Type I or II lesion.

(2) *Narrowed true lumen.* The false lumen may be partially or completely thrombosed, and contrast medium filling the false channel may not be seen. The true lumen, however, frequently has a flattened side in the ascending aorta, and may be narrowed to varying degrees in the arch and distal aorta due to compression by the nonopacified false channel (Fig. 4; 5, *A* and *B*; and 7, *A* and *B*).[9]

(3) *Double channel.* A double channel is composed of the true and false lumen,

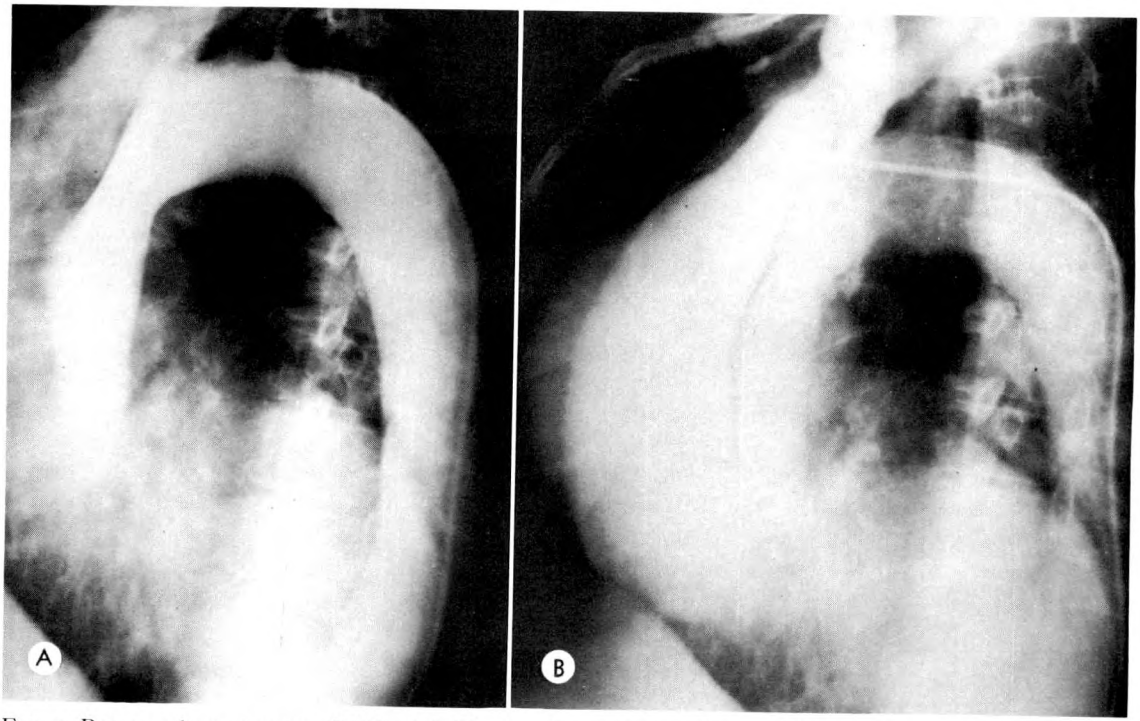


FIG. 5. Retrograde aortogram (RPO). (*A*) The true lumen is densely opacified and flattened on its anterolateral aspect by the faintly opacified false lumen. (*B*) A second injection with the catheter advanced further demonstrates equal opacification of the true and false channels (false lumen injection?), and the double channel in the ascending aorta is well seen, with the lucent stripe representing the elevated intima and media, separating the 2 channels. Aortic insufficiency is present, secondary to involvement of the aortic valve cusps.

separated by a thin lucent stripe, representing the elevated intima and a portion of the media (Fig. 3; 5, *A* and *B*; and 7, *A* and *B*). The visualization of this stripe may be difficult if both channels opacify simultaneously.[9] Usually the channels fill sequentially, however, with the true lumen filling first and the false lumen opacifying later. There frequently is a differential contrast density between the 2 lumens (Fig. 5*A*; and 7*A*). A recent communication indicates that patients with opacification of both channels have a worse prognosis and should be operated upon, as compared with those with thrombosed false channels.[4]

(4) *Aortic insufficiency.* Aortic insufficiency results from loss of support of the aortic valve cusps and dilatation of the aorta in dissecting aneurysms involving the ascending aorta (Fig. 5*B*). The degree of aortic insufficiency determines the necessity for surgical therapy and is important to quantitate. The prognosis in this group of patients is considerably worse than those without aortic insufficiency.

(5) *Aortic wall thickness.* This sign is particularly important when the false lumen does not opacify with contrast medium. The opacified true lumen usually occupies the more medial aspect of the descending aorta. A distance of greater than 1 cm. between the opacified lumen and the outer wall of the aorta outlined by the left lung represents space occupied by the false channel.[9] Occasionally, ulcer-like projections of contrast medium will extend from the true lumen into this space, which represent points of communication between the 2 channels, allowing the contrast medium to locally escape into the false lumen (Fig. 6).[11]

(6) *Catheter position.* During the injection under pressure, the catheter will re-coil against the outer wall of the aorta, and

abut against the most superior aspect of the aortic arch. When a dissection is present, the elevated intima and media do not allow the catheter to re-coil to the outer margin of the aorta as outlined by adjacent lung (Fig. 7, *A* and *B*).[1]

(7) *Branch involvement.* Dissections of the ascending aorta may extend into the brachiocephalic vessels as a localized widening of the branch near its origin, or may actually show a double lumen for a varying distance. A vessel may be partially or completely occluded by severe narrowing of the true lumen by the dissection. In the abdominal viscera, any branch may not opacify from the true channel (Fig. 7, *C* and *D*). In these cases, it is important to establish whether the false channel has a re-entry point at the origin of these vessels not fed by the true lumen. Flow in the false channel may be sufficient to maintain viable organs in the absence of any blood supply from the true lumen. The angiographic findings must be correlated with the clinical ones, since it is not unusual to see a lack of blood supply to an organ which is clinically viable. This illustrates the importance of delayed abdominal aortograms to demonstrate blood supply to the viscera *via* the false channel. The presence of a nephrogram and pyelogram often helps to demonstrate satisfactory blood supply to the kidneys.

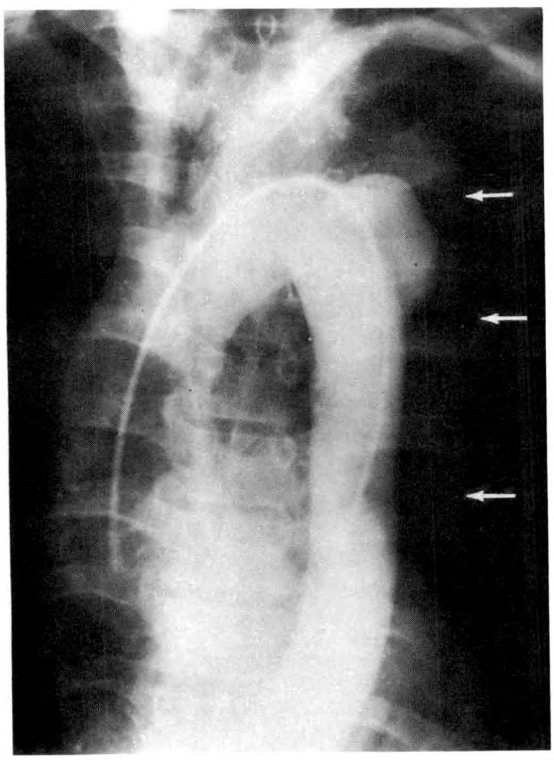


Fig. 6. Retrograde aortogram (anteroposterior view). An aneurysm-like projection extending laterally from the true lumen is evident. The abnormal aortic wall thickness indicates that this is a dissecting rather than a saccular aneurysm. The remainder of the dissected false channel was apparently thrombosed, preventing its opacification (arrows).

If doubt exists, an arterial puncture from the opposite femoral artery may allow catheterization of the false channel, and the viscera supplied from it can then be outlined. A very interesting example of this technique is illustrated in a previous communication.[11] It is frequently not possible to visualize the site of re-entry, but it can sometimes be surmised by visualizing the transition from the dissection to normal appearing aorta and iliac vessels.

MORTALITY

The most common cause of death in our series was due to rupture of a dissecting aneurysm into the pericardium, with acute tamponade. The mortality figures indicate that those dissections beginning in the ascending aorta (Type I and II) have a much higher mortality than those beginning distal to the left subclavian artery (Type III). The over-all hospital mortality including patients who died before therapy could be instituted, was 75 per cent (18/24) in Type I, 56 per cent (5/9) in Type II, and 19 per cent (3/16) in Type III.

It is evident that the angiographic demonstration of the origin of the dissection is essential in the determination of both therapy and prognosis.

CONCLUSION

1. Acute dissecting aneurysms of the aorta carry a high mortality rate, and a

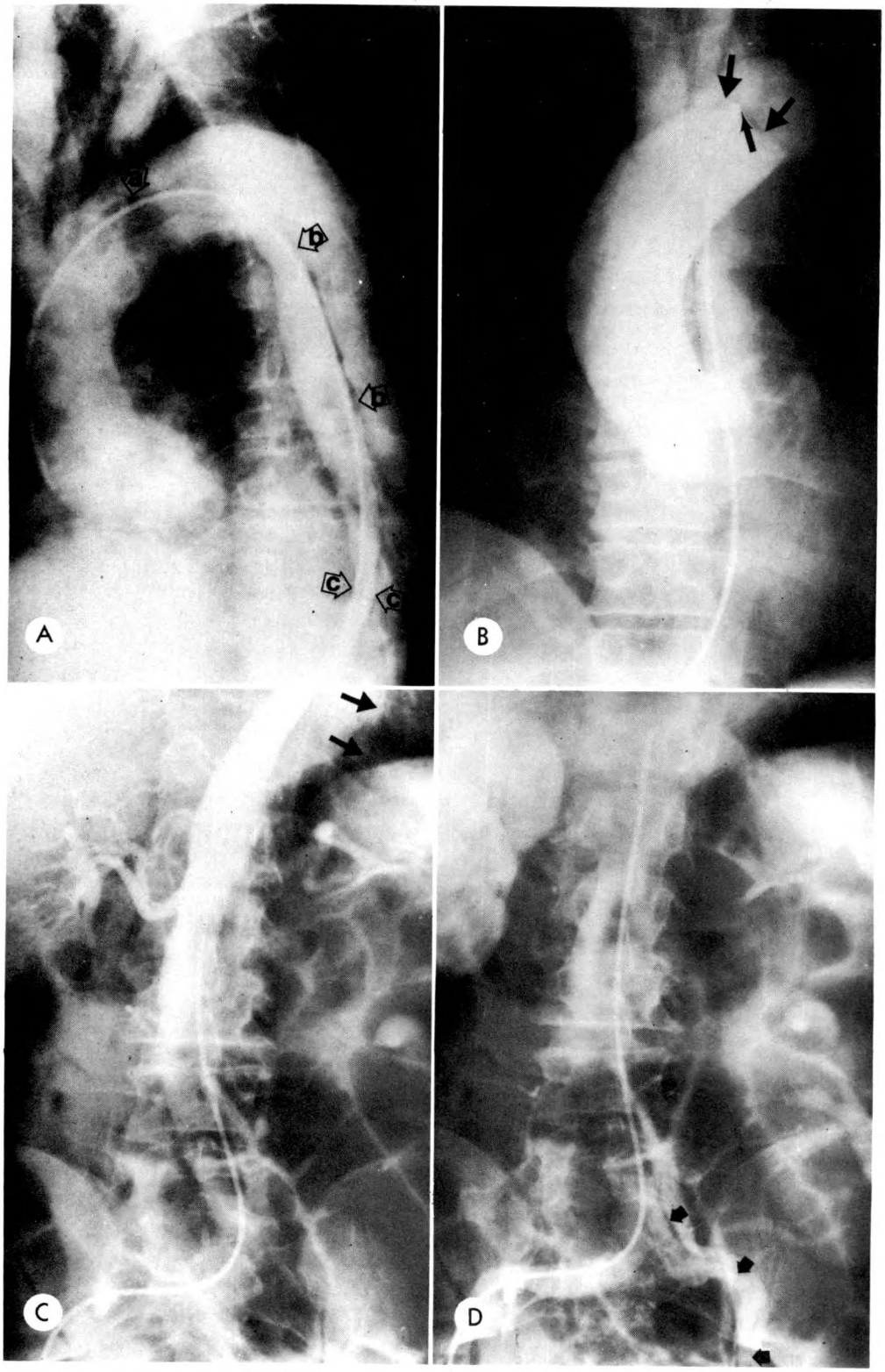

A
B
C
D

Fig. 7. (*A*) Retrograde aortogram (RPO). The intimal flap at the origin of the left subclavian artery is visible (arrow a). The elevated intima and media separate the true channel medially from the false channel laterally (arrows b). Marked narrowing of the true lumen is seen distally (arrows c). (*B*) Anteroposterior view. Both channels can be seen on end with the elevated intima and media separating them (large arrows). Note that during the injection, the catheter does not recoil to the outer margin of the aorta (small arrow). (*C*) Abdominal aortogram (early). The true channel is opacified, and the outer margin of the aorta is outlined by adjacent lung (arrows). The right renal artery and celiac axis are opacified. (*D*) Abdominal aortogram (late). The left kidney receives its blood supply from the false channel, so that a marked difference in the nephrogram is evident between the 2 sides. The left renal pyelogram (ectopic in the left lower quadrant) indicates that this kidney is still functioning. The arrows show the dissection extending into the left common and external iliac arteries.

definitive diagnosis depends on prompt aortography.

2. The underlying causes for dissection are degeneration of the aortic media, rupture of the vasa vasorum, intimal tear with longitudinal cleavage along the media, and systemic hypertension.

3. Aortographic abnormalities in dissecting aneurysms include:

a. lucent transverse intimal flap at the origin of the dissection.
b. narrowing of the true aortic lumen.
c. double aortic channels separated by elevated intima and part of the media.
d. aortic insufficiency.
e. abnormally thick aortic wall.
f. abnormal catheter re-coil and position.
g. involvement of branch vessels.

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REFERENCES

1. Cramer, G. G., and Amplatz, K. Catheter position: aid in diagnosis of dissecting aneurysm of thoracic aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 836–839.
2. Daily, P.O., Trueblood, H. W., Stinson, E. B., Wuerflein, R. D., and Shumway, N. E. Management of acute aortic dissections. *Ann. Thoracic Surg.*, 1970, *10*, 237–247.
3. DeBakey, M. E., Henly, W. S., Cooley, D. A., Morris, G. C., Jr., Crawford, E. S., and Beall, A. C., Jr. Surgical management of dissecting aneurysm involving ascending aorta. *J. Cardiovasc. Surg.*, 1964, *5*, 200–211.
4. Dinsmore, R. E., Willerson, J. T., and Buckley, M. J. Dissecting aneurysm of aorta: aortographic features affecting prognosis. *Radiology*, 1972, *105*, 567–572.
5. Dow, M. C., Roebuck, E. J., and Cole, F. Dissecting aneurysms of aorta. *Brit. J. Radiol.*, 1966, *39*, 915–927.
6. Hirst, A. E., Jr., Johns, V. J., Jr., and Kime, S. W., Jr. Dissecting aneurysm of aorta: review of 505 cases. *Medicine*, 1958, *37*, 217–279.
7. Kirschner, L. P., Twigg, H. L., Conrad, P. W., and Hufnagel, C. Retrograde catheter aortography in dissecting aortic aneurysms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 349–353.
8. Lindsay, J., Jr., and Hurst, J. W. Dissecting aneurysms of aorta. *J.A.M.A.*, 1971, *217*, 1533–1536.
9. Shuford, W. H., Sybers, R. G., and Weens, H. S. Problems in aortographic diagnosis of dissecting aneurysm of aorta. *New England J. Med.*, 1969, *280*, 225–231.
10. Soto, B., Harman, M. A., Ceballos, R., and Barcia, A. Angiographic diagnosis of dissecting aneurysm of aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 146–154.
11. Stein, H. L., and Steinberg, I. Selective aortography: definitive technique for diagnosis of dissecting aneurysm of aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 333–348.
12. Wheat, M. W., Jr., Harris, P. D., Malm, J. R., Kaiser, G., Bowman, F. O., Jr., and Palmer, R. F. Acute dissecting aneurysms of aorta: treatment and results in 64 patients. *J. Thoracic & Cardiovasc. Surg.*, 1969, *58*, 344–351.
13. Wheat, M. W., Jr., and Palmer, R. F. Dissecting aneurysms of aorta: present status of drug versus surgical therapy. *Prog. Cardiovasc. Dis.*, 1968, *11*, 198–210.

CONTINUOUS ULTRASOUND "B" SCANNING OF ABDOMINAL AORTIC ANEURYSMS*

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PATIENTS with abdominal aortic aneurysms usually present with a pulsatile abdominal mass with or without pain in the upper abdomen radiating into the low back. Palpation of a pulsatile abdominal mass with an overlying systolic bruit, and observation of curvilinear calcifications on routine abdominal roentgenograms lead to an accurate diagnosis of these aneurysms in the majority of patients.[9] However, clinical assessment of the abdominal aorta is difficult in the obese patient or in the patient with abdominal pain and guarding, and does not differentiate intra-abdominal masses transmitting aortic pulsations from aortic aneurysms. Since calcifications are not always present in the aneurysmal sac, plain roentgenograms cannot be relied upon. Aortography adds to the accuracy of a diagnosis, but has considerable risk in this disease and age group, and may be misleading when the noncalcified aneurysmal sac is filled with thrombus and the aortic lumen appears normal.[9] Scintigraphic flow studies may be similarly misleading.

Conventional contact compound ultrasonic "B" examination of the abdominal aorta provides a valuable adjunct to clinical and roentgenologic studies.[1–7]

Continuous ultrasonic "B" scanning provides a rapid and more dynamic biplane assessment of the abdominal aorta.[12] We have evaluated the use of continuous ultrasonic "B" scanning of the abdomen as an aid in the diagnosis of suspected aortic aneurysms in 227 patients.

MATERIAL AND METHOD

Each patient was examined in the supine position using an ultrasonic sectional imaging unit (Vidoson 636-Siemens) which consisted of a display oscilloscope (CRT) and a transmitter-receiver in which two 2.5 MHz transducers rotate at the focus of a parabolic acoustical mirror. The sound beam is directed toward the mirror and reflected from it to the patient through a coupling water bath. As the transducers rotate a continuous linear "B" scan is produced and is repeated approximately 16 times per second. The tomoscopic 14×16 cm. image is displayed on the CRT and recorded with a Polaroid camera or on videotape. The aorta is viewed in both longitudinal and transverse planes employing a variety of gain settings for the same transmitter positions. Aortic diameters were measured with the 2 cm. graticule on the CRT. The criteria of Steinberg *et al.*[11] for abdominal aortic aneurysms were utilized.

Confirmation of the ultrasound diagnosis was obtained in 91 patients: by operation in 72 patients; autopsy in 11 patients; and angiography in 8 patients. Of these 91 confirmed cases, 67 patients had abdominal roentgenograms.

RESULTS

It was possible to identify the abdominal aorta with continuous ultrasonic "B" scanning in 90 of the 91 confirmed cases.

As shown in Table 1, the study provided a correct diagnosis in all but 1 of the 91 cases where confirmation was available including 74 fusiform aneurysms (Fig. 2, *A* and *B*), 8 dissecting aneurysms (Fig. 4, *A–C*) and 3 false aneurysms associated with teflon grafts (Fig. 6, *A–D*).

Characteristic aneurysmal calcifications

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P.Q., Canada, September 25–28, 1973.
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Table I

SUSPECTED ABDOMINAL AORTIC ANEURYSM: DIAGNOSIS CONFIRMED—91 PATIENTS

	Predicted Ultrasound Diagnosis	Confirmed Diagnosis
Fusiform or Saccular Abdominal Aortic Aneurysm	74	75
Dissecting Aneurysm	8	8
False Aneurysm	3	3
Normal Aorta	1	1
Normal Aorta with Pre-Aortic Tumor Mass	4	4
Total	90	91

Table II

SUSPECTED ABDOMINAL AORTIC ANEURYSM: DIAGNOSIS NOT CONFIRMED—136 PATIENTS

Fusiform or Saccular Abdominal Aortic Aneurysms	29
Normal Aorta	107

were visible in only 34 of the 67 patients with proved aneurysms who had abdominal roentgenograms.

In 1 patient the abdominal aorta was not identified by ultrasound. This was a case of a ruptured abdominal aortic aneurysm with marked paralytic ileus. The gas filled bowel loops prevented visualization of the abdominal aorta. Four patients thought clinically to have abdominal aortic aneurysm were correctly diagnosed preoperatively as pre-aortic tumor masses (Fig. 7, *A* and *B*; and 8, *A* and *B*). One patient early in our series who had prominent abdominal pulsation and calcification of the aorta on an abdominal roentgenogram underwent a laparotomy despite a negative ultrasonic diagnosis and a normal aorta was found.

In 136 patients no confirmation of the ultrasound diagnosis is available (Table II). Twenty-nine of these were diagnosed as abdominal aortic aneurysms. Surgery is planned in several and has been deferred in others because of the small size of the aneurysm or the medical condition of the patient.

Intraluminal clot was readily identified by its fill in at higher gain settings (Fig. 2, *A* and *B*; and 3, *A–D*). The pulsations of the aortic wall anterior to the thrombus were damped (Fig. 3*D*).

In our 8 cases of dissecting aneurysm a second sonolucent channel anterior or pos-

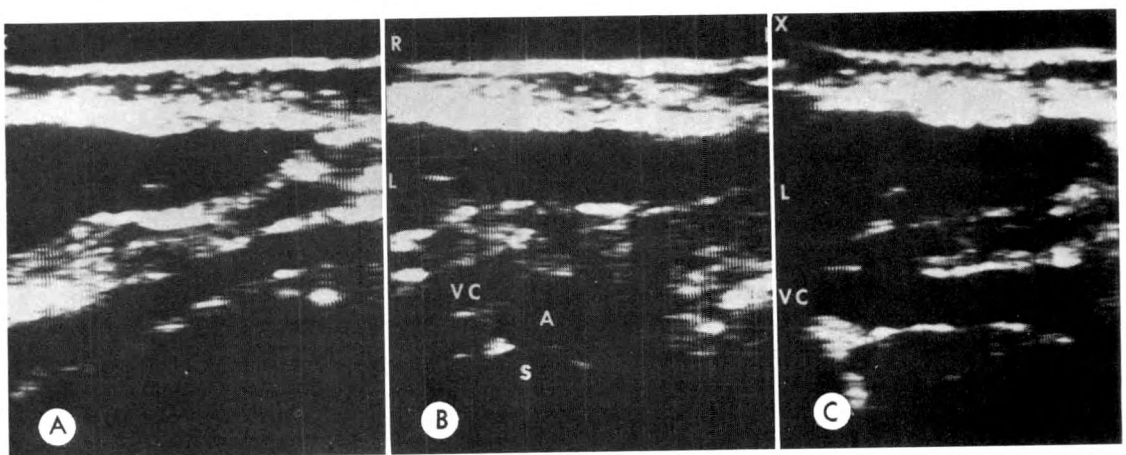


Fig. 1. Normal aorta and inferior vena cava of a 70 year old man (*A*) Longitudinal section of aorta (A) below the liver (L) from the level of the xiphoid (X) to the umbilicus. (*B*) Transverse section through the aorta beneath the liver (L). Note the inferior vena cava (VC) superior and to the right of the aorta. R = right, L = left, S = spine. Graticule indicates 2 cm. (*C*) Longitudinal section of the inferior vena cava. Note the horizontal direction of the vena cava below the liver.

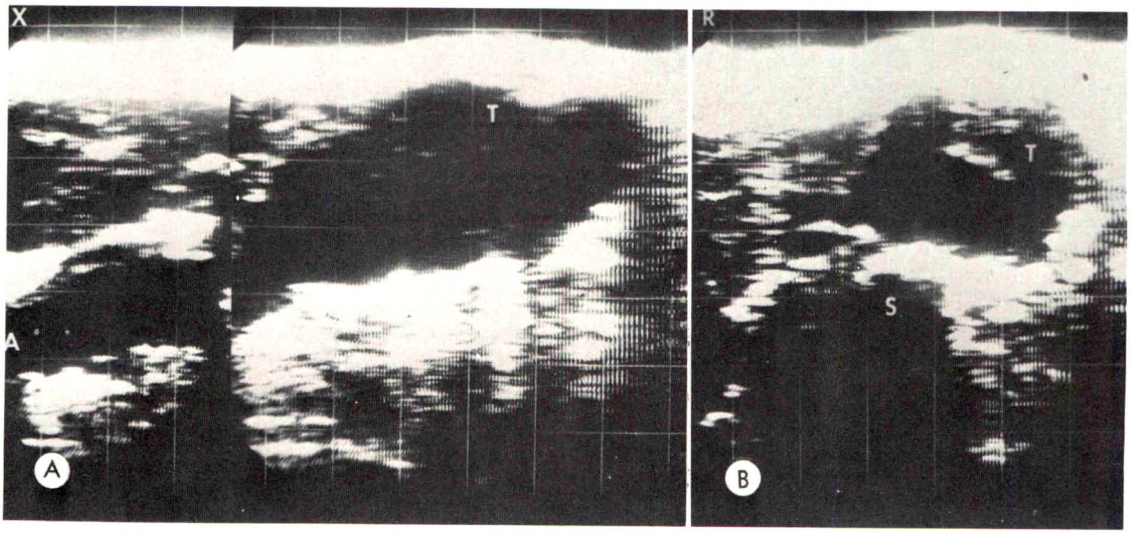


FIG. 2. Lobulated fusiform abdominal aortic aneurysm. Surgical confirmation. (*A*) Composite longitudinal echogram in a 57 year old man with a pulsatile abdominal mass. Note that the normal aorta (A) under the liver expands from an anteroposterior diameter of 3 cm. to 6 cm. below the umbilicus. Thrombus (T) along the anterointernal aortic wall is represented by linear echo reflections. (*B*) Transverse section at the widest portion showing the internal thrombus (T) on the anterior left (L) wall. The aorta is anterior to the spine (S) and only 2 cm. below the anterior abdominal wall.

terior to the true lumen was identified (Fig. 4, *A–C*). This did not fill in at high gain settings.

Teflon grafts were readily identified by their strong parallel echoes, the spacing of which corresponded to the size of the graft. They did not exhibit expansile pulsations, whereas adjacent false aneurysms pulsated arterially (Fig. 6, *A–D*).

Although the size and topographic location of the aneurysm could be accurately predicted, we made no systematic attempt to relate precisely the aneurysm to the renal arteries.* Involvement of the iliac artery was, however, readily identified (Fig. 5, *A* and *B*). The vena cava was some-

* Only 1 case in this series actually had renal artery involvement and this was suspected from the ultrasonic study.

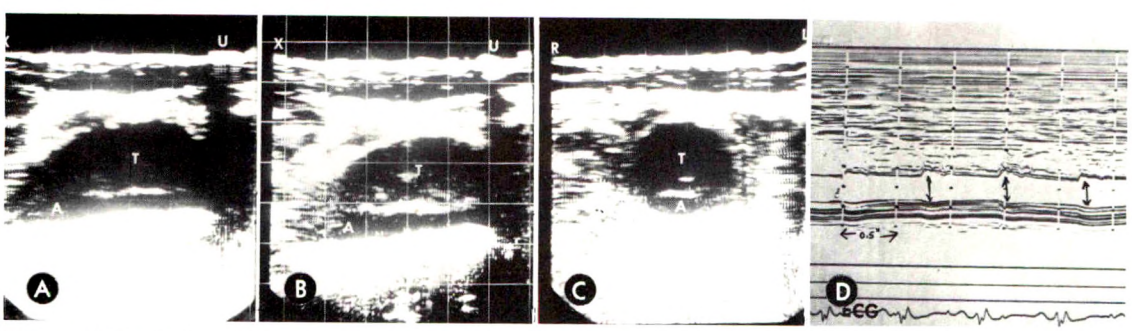


FIG. 3. Abdominal aortic aneurysm with extensive thrombus filling the lumen in a 55 year old man. (*A*) Longitudinal section of abdomen through aorta (A) which is expanded to 4×6 cm. The true lumen is the transonic posterior stripe measuring about 1 cm. The 4 cm. anterior portion of the aorta is occupied by an anterior wall thrombus (T). (*B*) Increased gain setting at the same longitudinal section shows aortic lumen (A) and the filling in of the anterior wall thrombus (T). Graticule scale is 2 cm. (*C*) Transverse section at the widest portion of the aneurysm. R = right, L = left. (*D*) Strip recording of M mode ultrasound over the abdominal aorta. The true aortic lumen marked with vertical arrows.* Note the expansile pulsations following systole as recorded on the ECG. Surgical confirmation. (*Vertical dots represent 1 cm.)

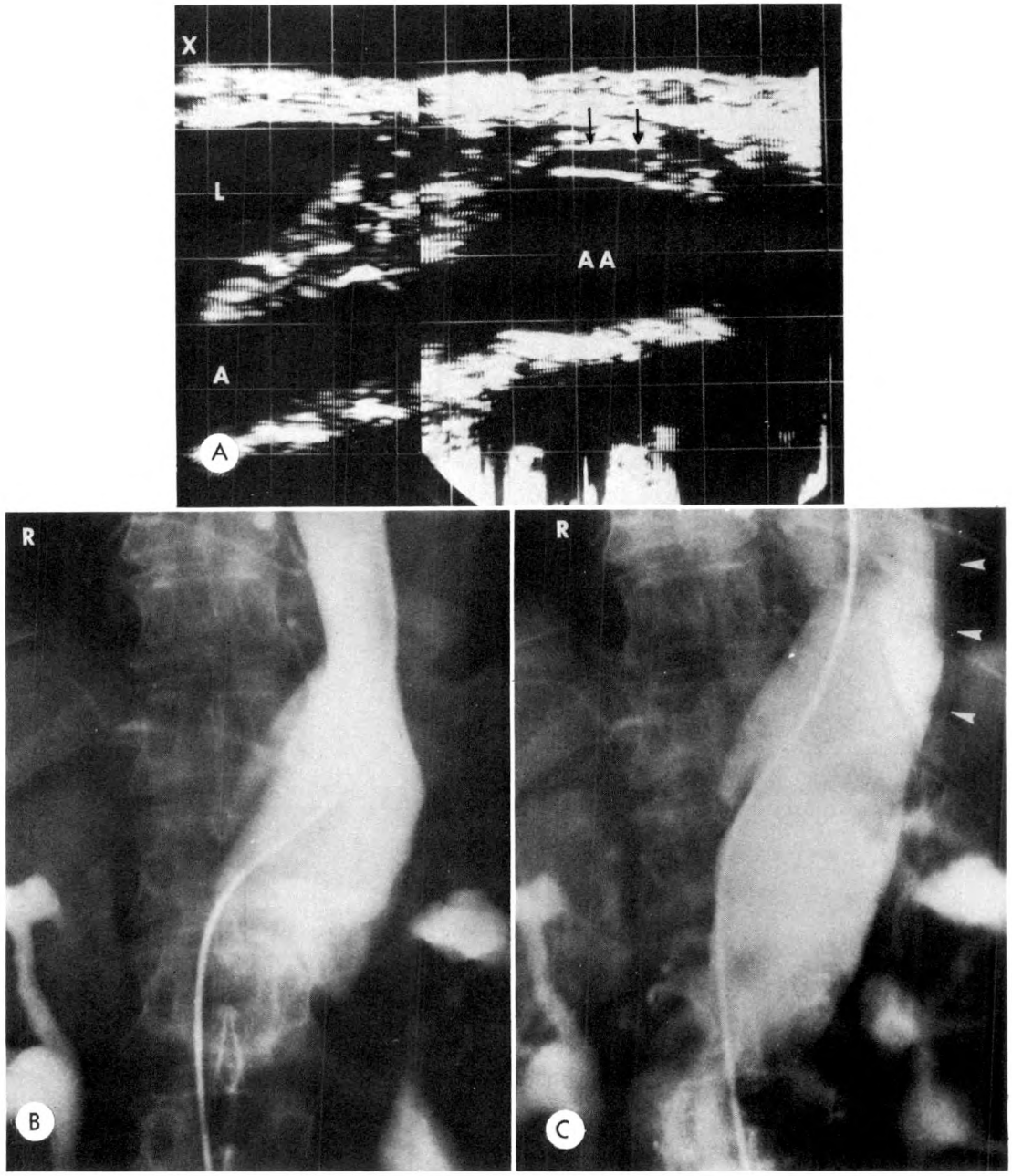


FIG. 4. A 65 year old man with a left lower quadrant pulsatile mass. (*A*) Composite longitudinal section through aorta (A) and aortic aneurysm (AA). Sonolucent stripe anteriorly (arrows) represents a false channel. X = xiphoid, L = liver. Graticule scale is 2 cm. (*B*) Retrograde femoral aortogram shows the dilated contrast filled lumen early in the film series with the left renal artery arising from this segment. (*C*) Delayed aortogram shows filling of the second channel superiorly on the left confirming the dissection (arrows).

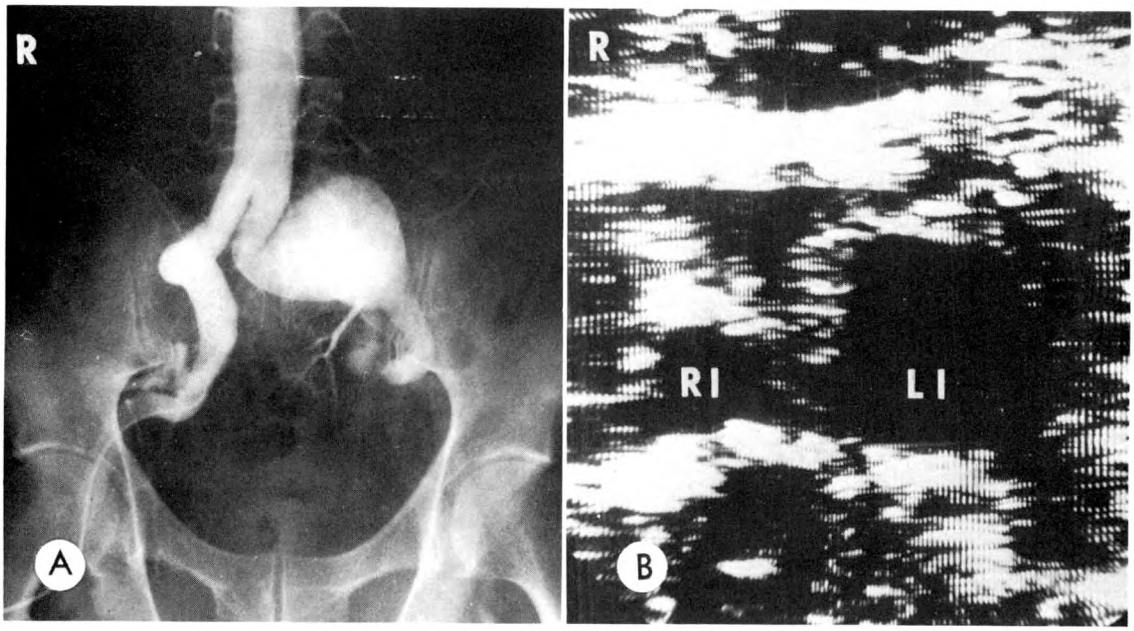


FIG. 5. A 71 year old man with a left lower quadrant pulsatile mass. (*A*) Aortogram shows a normal aorta and a left common iliac artery aneurysm. R = right. (*B*) Transverse echogram below the umbilicus shows the normal right common iliac artery (RI) and the left common iliac aneurysm (LI). Surgical confirmation.

times seen in longitudinal and transverse sections and can be differentiated from normal aorta or aneurysm by its location, its more horizontal direction and by its movements which are influenced by the phase of respiration (Fig. 1, *A–C*).

All ultrasound examinations were completed within 10 minutes.

DISCUSSION

The value of an accurate noninvasive atraumatic technique for the diagnosis of abdominal aortic aneurysm is apparent. Continuous ultrasonic "B" scanning is faster, easier, and more convenient than conventional contact "B" scanning. It also permits visualization of pulsations which facilitate differentiation of the aorta from confusing parallel echoes, the vena cava, pre-aortic tumors or teflon grafts. With contact "B" scanning, pre-aortic transonic masses silhouette the anterior wall of the aorta and it may not be demonstrated ultrasonically.[3] We have not found this to be a problem with continuous ultrasound "B" scanning as the pulsatile wall of the aorta is visualized separately from the pre-aortic masses (Fig. 7, *A* and *B*; and 8, *A* and *B*). The appropriate horizontal, longitudinal or oblique section can be rapidly obtained by visual feedback. Gain settings can be varied without recourse to repeated scanning, thus permitting ready differentiation of clot from lumen. The pressure of the scanning head helps displace overlying bowel which sometimes prevents visualization of the aorta. Continuous ultrasonic "B" scanning is more accurate than clinical examination, plain roentgenography, aortography or scintigraphic flow studies in the diagnosis of abdominal aortic aneurysms. It also provides evidence of dissection, intraluminal thrombus and aneurysmal involvement of the iliac arteries.

The simplicity and rapidity of the technique allow serial examinations of the abdominal aorta to assess progress of the aneurysm in the patient in whom surgery has not been elected. We have used the study postoperatively to detect false aneurysms.

SUMMARY

A series of 227 patients with suspected

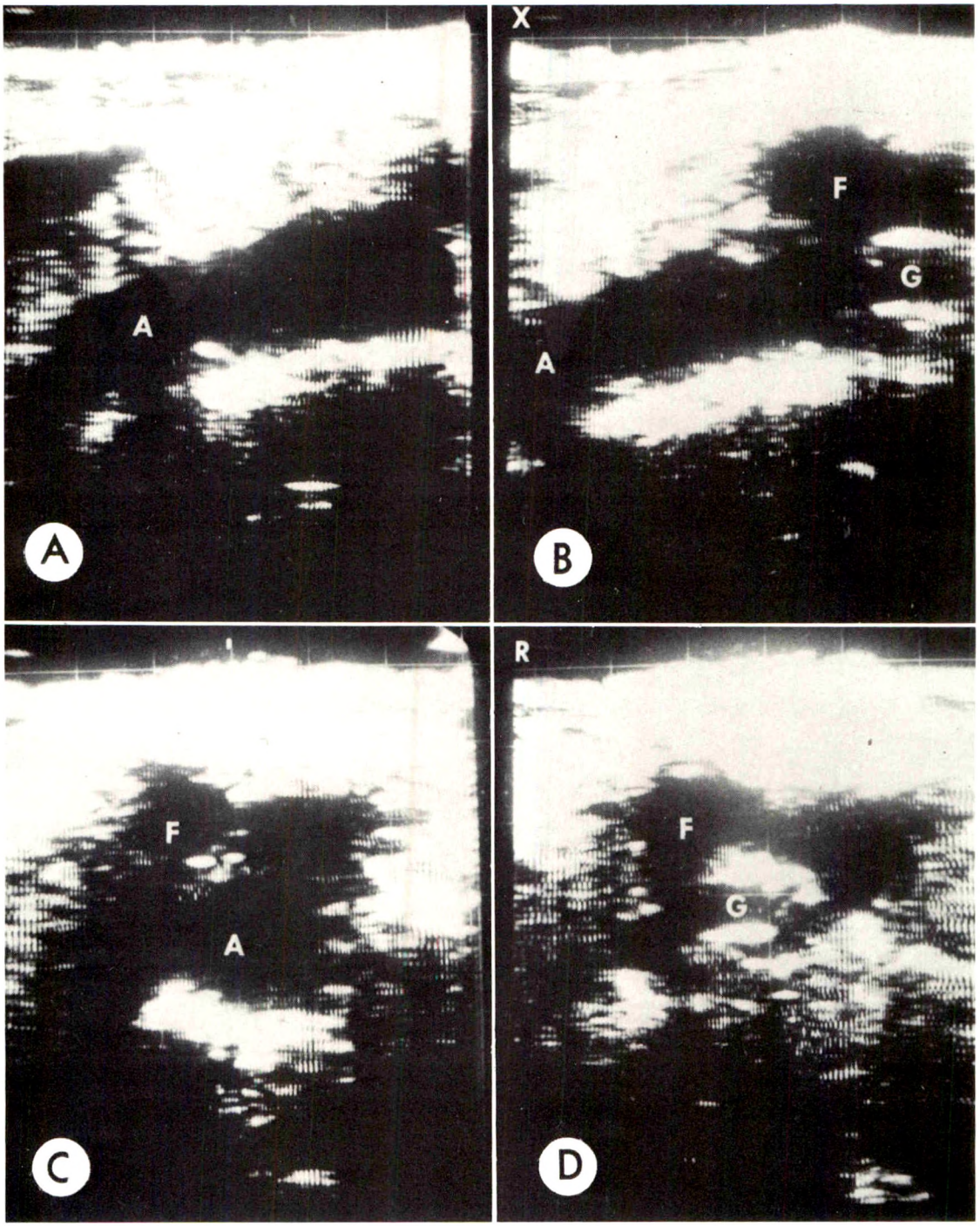


FIG. 6. An 80 year old female with pulsatile abdominal mass 4 years following resection of abdominal aortic aneurysm and insertion of teflon graft. (*A*) Left upper shows a longitudinal section of the suprarenal aorta (A) from under the xiphoid (X) to the umbilicus. (*B*) Right upper shows a false aneurysm (F) at the junction of the teflon graft (G) and the aorta. (*C* and *D*) Lower photos show transverse sections at the level of the aorta and false aneurysm (F) and the teflon graft and the false aneurysm. Autopsy confirmation.

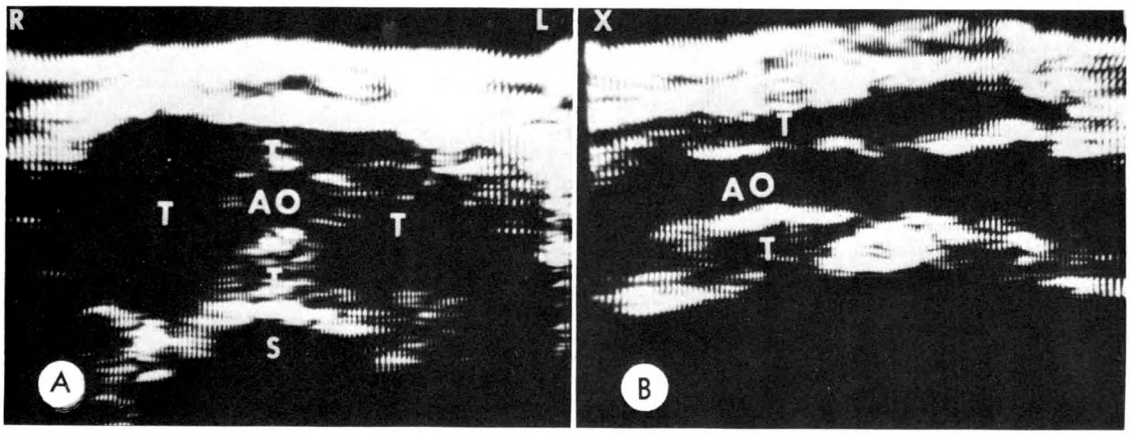


FIG. 7. Suspected aortic aneurysm in a 68 year old man. (*A*) Transverse section through area of pulsatile abdominal mass. The aorta (AO) is normal in size. A transonic mass surrounds the normal aorta (T). R = right, L = left, S = spine. (*B*) Longitudinal section through the aorta (AO) showing the tumor mass (T) surrounding the aorta. Laparotomy showed enlarged para-aortic lymph nodes with metastatic carcinoma from the prostate. X = level of xiphoid, U = level of umbilicus.

abdominal aortic aneurysm was studied by continuous ultrasonic "B" scanning. The precise diagnosis has been confirmed by surgery, autopsy, or angiography in 91 patients.

The predicted ultrasound diagnosis was correct in all but 1 of the 91 cases.

Additional aortic pathology was accurately demonstrated including intraluminal thrombus, dissections, false aneurysm,

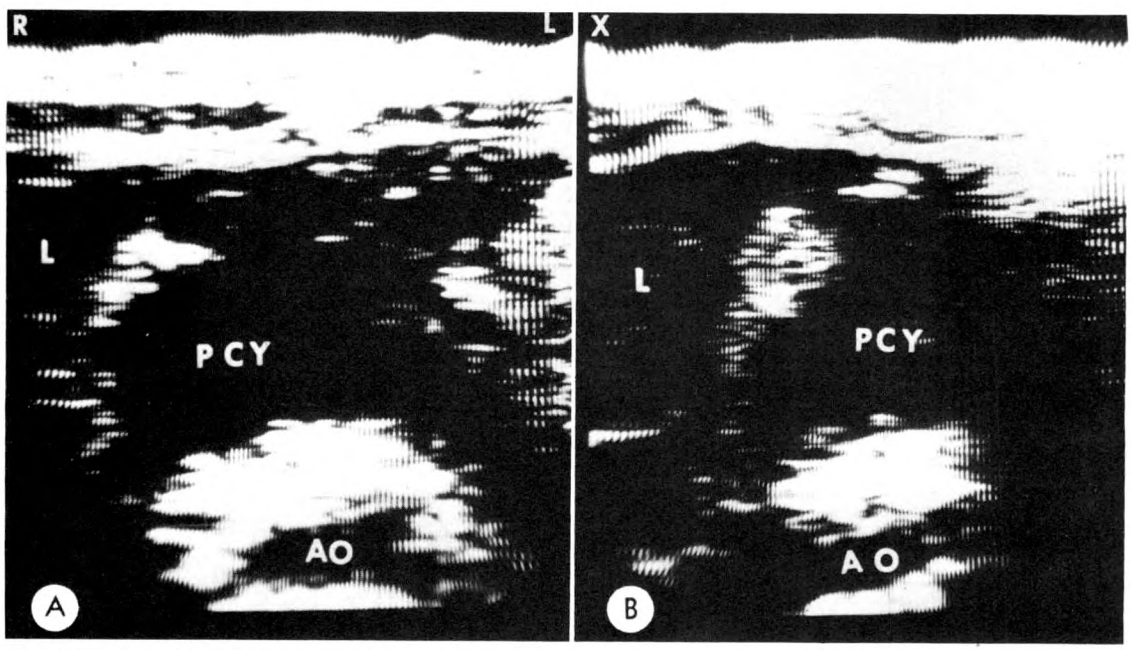


FIG. 8. Differential diagnosis included pre-aortic masses. (*A*) Transverse section through the upper abdomen over the pulsatile abdominal mass. The normal aorta (AO) showed pulsations which were transmitted through the fluid filled pancreatic pseudocyst (PCY) located under the liver (L). R = right, L = left. (*B*) Longitudinal section to the right of the midline. Note the pancreatic pseudocyst above the normal aorta and below the inferior edge of the right lobe of the liver. Surgical confirmation. X = level of xiphoid, U = level of umbilicus.

teflon grafts and aneurysmal involvement of the iliac arteries.

In 136 patients, including 29 diagnosed as abdominal aneurysm, no confirmation is yet available.

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REFERENCES

1. BROWN, R. E. Echoaortography. *Ultrasonographia medica*, 1972, *3*, 139–146.
2. EVANS, G. C., LEHMAN, J. S., SEGAL, B. L., LIKOFF, W., ZISKIN, M., and KINGSLEY, B. Echoaortography. *Am. J. Cardiol.*, 1967, *19*, 91–96.
3. FREIMANIS, A. K., and ASCHER, W. M. Development of diagnostic criteria in echographic study of abdominal lesions. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *108*, 747–755.
4. GOLDBERG, B. B., OSTRUM, B. J., and ISARD, H. J. Ultrasonic aortography. *J.A.M.A.*, 1966, *198*, 353–358.
5. HOLM, H. H. Ultrasonic scanning in diagnosis of space occupying lesions of upper abdomen. *Brit. J. Radiol.*, 1971, *44*, 24–36.
6. HOLM, H. H., KRISTENSON, J. K., MORTENSEN, T., and GAMMELGAARD, P. A. Ultrasonic diagnosis of arterial aneurysms. *Scandinav. J. Thoracic & Cardiovasc. Surg.*, 1968, *2*, 140–146.
7. LEOPOLD, G. R. Ultrasonic abdominal aortography. *Radiology*, 1970, *96*, 9–14.
8. RETTENMAIER, G. Intraabdominelle Bewegungsvorgänge im Ultraschallschnittbild. *Ultrasonographia medica*, 1972, *3*, 71–75.
9. ROBICSEK, F., DAUGHTERY, H. K., MULLEN, D. C., TAM, W., and SCOTT, W. P. Value of angiography in diagnosis of unruptured aneurysms of abdominal aorta. *Ann. Thoracic Surg.*, 1971, *11*, 538–550.
10. SEGAL, B. L., LIKOFF, W., ASPERGER, Z., and KINGSLEY, B. Ultrasonic diagnosis of abdominal aortic aneurysm. *Am. J. Cardiol.*, 1966, *17*, 101–103.
11. STEINBERG, C. R., ARCHER, M., and STEINBERG, I. Measurement of abdominal aorta after intravenous aortography in health and arteriosclerotic peripheral vascular disease. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *95*, 703–708.
12. WINSBERG, F., and COLE, C. M. Continuous ultrasound visualization of pulsating abdominal aorta. *Radiology*, 1972, *103*, 455–457.

DOUBLE CATHETER TECHNIQUE IN AORTOGRAPHY*

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MULTIPLE systems have been developed for arteriographic opacification of the aorta and its branches. Since the successful puncture and opacification of the abdominal aorta by dos Santos, the approach to direct aortography has become more sophisticated.[1,2,10,11] The Seldinger technique has made the aorta more accessible *via* various routes including brachial, femoral, and axillary.[4,9] Less commonly used points of entry for aortography include the carotid and suprasternal approach.[3,6] Recently, the aorta has become accessible *via* the umbilical artery and power injections of contrast material by this route can be made without significant sequelae.[7]

Technical improvements have yielded not only better catheters, but also forms of guidewires including the deflector, preformed curve, and variable stiffness types which have facilitated ease of catheter aortography and selective catheter arteriography.[2,5,8]

Little has been published about the effectiveness of curved catheters for directional control in the aorta, although Hanafee[4] has used a type of double catheter approach to negotiate curves.

A double catheter technique, to be described in detail here, requiring one exchange over a guidewire has proven helpful when a deflector guide is not available or other guides have been unsuccessful in traversing the aortic arch. On patients in whom abdominal and thoracic aortography is desired and the only approach available is through the right axillary artery, this

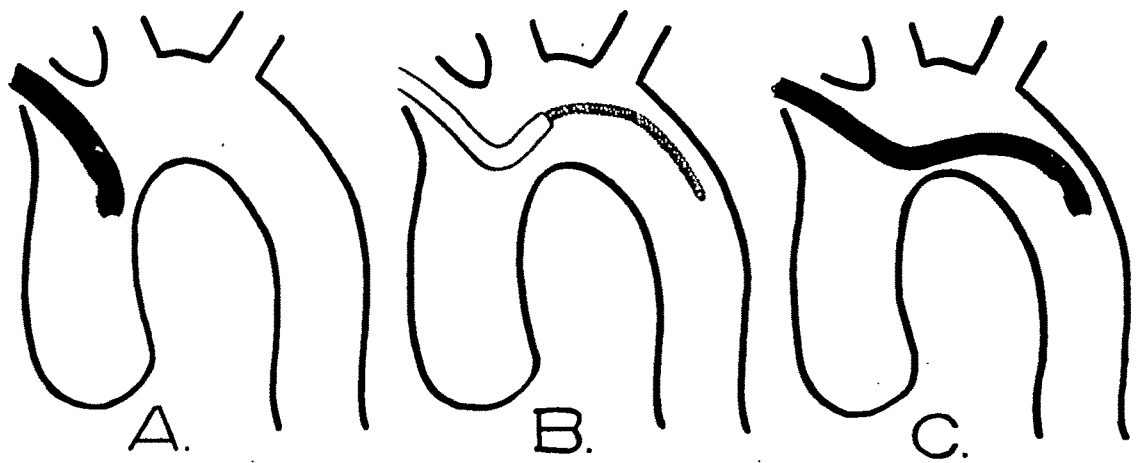


FIG. 1. (*A*) Multiple side hole catheter is in the arch aortography position. The guidewire is replaced following contrast material injection and passage into the descending aorta attempted. (*B*) If the guidewire does not enter the descending aorta, a single end hole curved catheter is placed in the aortic arch pointing towards the descending aorta. (*C*) The guidewire is advanced into the descending aorta, the flush catheter placed in the abdominal aorta over the guide, and aortography performed.

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double catheter technique can be extremely helpful.

TECHNIQUE

By means of the Seldinger technique *via* the right axillary artery, an aortic flush catheter is placed in the ascending aorta above the aortic valve and arch aortography is carried out (Fig. 1*A*). An attempt is then made to pass a guidewire including various forms of the curved aortic J's into the descending aorta. If these attempts are unsuccessful, the initial catheter is removed with the guidewire remaining in the ascending aorta. A single end hole curved catheter with good "memory" is then placed in the aortic arch with its end hole pointing toward the descending aorta (Fig. 1*B*). The guidewire is passed through the catheter and advanced into the descending aorta. Following this, the curved catheter is removed with the guidewire maintained in the descending aorta and the flush catheter advanced over the guidewire (Fig. 1*C*). Abdominal aortography is carried out and the examination completed. If desired, selective abdominal arteriography can also be carried out utilizing this system.

SUMMARY

A double catheter technique for traversing the tortuous, unfolded, and elongated aortic arch is described which does not necessitate using a deflector technique.

It has particular applicability in patients in whom both thoracic and abdominal aortography is desired. It is extremely useful in patients in whom the only approach permitted the angiographer is the right axillary artery.

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REFERENCES

1. Amplatz, K. Translumbar catheterization of abdominal aorta. *Radiology*, 1963, *81*, 927–931.
2. Cornell, S. H., and Lerona, P. T. Refinement in translumbar catheterization: use of J-tip movable core guide wire. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 194–195.
3. González, L. L., Olinger, C., and Wiot, J. F. Percutaneous transcarotid thoracic aortography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *91*, 385–393.
4. Hanafee, W. Axillary artery approach to carotid, vertebral, abdominal aorta, and coronary angiography. *Radiology*, 1963, *81*, 559–567.
5. Hawkins, I. F., Jr. Deflector catheter approach to abdominal aorta. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 196–198.
6. Lehman, J. S., Lemmon, W. M., Boyer, R. A., and Fitch, E. A. Suprasternal thoracic aortography: suprasternal transthoracic needle puncture opacification of thoracic aorta. *Radiology*, 1959, *73*, 18–36.
7. Lowery, B., and White, R. I. Modified umbilical artery catheter for power-injection aortography. *Radiology*, 1972, *105*, 711–712.
8. McFadden, J. T. Needle-catheter aortography and selective angiography: technical note. *J. Neurosurg.*, 1970, *32*, 491–495.
9. Roy, P. Percutaneous catheterization via axillary artery: new approach to some technical roadblocks in selective arteriography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *94*, 1–18.
10. Sanders, R. J., and Ramirez, F. Translumbar aortography. *S. Clin. North America*, 1969, *49*, 1317–1324.
11. Stocks, L. O., Halpern, M., and Turner, A. F. Complete translumbar aortography: teflon sleeve technique. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 835–839.

RADIATION REDUCTION BY MEANS OF LOW PULSE-RATE FLUOROSCOPY DURING CARDIAC CATHETERIZATION AND CORONARY ARTERIOGRAPHY*

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RADIATION protection and dose limitation should be a matter of concern to all modern physicians including those who perform vascular catheterization procedures. Many of us who are performing these procedures were taught to use intermittent bursts of radiation during catheter manipulation in order to limit radiation exposure both to us and the patient. However, this is not always possible, as repeated short images on the television monitor frequently will not yield enough information to selectively catheterize the desired artery, especially in the more difficult procedures.

Recently, techniques developed by the commercial television industry have been applied to medical fluoroscopy which have resulted in significant dose reduction. Initial results of low pulse-rate fluoroscopy have been previously reported.[3,4] The purpose of this paper is to update this information giving the results of our clinical use during the past $2\frac{1}{2}$ years.

MATERIAL AND METHOD

Dose reduction low pulse-rate fluoroscopy has been accomplished by integrating a magnetic video disc recorder into our x-ray system (Fig. 1; and 2). The advantage of the disc recorder over the tape recorder for this purpose is that a single television image can be recorded on a single revolution of the disc. This single image after being recorded on the disc may be played back continuously without further radiation and with no deterioration of the image because the disc is constantly revolving (3,600 rpm). The result is a stop-action view of a previously recorded fluoroscopic event.

The essence of this technique, then, is to play back a prerecorded event without having to continuously radiate the patient. Up to this time the greatest applicability of this technique was in orthopedic and gastrointestinal radiology[2,3] where rapidly moving objects are not of interest, the important advantage being the instant replay. The immediate image obviates waiting for a permanent roentgenogram to be developed and continuously irradiating the patient to maintain a constant fluoroscopic image. To obtain what might be called a pseudocontinuous image a disc recorder has been integrated into the radiographic equipment in our cardiovascular catheterization laboratory so that an image produced by an x-ray pulse is automatically recorded and can be played back at various rates of up to 15 per second, the eye fusion rate.[4]

To understand the method of dose reduction it must be recalled that television images (frames) are normally produced at a rate of 30 frames per second or 1 frame in 1/30 of a second. One television frame consists of 2 individual fields with 1/60 second per field while 1 disc revolution at 3,600 rpm (60 revolutions/second or 1 revolution in 1/60 second) will record 1 television field. In our system each single field that is recorded must be displayed twice during playback and superimposed to fit a 30 frames/second television system.

The disc recorder triggers pulses at preselected programmed rates of 15/sec., $7\frac{1}{2}$/sec., $3\frac{3}{4}$/sec., $1\frac{7}{8}$/sec., $\frac{15}{16}$/sec. and

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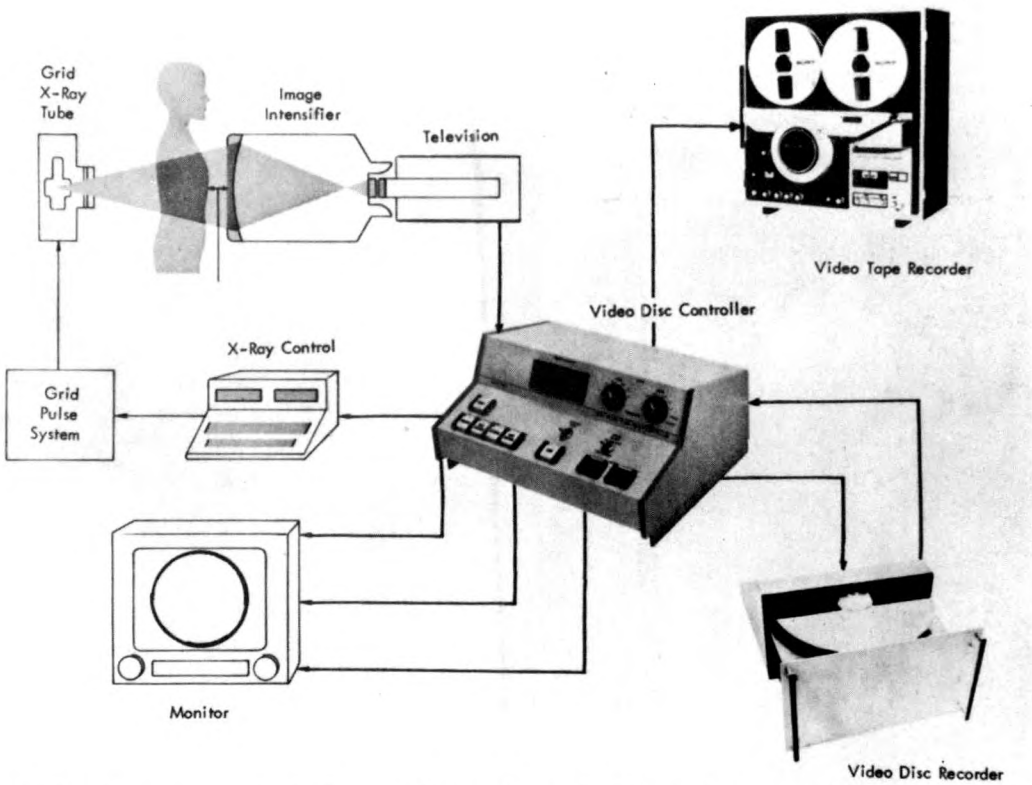


FIG. 1. The video disc recorder is the central controlling unit of the entire system. The video tape recorder is desirable only because of the limited storage capabilities of our disc recorder. With increased capabilities the tape recorder may become unnecessary.

single pulses. These pulses energize the grid-controlled generator to produce x-rays instantly on command of the disc recorder at the selected programmed time intervals (Fig. 3). After a $\frac{1}{60}$ second recording cycle, the disc recorder is switched automatically from record to playback mode. The previously recorded picture is then displayed on the television viewing monitor until the system is recycled automatically at the preset pulse rate. Because this system displays 1 of the 2 fields twice for each presented picture on the viewing monitor, a 945-line, 30 frames/sec. television system is necessary to deliver acceptable resolution.

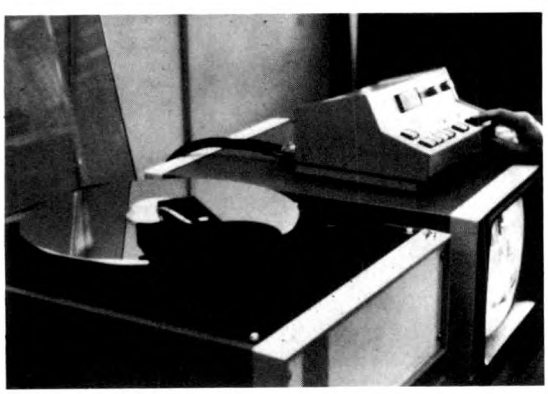


FIG. 2. Video disc recorder with control module.

Dosimetry during low pulse-rate fluoroscopy at various rates has been determined and reported previously.[2] In brief we have found a greater than 50 per cent reduction in dose between standard fluoroscopy performed without the disc recorder and low pulse-rate fluoroscopy of 15/sec. (Table 1). It can be seen that when using the pulsed mode at 15/sec. the dose is reduced to approximately 35 per cent of that during the standard mode. Subsequent decreases in the pulse rate by one-half decreases the dose by almost exactly 50 per cent in each case.

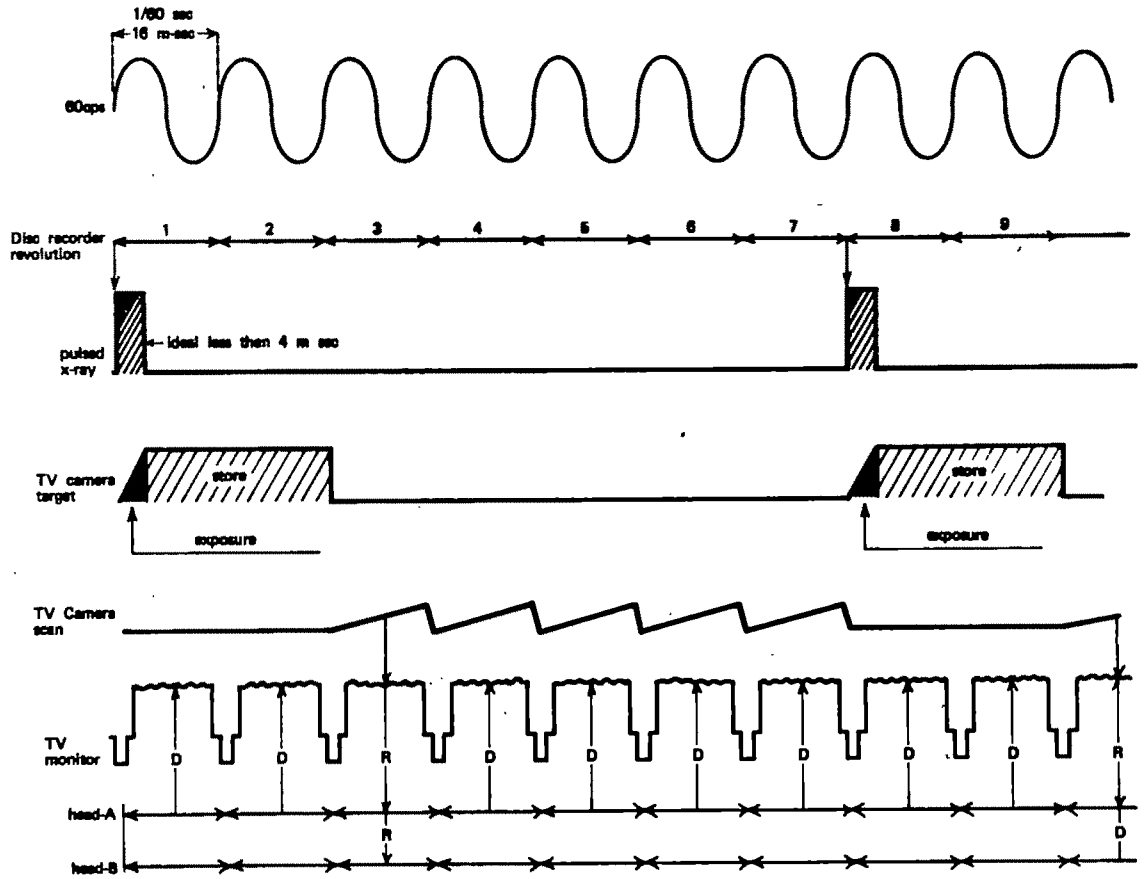


FIG. 3. Time sequence during low pulse-rate fluoroscopy. At the onset of revolution 1 the disc recorder turns the x-ray generator on and the scan of the TV camera off. At revolution 3 the image information is scanned off the TV camera tube target and recorded (R) on the magnetic disc (1 field only of the 2 fields). The image recorded during revolution 3 is displayed (D) from the disc during revolutions 4 through 10. With revolution 8 a new cycle starts; *i.e.*, radiation at 8, recording at 10, and display from 11 through 17. The 3/60 second delay from radiation pulse to image display is not noticeable during actual fluoroscopy. Two heads are necessary to provide a flicker-free image. (Reproduced with permission of *Radiology*.[4])

TABLE I

RADIATION DOSE AT VARIOUS SITES

(85 kvp., 8 ma.)

Rate (pulses/sec.)	Patient Entrance Dose (rad/min.)	Patient Exit Dose (mrad/min.)	Scatter Exposure 2 Feet from Intensifier Gonadal Height (mr/min.)	Scatter Exposure 2 Feet from Intensifier Eye Level Height (mr/min.)
Standard	3.6	29	2.1	1.5
15	1.2	11	0.74	0.59
7 1/2	0.58	5.7	0.36	0.30
3 3/4	0.28	2.9	0.18	0.14
1 7/8	0.13	1.5	0.08	0.08
15/16	0.06	0.7	0.04	0.04

CLINICAL RESULTS

Our initial application of low pulse-rate fluoroscopy to cardiac catheterization and coronary arteriography was with the 15 pulses per second mode. As reported previously, we found both subjectively and objectively that there was no increased difficulty in the performance of these procedures with 15 pulses per second as compared to standard fluoroscopy.[4] The procedures were performed both from the brachial artery using the more difficult Sones' technique,[10] and the femoral artery using the relatively easier Judkins' technique.[5] More important, there was no prolongation of the duration of the procedure by using the 15 pulses per second mode. The estimated average reduction of dose to patient and paramedical personnel from both fluoroscopy and angiography was approximately 50 per cent.

Subsequently we have utilized the $7\frac{1}{2}$ pulses per second mode for all routine fluoroscopic monitoring during the physiologic portion of the study and alternated every other case between $7\frac{1}{2}$ pulses and 15 pulses per second during the actual performance of the coronary arteriography study (Table II). There were 64 patients in each group with no significant difference being found in the procedural times between both groups of patients. Because those procedures in which the Sones' technique was employed were generally more difficult and potentially more time consuming, the patients in each group in which this technique was employed were compared. Again there was no difference in over-all duration of the procedure.

Table II

PROCEDURE DURATIONS WITH LOW PULSE-RATE FLUOROSCOPY IN 128 PATIENTS

Rate (pulse/sec.)	No. of Patients	Average Total Procedure Time
$7\frac{1}{2}+15$/sec.		
Total group	64	39 min.
Sones' technique	40	44 min.
$7\frac{1}{2}$/sec.*		
Total group	64	37 min.
Sones' technique	42	40 min.

* In 5 cases the angiographer requested a higher pulse rate.

During the manipulation of the catheter for coronary arteriography, fluoroscopy at $7\frac{1}{2}$ pulses per second was generally found acceptable. All the trainees have accepted this rate quite readily, although one of the regular staff members objected on occasion because he felt that he was missing certain movements. In the group in which the $7\frac{1}{2}$/second mode was used during the entire procedure there were 5 instances in which the angiographer requested increasing the pulse rate because he felt he was not seeing sufficient detail.

In Table III a comparison of the estimated dosage to the patient as well as scat-

Table III

CALCULATED X-RAY DOSAGE AT VARIOUS LOCATIONS FOR "AVERAGE" CARDIAC CATHETERIZATION WITH CORONARY ARTERIOGRAPHY USING THE 15/SEC. AND $7\frac{1}{2}$/SEC. MODES AND A COMBINATION OF BOTH MODES*

Mode	Patient Entrance Dose (rads)	Patient Exit Dose (rads)	Physician Scatter at Gonadal Height (mr)	Physician Scatter at Eye Level (mr)
Standard	153	1.3	95	67
15/sec.	57	0.56	39	31
15/sec. and $7\frac{1}{2}$/sec.†	45	0.46	33	25
$7\frac{1}{2}$/sec.	32	0.35	25	19

* Based on 40 minutes average fluoroscopy time plus 3,500 total cineframes exposed at 85 kvp. and 400 peak ma.[4]

† In this group the fluoroscopic time used in each mode was approximately equal, therefore the calculated dose values are based upon 20 minutes in each mode.

ter exposure may be found. Assuming a total of 3,500 cineframes per case and adding this to the radiation dose accumulated during 40 minutes of fluoroscopy, it can be seen that when using low pulse-rate fluoroscopy of 7½ per second the dose to the patient is reduced to approximately 20 per cent of that of standard fluoroscopy. Even in the group in which 7½ and 15 pulses per second were both used the radiation dosage was reduced to approximately 30 per cent of that of standard fluoroscopy.

DISCUSSION

Radiation exposure to both physicians and patient is at significant levels during cardiac catheterization.[6,11] It has been indicated by Riley *et al.*[9] that the majority of radiation exposure is obtained during performance of the arteriographic procedure itself, and Ardran and Fursdon[1] have challenged the significance of the level of radiation exposure during cardiac catheterization. I think, however, that the level is largely dependent on the type of procedure to be performed. There are certainly many procedures that involve only a very limited fluoroscopy time, such as might be seen in selective renal arteriography in which the technical difficulty in catheterizing the arteries is generally minimal. On the other hand during coronary arteriography and visceral angiography with superselective techniques, fluoroscopic times of 30 minutes are common with occasional times approaching 1 hour, particularly in training institutions. In addition, placing catheters for the performance of physiologic studies can add considerably to the radiation exposure level.

The results of this study have demonstrated that even a technically difficult procedure as coronary arteriography performed by the Sones' technique may be carried out using pulse rates of 7½ per second. The few times that this mode was unsatisfactory to the angiographer were generally in obese patients. In our catheterization laboratory the performance of the "average" total cardiac examination at 7½ pulses per second results in a dose rate to the patient of 20 per cent of that during standard fluoroscopy. Likewise, the exposure dose to the angiographer by scatter has been reduced to 25 per cent of the dose that would be obtained during the performance of the same examination using standard fluoroscopy. This reduction was obtained without adding significantly to the duration, risk or difficulty of the procedure. In an uncommon instant when the image is unsatisfactory to the angiographer, the pulse rate can immediately be increased. Attempts at low pulse-rate fluoroscopy in the 3¾ per second mode have been unsuccessful, partially because of lack of acceptance by the angiographers, but also because it is hard to monitor difficult catheter manipulations.

There are other advantages to the use of a video disc recorder in a fluoroscopic system. Stop-action electronic radiography is of great advantage in determining adequacy of injections and in making diagnostic decisions. The use of the disc recorder for electronic radiography opens a whole new field that may, at least in part, bypass conventional film techniques. A commonly noted advantage of the video disc recorder is its use for live subtraction techniques. I have not found this to be of any real clinical use even in the teaching situation, since the capable angiographer has a 3 dimensional concept of the vessels he is studying when looking at the fluoroscopic image and the novice quickly learns this concept. It is preferable for the inexperienced angiographer to develop this 3 dimensional feeling as well as the other techniques by practicing on dogs[8] and the human aortic model.*

There are some major disadvantages to the disc recorder, the most important of which is its cost. The video disc recorder† cost us over $25,000 including purchase and integration.‡ However, the cost is rapidly diminishing as occurred with the video tape recorders some years back. Newer disc recorders are now available at half the price.

* Medi-Tech, Inc., Watertown, Massachusetts.
† Data Disc, Sunnyvale, California.
‡ Picker, Inc., Cleveland, Ohio.

It will be important to increase their reliability and decrease their cost, so that their use for dose reduction can increase without adding too much expense to the already high cost of equipping a modern facility.

Another disadvantage is the limited storage capability of the video disc recorder, particularly as compared to the video tape recorder. Our disc is limited to a total storage of 300 frames. However, this too is being improved with disc units now available with storage of close to 600 frames.

Silicon storage tubes are less expensive than disc recorders and can be integrated into a pulsed x-ray system.[7] However, they are limited by storage of no more than 1 image at a time and low maximum pulse rate. It is doubtful that they will be of use in fluoroscopy during catheterization procedures, but will be of value in orthopedic and gastrointestinal fluoroscopy.[2]

The final disadvantage that we have noted with the video disc recorder has been the cost of its maintenance. It requires meticulous care in handling and careful control of its environment. This is in part a function of the newness of its use in the medical situation and is improving with technological advancements by industry.

SUMMARY

Dose reduction low pulse-rate fluoroscopy utilizing a magnetic video disc recorder integrated into an x-ray system has been employed successfully during the performance of cardiac catheterization and angiography for the past 2½ years.

A short x-ray pulse is recorded by the disc recorder and then played back immediately on the television viewing monitor. Subsequent pulses are recorded and played back automatically at a preset time interval resulting in a flicker-free, pseudo-continuous fluoroscopic image.

Pulse rates as low as 7½ frames per second have been used during coronary arteriography usually without difficulty, resulting in dose reduction over 5 times that which would have resulted during performances of these procedures with standard fluoroscopy.

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REFERENCES

1. Ardran, G. M., and Fursdon, P. S. Radiation exposure to personnel during cardiac catheterization. *Radiology*, 1973, *106*, 517–518.
2. Berci, G. and Steckel, R. J. Modern radiology in operating room. *A.M.A. Arch. Surg.*, 1973, *107*, 577–586.
3. Dorph, S., Mygind, T., Northeved, A., Okholm, B., Petersen, K. O., and Oigaard, A. A dose-reducing fluoroscopy system: dose measurements and clinical evaluation. *Radiology*, 1970, *97*, 399–403.
4. Grollman, J. H., Jr., Klostermann, H., Herman, M. W., Moler, C. L., Eber, L. M., and MacAlpin, R. N. Dose reduction low-pulse rate fluoroscopy. *Radiology*, 1972, *105*, 293–298.
5. Judkins, M. P. Selective coronary arteriography. Part I. Percutaneous transfemoral technique. *Radiology*, 1967, *89*, 815–824.
6. Malsky, S. J., Roswit, B., Reid, C. B., and Haft, J. Radiation exposure to personnel during cardiac catheterization: preliminary study. *Radiology*, 1971, *100*, 671–674.
7. Mistretta, C. A., Ort, M. G., Cameron, J. R., and Moran, P. R. Silicon storage tube: new method of image storage in fluoroscopy. *Invest. Radiol.*, 1972, *7*, 174–176.
8. Olmsted, W. W., Snow, H. D., Grollman, J. H., Jr., Steckel, R. J., and Jaco, L. M. New methods for teaching angiographic techniques to residents. *Invest. Radiol.*, 1972, *7*, 341–342.
9. Riley, R. C., Birks, J. W., Palocios, E., and Templeton, A. W. Exposure of radiologists during special procedures. *Radiology*, 1972, *104*, 679–683.
10. Sones, F. M., Jr., and Shirley, E. K. Cine coronary arteriography. *Mod. Concepts Cardiovasc. Dis.*, 1962, *31*, 735–738.
11. Wold, G. J., Scheele, R. V., and Agarwal, S. K. Evaluation of physician exposure during cardiac catheterization. *Radiology*, 1971, *99*, 188–190.

HYSTEROSALPINGOGRAPHY IN INFERTILITY*

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HYSTEROSALPINGOGRAPHY has proven to be an important diagnostic method in clinical gynecology for over 50 years. It is of particular value in the investigation of the uterine and tubal factors of female infertility.[2–7,11,13,14] Until recently the technique of the procedure has not been significantly modified. A simpler and less painful technique utilizing a flexible vacuum cervical adaptor is now available.[1,11,12] In spite of its advantage this technical modification has not gained the popular acceptance which it deserves.

The purpose of this paper is to report our experience with the newer modified technique for hysterosalpingography in the study of patients with female infertility.

MATERIAL AND METHOD

One hundred and forty-seven hysterosalpingographies have been performed during the past 3 years as a part of the diagnostic work-up of infertile patients. During the first year, hysterosalpingographies were performed in a conventional manner utilizing a cervical tenaculum, a rigid uterine cannula (acorn or screw tip) and an oily contrast material. Forty-one cases were studied by this technique. During the past 2 years a vacuum cervical adaptor (Malmström or Semm) and an aqueous contrast material were used. There were 106 cases in this group. In 6 instances the vacuum cervical adaptor could not be utilized because of the morphologic abnormality (*i.e.*, uterus didelphia). During the last 1½ years the Semm adaptor has been used exclusively (see Technique).

Patients were referred to the Radiology Department for hysterosalpingography by several gynecologists; however, 2 (who are also co-authors of this paper—R.E.C. and L.P.) have contributed 73 of the cases in the past 2 years. Complete clinical and laboratory records are available on each of these patients. Their up-to-date fertility status is known. This group is the source of the clinical analysis of the significance of the hysterosalpingography information.

TECHNIQUE OF HYSTEROSALPINGOGRAPHY

A. THE CONVENTIONAL METHOD

A detailed description of the conventional technique is not warranted. It has been in use for the past half century and, even today, is utilized by the majority of clinicians. The significant differences of the conventional technique in contrast to the newer modifications are: (1) the use of a cervical tenaculum to provide countertraction against an intrauterine cannula; (2) use of a rigid intrauterine cannula (acorn rubber or screw tip); (3) an oily contrast material; (4) 2 roentgenograms following low (3 to 4 cc.) and high (10 cc.) volume contrast material injection, often obtained without fluoroscopic monitoring, and a third roentgenogram of the abdomen obtained 24 hours later.

B. THE VACUUM CERVICAL ADAPTOR TECHNIQUE

The essential items of equipment needed are: (1) a vacuum cervical adaptor (Semm or Malmström)* (Fig. 1, a); (2) a cervical adaptor application clamp (Fig. 1, b); (3) a controlled pressure hand vacuum pump; (4) a removable vaginal speculum (Fig. 1, c); (5) water soluble contrast material (Sinografin or Salpix)† (Fig. 1, d); (6) an

* Semm hydrotubation cup (portio-adaptor), WISAP, GmbH, 8 München 71, Germany.
Malmström (AB vacuum extractor), Götaborg, Sweden.

† Sinografin (meglumine diatrizoate and meglumine iodipamide injection—38% iodine), E. R. Squibb & Sons, Inc., Princeton,

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P.Q., Canada, September 25–28, 1973.
From the Department of Radiology,† and the Department of Obstetrics and Gynecology,‡ Indiana University Hospital, Indianapolis, Indiana.

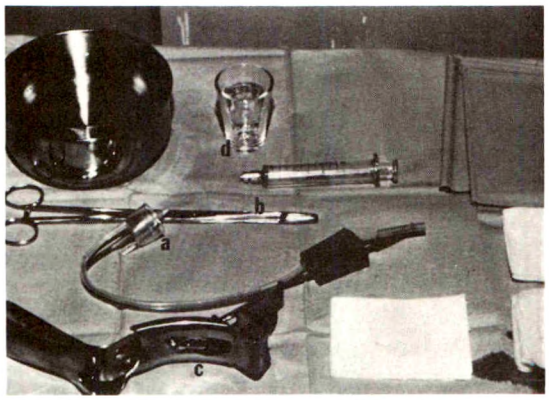


FIG. 1. *Instrument tray for hysterosalpingography.* a. Vacuum cervical adaptor (Semm); b. cervical adaptor application clamp; c. removable vaginal speculum; d. water soluble contrast material. A disposable, presterilized cervical adaptor has 2 tubes connected to its cup. One that opens to the central cone is for the contrast material injection. The other tube is to be connected to a controlled pressure vacuum pump. Application of this adaptor does not require a cervical tenaculum or countertraction against an intrauterine cannula which is needed for the conventional technique.

image intensified fluoroscopic table with a small focal spot tube and a high ratio reciprocating grid.

The Semm cervical adaptor is favored because of the flexibility of its connecting tubes. This allows a wider range of movement of the patient during the examination such as turning to the prone position. The Malmström vacuum pump is employed because it has a glass bottle trap which prevents the vacuum pump from becoming clogged with tissue debris, mucus, etc.

The patient is placed in a lithotomy position at the foot of the fluoroscopic examining table. Usual perineal and vaginal preparation as for a sterile pelvic examination is made. A removable vaginal speculum is placed and the cervix is brought to full view so its external os is identified. The vacuum cervical adaptor is presterilized. The contrast material injection tube in the center of the cup is completely filled with water soluble contrast material which has been warmed to the body temperature. The hand operated vacuum pump is connected to the vacuum suction tube opening at the base of the cup of the cervical adaptor. The cervical adaptor is placed in position over the cervix and vacuum is applied (Fig. 2). This eliminates the need for the cervical tenaculum. The vaginal speculum is then removed. Under fluoroscopic monitoring, small fractions of contrast material are slowly injected. Overt pressure is not used. Tightly coned down cervical-uterine canal spot roentgenograms are obtained in the anteroposterior and oblique positions (4 on 1 film; Fig. 3, *A–D*). The use of a small volume (about 2 cc.) of a water soluble contrast material makes it possible to demonstrate small lesions often obscured by a larger quantity of oily contrast material. An additional 4 to 5 cc. of contrast material is then injected to fully distend the uterine cavity and the full length of the fallopian tubes. The injection is performed slowly and fluoroscopically monitored. This helps to prevent uterine or tubal spasms. When the uterus and tubes are well opacified, spot roentgenograms of the pelvis are obtained in the anteroposterior and oblique projections (Fig. 3, *E–G*). Fluoroscopic

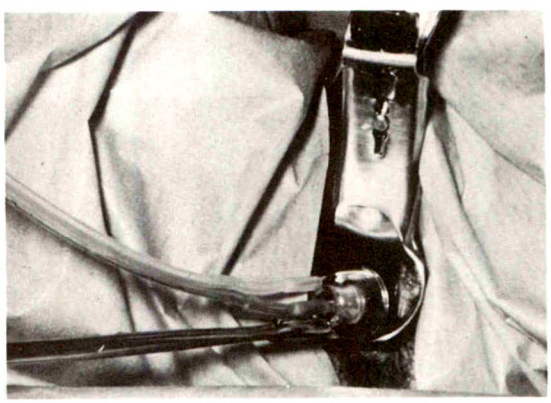


FIG. 2. *Cervical adaptor being applied.* The transparency of the cup of the cervical adaptor permits identification of the cervical os during its application. The vaginal speculum is readily disassembled and removed. Rotations and turning of the patient's position during the examination are freely accomplished without discomfort.

Salpix (sodium acetrizoate—53% iodine,) Ortho Pharmaceutical Corporation, Raritan, New Jersey 08869.

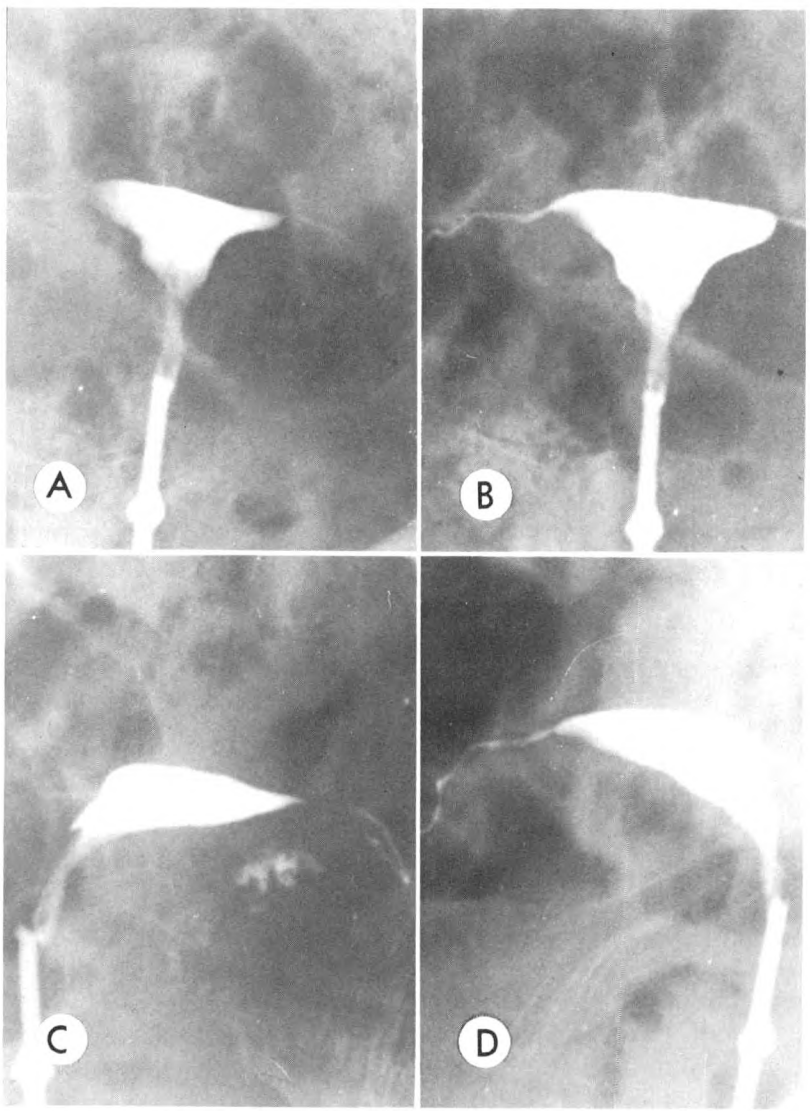


FIG. 3. *Normal examination (full series).* (*A—D*) Composite of 4 uterine body spot roentgenograms with low volume of contrast agent (2–3 cc.) injected.

monitoring eliminates lack of filling or overfilling of important structures. The average total fluoroscopic time is 2 to 3 minutes. Peritoneal spillage of the contrast material is readily demonstrated and a delayed 24 hour roentgenogram has been unnecessary. The length of time from preparation to termination of the procedure is approximately 20 to 30 minutes.

RESULTS

With the conventional technique the results were inconclusive in a number of cases. The presence or absence of an organic lesion cannot be ascertained with nonvisualization of one or both of the fallopian tubes. During fluoroscopic observation it is frequently observed that one or both tubes are functionally obstructed to the flow of contrast material. A few minutes rest and relaxation of the patient eliminates this process. Had the examination been terminated prior to rest and repeated attempts,

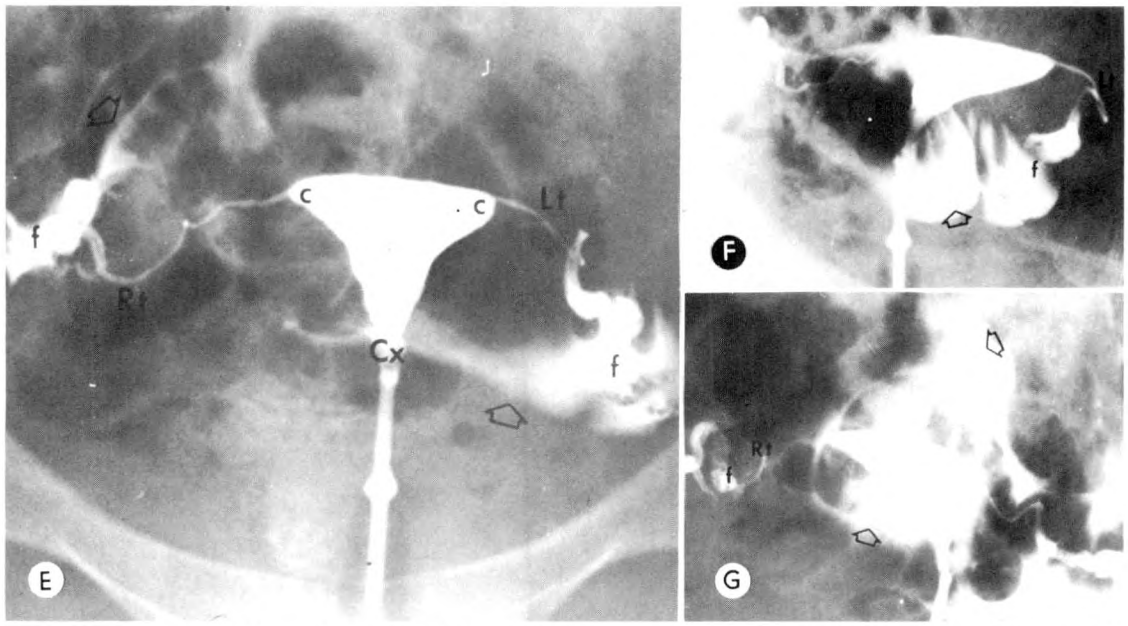


FIG. 3. (*E*) Medium volume. Cx—Internal cervical os; c—cornua of the uterus; Rt and Lt—right and left fallopian tubes; f—fimbriated portion; arrows—contrast spilling in peritoneal cavity. (*F* and *G*) Left posterior oblique and right posterior oblique views. Oblique views are needed for a better appreciation of the axis and the configuration of the uterine cavity. Note a generous intraperitoneal spilling of the contrast material. An air tight seal around the cervix by the vacuum cervical adaptor eliminates the intravaginal loss of the contrast material. Unless the uterine cavity is very large, 10 cc. of contrast material is usually quite sufficient. The roentgenograms obtained are all fluorographic spot roentgenograms.

an organic obstructive lesion of the fallopian tubes would have been incorrectly thought to be present.

Utilizing the conventional technique with a rigid cervical adaptor, the cervical os is not as well sealed off from the vagina as with the vacuum cervical adaptor cup. Spillage of contrast material back into the vagina was much more commonly seen. There were also cases in whom one or both of the 2 roentgenograms obtained were not properly positioned to cover all pertinent structures. Because of these technical discrepancies, clinical statistics would be meaningless. The following statistics were taken only from those 73 cases mentioned earlier whose records are complete and fertility status up-to-date.

Of the 73 cases 44 (60 per cent) were normal on hysterosalpingography. Eighteen of these cases (41 per cent) became pregnant between 1 to 8 months after the hysterosalpingography. Seven cases became pregnant within the first month following hysterosalpingography.

Abnormalities demonstrated by the examination were: (1) 4 cases of unilateral tubal obstruction, 1 of these patients became pregnant within 1 month after the hysterosalpingography; (2) 5 cases of bilateral tubal obstruction (Fig. 4, *A* and *B*); (3) 6 cases of unilateral ovarian or adnexal masses (Fig. 5, *A* and *B*), 1 of these patients became pregnant 4 months after the hysterosalpingography; (4) 3 cases of bilateral ovarian or adnexal masses (Fig. 6, *A* and *B*); (5) 4 cases of intrauterine filling defects by a myoma or a polyp (Fig. 7, *A–C*), 1 became pregnant 1 month after the hysterosalpingography; (6) 1 case of congenital deformity and dysplasia of the genitalia (Fig. 8, *A–D*); (7) 3 cases had a combination of 2 or more abnormal findings (Fig. 9, *A* and *B*); (8) 3 cases in which the

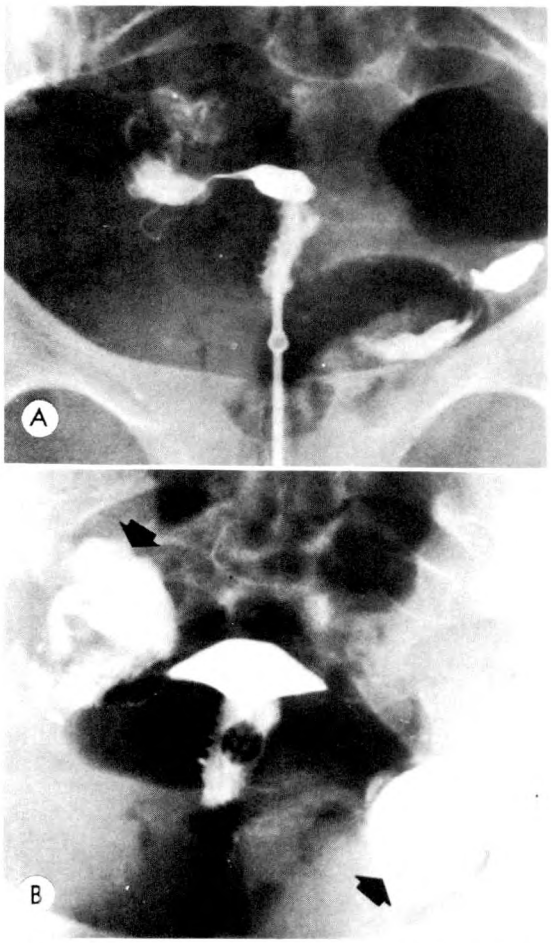


FIG. 4. *Bilateral hydrosalpinx.* (*A*) Moderate volume filling fails to disclose the presence of a bilateral hydrosalpinx. (*B*) Large, bulbous dilatation at the end of each tube (arrows) without apparent peritoneal spilling of the contrast material is well demonstrated later with a high volume filling.

diagnosis was uncertain because of a technically incomplete study (Table 1).

DISCUSSION

An ideal special radiologic procedure should be accurate, technically simple and have a minimum of patient morbidity. The vacuum cup technique of hysterosalpingography approaches this ideal.

The vacuum cervical adaptor better serves the combined functions of the cervical tenaculum and the intrauterine cannula which are still widely utilized for hysterosalpingography. It is much less painful than the combined instrumentation and allows excellent visualization of the cervical canal. Patients may note a slight *cramping sensation* when the vacuum cup is attached to the cervix. This normally spontaneously resolves in a few minutes. We have not encountered a single case of *peritoneal-irritation* caused by the water soluble contrast material spilling into the pelvic peritoneal cavity (Sinografin was used exclusively for these studies). In rare instances, pelvic *cramps* lasted longer than a few minutes. This was more frequent in nulliparous pa-

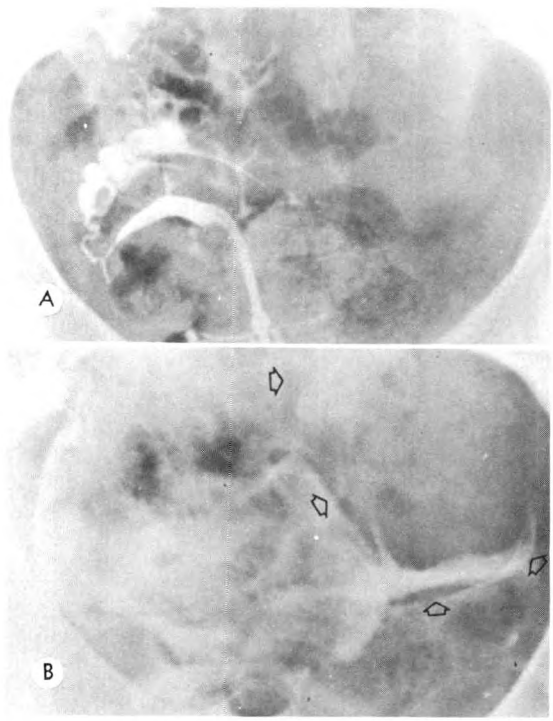


FIG. 5. *Pelvic kidney.* (*A*) Moderate volume contrast material filling and (*B*) postevacuation roentgenograms demonstrate patent right tube, a left pelvic mass (arrows) with the right side deviation of the uterine axis and nonvisualization of the left tube. The left tube was found to be hypoplastic. The patient had multiple other urogenital anomalies. The same size mass in the pelvis, if intraperitoneal, will have a better chance of having more of its surfaces delineated by the intraperitoneal contrast material. Small intraperitoneal "*masses*" such as small-bowel loops frequently mimic a normal ovary (see Fig. 6, *A* and *B*).

tients (primary infertility). These patients' symptoms subsided shortly after the removal of the vacuum cervical adaptor. The intensity of discomfort is much less than the combination of a tenaculum and intrauterine cannulation. Of 106 cases studied with the vacuum cup method, only 3 required an analgesic agent or tranquilizer. There were individuals of low pain threshold who had marked symptoms even during preparation of the perineum and vagina prior to the examination.

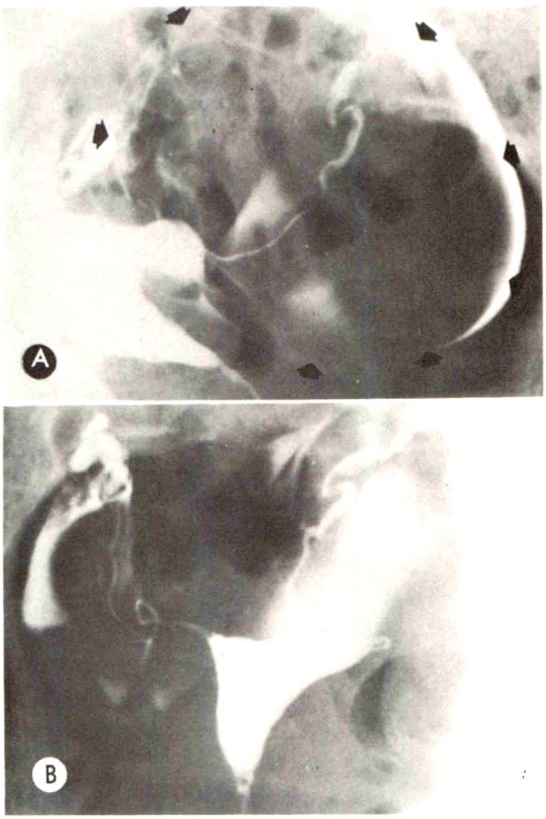


FIG. 6. *Simple ovarian cysts.* (*A*) Left posterior oblique and (*B*) right posterior oblique views with high volume contrast material filling demonstrate a $7\frac{1}{2} \times 10\frac{1}{2}$ cm., nonlobulated, smooth surfaced cystic left ovary (arrows) and a smaller, incompletely delineated right ovarian mass (*B*). These were exogenous hormone-induced simple cysts. Note that the tubes are entirely patent. When more than half of a circumference of a pelvic mass comes into contact with the contrast material which has spilled into the peritoneal cavity, the over-all size and shape of the mass become obvious.

It has long been debated whether or not hysterosalpingography has any therapeutic effect on certain conditions of female infertility.[8–10,16] This series is too small to be conclusive; however, in this small patient sample the conversion to fertility from an infertile status was noted in a number of patients whose hysterosalpingograms were normal.

Hysterosalpingography does not consistently visualize the ovaries; however, an abnormally enlarged ovary is frequently outlined when a generous quantity of contrast material has freely spilled into the pelvic peritoneal cavity. A mass outlined in the region of the ovary may have multiple etiologies. The combination of hysterosalpingography and pelvic pneumography or laparoscopy is necessary if investigation of the status of the ovaries is to be made.[13,15]

On 3 occasions the combination of an ovarian or adnexal mass, partial loculation of the contrast material in the vicinity of the mass and elsewhere in the Douglas pouch, and ipsilateral tubal obstruction or peritubal adhesions was present (Fig. 9, *A* and *B*). These patients had never had a clinical episode of pelvic inflammatory disease or pelvic operation. Extrauterine endometriosis was strongly suspected and subsequently proven by laparoscopy or laparotomy. However, on 3 other occasions, a normal hysterosalpingogram was present with subsequently proven extrauterine endometriosis.

SUMMARY

1. A detailed description of hysterosalpingography utilizing a vacuum cervical adaptor is given.

2. The authors' experience with this technique is briefly stated.

3. The vacuum cervical adaptor modification of the hysterosalpingographic technique is a significant improvement and merits wider acceptance because of its technical simplicity, assurance of thorough-

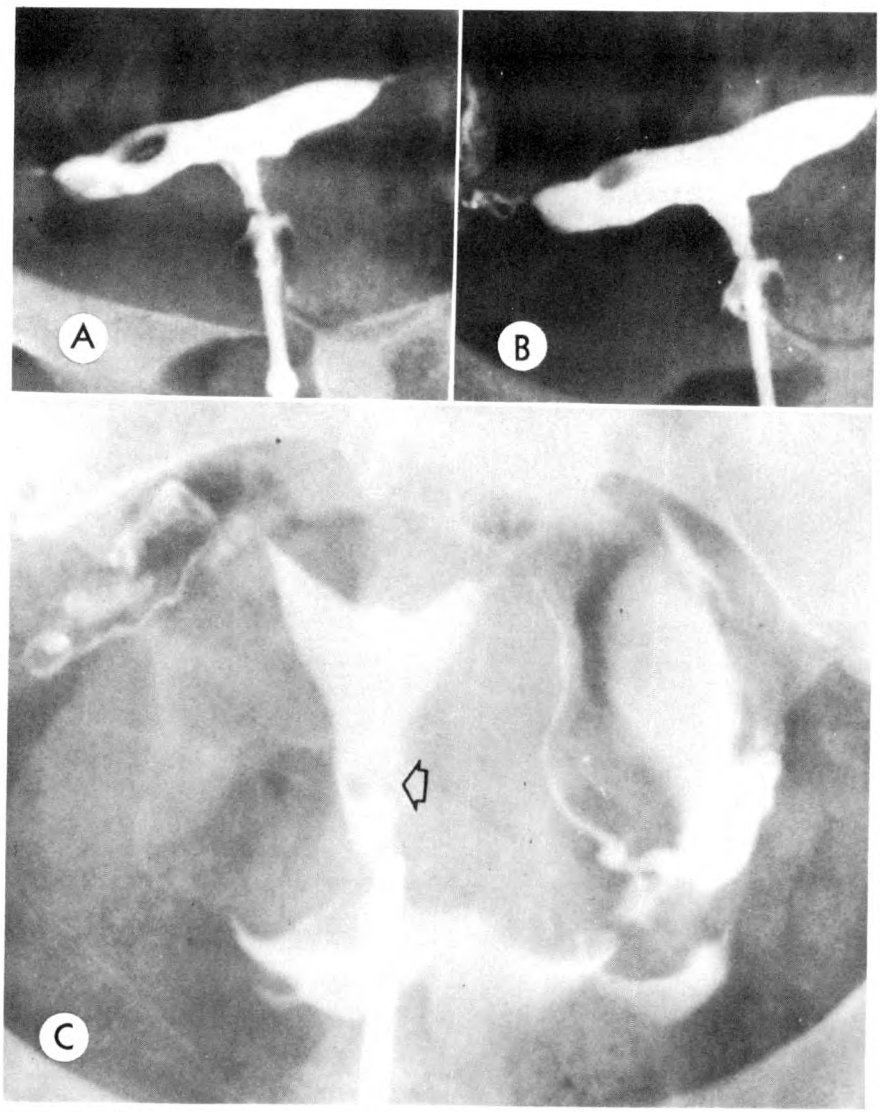


FIG. 7. *Polyps.* (*A* and *B*) Low volume contrast material filling of the uterine cavity outlines a solitary polypoid lesion on the right side fundus portion which is less than 1 cm. in its longer dimension. (*C*) A small polypoid lesion is present in the supracervical region of the uterine cavity. Subsequent high volume filling with contrast material has obscured these lesions completely. Without preliminary low volume filling under fluoroscopic control, especially using a less diffusable contrast agent, a small polypoid lesion can be easily missed.

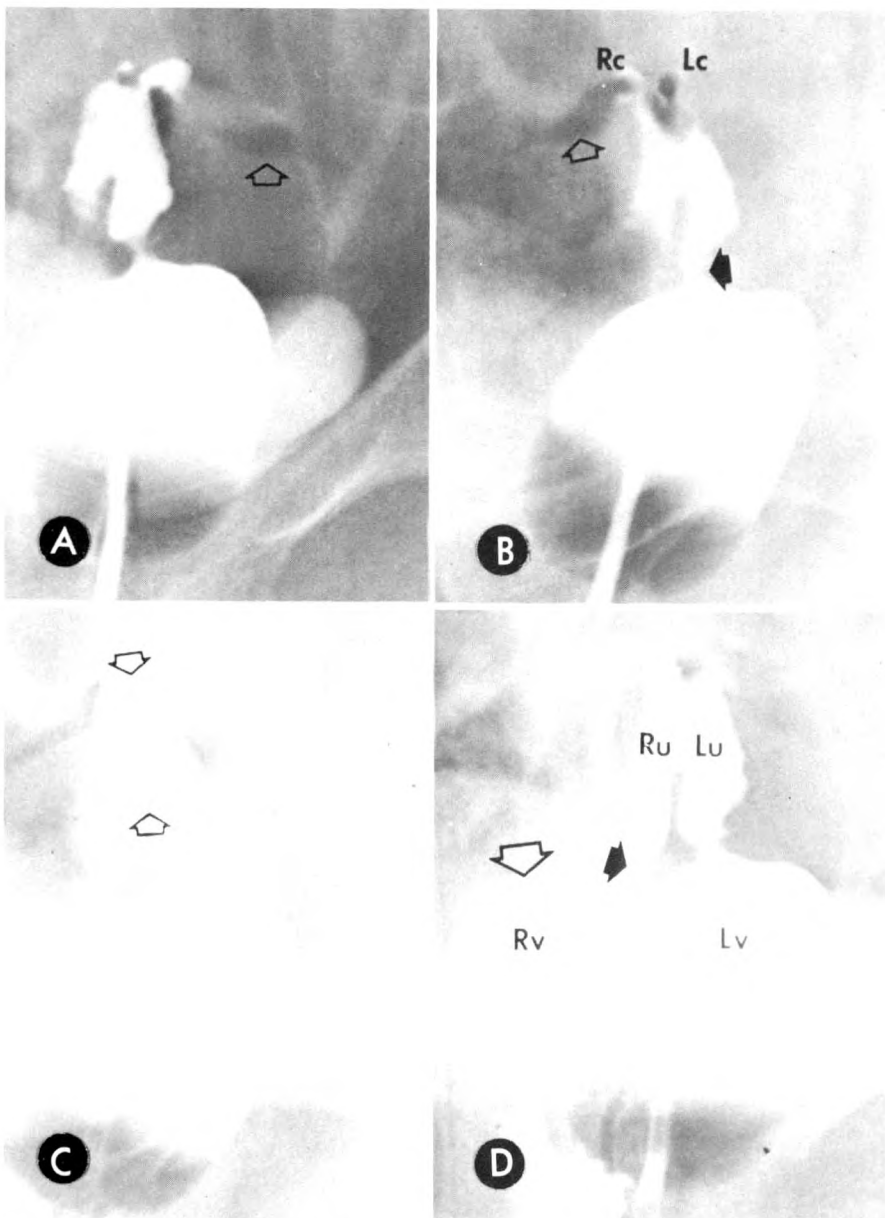


Fig. 8. *Complete duplication of genitalia.* (*A—D*) A sagittal septum divides the vagina to the left and the right halves, each leading to a separate cervical os (solid arrows in *B* and *D*). A Foley catheter was placed in the left vagina and the contrast material was injected. Rc and Lc—right and left cornu; Ru and Lu—right and left uterine cavity; Rv and Lv—right and left vaginal cavity; open arrows in *A* and *B*—air in fallopian tubes; open arrows in *C*—communication between Ru and Lu; open arrow in *D*—right vaginal fornix.

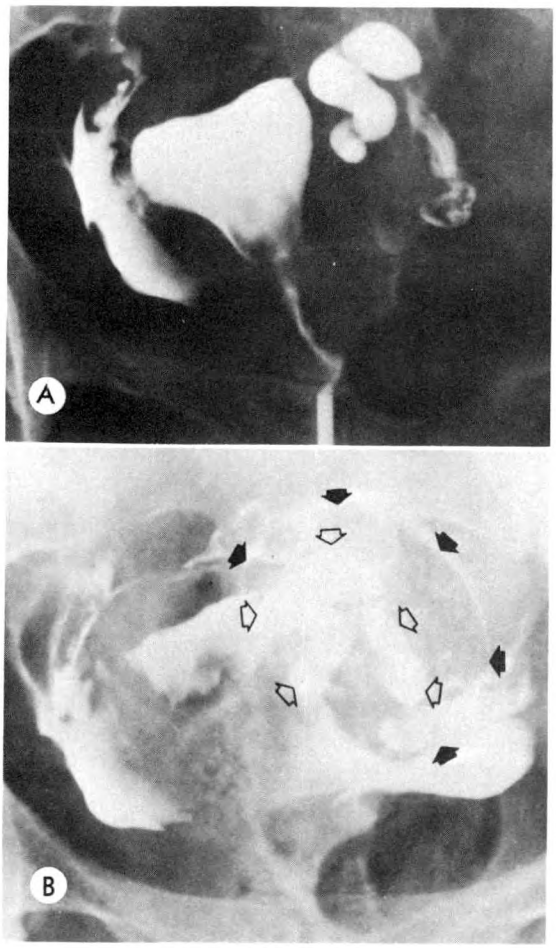


FIG. 9. *Peritubal-pelvic adhesions and pelvic masses.* (*A* and *B*) The right fallopian tube is patent and the contrast material spills freely into the peritoneal cavity from this side. The left tube is obstructed or very severely strictured at its fimbriated end resulting in a "*sausage-link*" like marked dilatation and elongation of the entire length of the tube (open arrows). A smooth surfaced, oval mass in the left adnexal region (solid arrows) spontaneously disappeared in 2 weeks after cessation of the ovulation-inducing agent (Clomid) medication. This enlargement was most probably a functional corpus luteum cyst. Subsequently a laparotomy was performed which disclosed pelvic and ovarian endometriosis. The combination of a strictured tube or peritubal adhesions and pelvic or ovarian masses very strongly suggests endometriosis.

TABLE I

RESULTS OF HYSTEROSALPINGOGRAPHY IN 73 INFERTILE FEMALES

	No. of Patients	Became Pregnant
Normal	44	18
Unilateral tubal obstruction	4	1
Bilateral tubal obstruction	5	0
Unilateral adnexal or ovarian mass	6	1
Bilateral adnexal or ovarian mass	3	0
Polyps or myomas	4	1
Congenital dysplasia of internal genitalia	1	0
Combination of more than 2 minor abnormalities	3	0
Inconclusive study	3	0
Total	73	21

ness and significant reduction of pain and discomfort.

4. Hysterosalpingography remains a principal method in the study of female infertility.

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REFERENCES

1. AARO, L. A., and STEWART, J. R. Hysterosalpingography with image-intensified fluoroscopy. *Am. J. Obst. & Gynec.*, 1969, *105*, 1124–1128.
2. AVNET, N. L., and ELKIN, M. Hysterosalpingography. *Radiol. Clin. North America*, 1967, *5*, 105–120.
3. BARWIN, B. N. Hysterosalpingography in infertility. *Ulster M. J.*, 1971, *41*, 61–65.
4. BEHRMAN, S. J., and POPPY, J. H. Hysterosalpingography. *Canad. M. A. J.*, 1957, *77*, 938–943.
5. BUXTON, L., and SOUTHAM, A. Critical survey of present methods of diagnosis and therapy in human infertility. *Am. J. Obst. & Gynec.*, 1955, *70*, 741–752.
6. CRON, R. S. Hysterosalpingography and infertility. *Australian & New Zealand J. Obst. & Gynec.*, 1965, *5*, 12–17.
7. GEARY, W. L., HOLLAND, J. B., WEED, J. C., and WEED, J. C., JR. Uterosalpingography. *Am. J. Obst. & Gynec.*, 1969, *104*, 687–692.
8. GILLESPIE, H. W. Therapeutic aspect of hys-

terosalpingography. *Brit. J. Radiol.*, 1965, *38*, 301–302.
9. Mackey, R. A., Glass, R. H., Olson, L. E., and Vaidya, R. Pregnancy following hysterosalpingography with oil and water soluble dye. *Fertil. & Steril.*, 1971, *22*, 504–507.
10. Palmer, A. Ethiodol hysterosalpingography for treatment of infertility. *Fertil. & Steril.*, 1960, *11*, 311–315.
11. Parekh, M. C., and Arronet, G. H. Diagnostic procedures and methods in assessment of female pelvic organs, with specific reference to infertility. *Clin. Obst. & Gynec.*, 1972, *15*, 1–104.
12. Parekh, M. C., Murthy, Y. S., and Arronet, G. H. Vacuum cervical adaptor. *Obst. & Gynec.*, 1970, *36*, 940–943.
13. Parekh, M. C., Murthy, Y. S., Kosasa, T., and Arronet, G. H. Validity of hysterosalpingography and pneumohysterosalpingography as evaluated by subsequent laparotomy. *Surg., Gynec. & Obst.*, 1972, *135*, 921–924.
14. Siegler, A. M. Hysterosalpingography. Harper & Brothers, New York, 1967.
15. Stern, W. Z., and Wilson, L. Pelvic pneumography with simultaneous hysterosalpingography. *Radiology*, 1970, *96*, 87–92.
16. Weir, W. C., and Weir, D. R. Natural history of infertility. *Fertil. & Steril.*, 1961, *12*, 443–451.

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EDITORIAL

HEURISTIC GEMS FROM THE AMERICAN RADIUM SOCIETY

FORTY years ago, July 4, 1934, Marie Sklodowska Curie died: martyr of the discovery she made for the inestimable benefit to suffering humanity.

Republication of the memorial tribute paid to her by this JOURNAL, through the pen of E. Walter Hall,[1] its illustrious contributor for many years, is more than appropriate, since the American Radium Society was named after "radium," the agent discovered by her and her husband (Fig. 1) and, symbolically, which has become the standard of nearly all forms of radiation used in therapy.

The memorial tribute, in its entirety, is as follows:

Madame Curie has ceased her labors. The test tube, the crucible, the pen, have fallen from her weary hands and she has entered on a well-earned rest. The world was startled when it learned that so great a life had ended. It paused a moment in shocked surprise—then, all the great and humble seemed to join in mourning and praise of her. However, words are weak and praise seems futile; her life and work speak for themselves in glowing terms that all can understand.

Marie Sklodowska was born in Warsaw, Poland, November 7, 1867, the youngest of five children. Hers was an unusual heritage, for her father, Ladislas Sklodowska was a physicist and professor in the Warsaw Lycée, and her mother, Bronsitawa Boguska was principal of a school for girls. As a child she played with test tubes and crucibles and soon showed such a keen mind and developed such studious habits that at an early age she became her father's chief assistant. At seventeen, she assumed her first teaching position as a governess.

Her independent spirit and her strong love of her native land led her into trouble with the Russians then ruling Poland. She was one of a group of young people encouraging Polish national spirit and making a determined effort to preserve the Polish language. It was necessary for her to leave Poland, so she went to Crakow, then under Austrian rule, thence to Paris where at the age of twenty-four she entered the University of Paris to work toward a doctorate. For five years she lived in a garret, carrying her own coal up five flights of stairs and allowing herself only ten cents a day for food. She was indeed happy to be given a job washing bottles in a laboratory and cleaning the furnace. Despite her humble occupations her spirit and her work attracted the attention of the great physicist, Lippmann, and the mathematician Poincaré who, with her father's assent and influence, had her assigned to the personal tutelage of the young physicist, Pierre Curie, the son of a Paris physician. In 1895 after years of constant association in scientific study, Marie and Pierre were married. They continued their studies as before. P. Curie was an instructor in a Paris Municipal School for Chemistry and Physics on rue Lohmand, and Marie obtained a position as a teacher in a girls' school in Sèvres. Their study and research work were carried on at night in the poorly equipped laboratory of the municipal school.

Roentgen had at this time just announced his discovery of the x-ray and the whole scientific world was started on a new set of experiments. Henri Becquerel, working with uranium salts, found by accident that some of his photographic plates were fogged, though pro-

[1] HALL, WALTER E., M.D. Editorial. Marie Sklodowska Curie. AM. J. ROENTGENOL. & RAD. THERAPY, 1934, *32*, 395–398.



Fig. 1.*

tected from ordinary light. He next discovered the ionizing effect of uranium on air and found that the effect was proportional to the strength of the rays. He then called in his friends, the Curies, as expert analysts to aid him in determining the nature and source of the radiant energy.

After verifying the experiments of Becquerel, Marie and Pierre started a set of experiments of their own, using an electrometer designed by Pierre to which Marie added a suitable ionization chamber. With her characteristic patience and thoroughness she put almost every known element and compound to the electroscopic test and found that only compounds of thorium and uranium manifested this ionizing power, to which she gave the name "radioactivité." In the course of her experiments she found that pitchblende, from which uranium was obtained, was a more powerful ionizing agent than could be accounted for by its uranium content and immediately concluded that pitchblende must contain some other radiant substance. She set herself to the task of finding that source of energy.

A huge task it was, for even the richest pitchblende contained only traces of radiant material. Through the influence of the Vienna Academy of Sciences a ton of pitchblende was placed at her disposal, to handle which it was necessary to rent an old abandoned warehouse for use as a laboratory. It had a patched glass roof which leaked when it rained, an old asphalt floor, and was poorly ventilated, being hot in summer and scantily heated in winter by a cast iron stove. Its furnishings consisted of three old deal tables, and on these Madame Curie prepared and mixed her chemicals, using crude apparatus that would be scorned by modern chemists. Enormous physical labor was required, for each part of pitchblende analyzed required five parts of added chemicals and about fifty parts of water. We wonder at the scientific zeal of this frail woman who could teach by day and spend many evening hours and much of the night in such exhausting mental and manual labor, at the same time maintaining a home. The work of those years with its brilliant results is the more remarkable due to an intervening pregnancy

* These photographs are revered memorabilia of the Editor from his days at the Fondation Curie and at the University of Paris.

and the birth of her first daughter in 1897. Small wonder Irene Curie Joliot has already become a scientist of note, following in the footsteps of her illustrious mother!

After many months of laborious and tedious crushing, dissolving, precipitation, and distillation, with fractional crystallization and painstaking ionization tests of each product at every step of the process, a thimbleful of white salt was obtained which was very strongly radioactive. From this Madame Curie isolated in July, 1898, a substance which she named "polonium," in honor of her beloved native land. On December 26, 1898, she announced the discovery of an element emitting two million times as much radiant energy as uranium and to which she gave the name "radium."

Madame Curie did not rest idly on her laurels and enjoy the fruits of her scientific discoveries. Her life work had only begun. She and her husband set about experimenting with the new-found substances, determining their properties and actions and evolving new and better methods of isolating them from the mother ores. Her experiments furnished the basis for her final thesis for her doctorate submitted in 1903. She and Pierre investigated and published papers concerning induced radioactivity, radium emanation, heat emission by radium and numerous other purely scientific studies, and began to investigate the physiological effect of radium rays. P. Curie intentionally produced a strong reaction on his own arm and they began a long series of experiments with animals to determine biological effects, which really laid the foundation for modern radium therapy.

In 1904 a second daughter Eve, now a dramatist and pianist of considerable talent, was born, and with slightly better financial circumstances it seemed that Madame Curie might be able to devote more time to her home and family. However, when the child was but two years of age Pierre Curie met a tragic death in a traffic accident and his wife was deprived of her dear companion and coworker and was left to carry on the struggle alone. She accepted his chair of physics at the Sorbonne where she continued his lectures and also carried on her experimental work at the Institut Curie—her private laboratory, established in 1912.

When the World War broke out, Marie Curie offered her services to France. She closed her Institut and with her daughter Irene and several student followers, put her knowledge of radiography to excellent use in hospitals near the battle front. She suggested the use of portable roentgenographic units in ambulances and was the means of saving many lives by early localization of foreign bodies.

Though shunning publicity, for the sake of science and humanity she wrote many papers and delivered many lectures. Among her writings are "Recherches sur les propriétés magnétiques des aciers trempes," "Recherches sur les substances radioactives," "L'isotropie et les eléments isotropes" and "Pierre Curie." Her most important work, however, is the scientific classic: "Traité de radioactivité" published in 1910. Madame Curie was a product of the past generation and her scientific theory was of the old school. Her discoveries and evolved theories, however, formed the starting point for the brilliant new school of scientific theorists. Her belief that radioactivity was a property of all matter, that all elements originated from radioactive elements instead of being distinct and indestructible, provided a basis for the modern ideas regarding the structure of the atom with its protons and electrons.

To few persons is it given to achieve greatness; to fewer still to have that greatness recognized and acclaimed by the entire world during their lifetime. To Marie Sklodowska Curie came not only the usual reward of work well done, to her came the rare privilege of seeing her labors bear great fruit in advancing the border of scientific knowledge as well as in being put to practical use in the world of industry and in the alleviation of human ills. And the world was not slow or niggardly in honoring the Curies. The street in Paris on which they lived was renamed rue Pierre Curie, and many institutions bear their name. The literature of radium physics incorporates their name as a unit of measurement, the

commonly used "millicurie," and radium therapy is properly called "curie therapy."

In 1901 Pierre Curie was awarded the Lacaze prise of 10,000 francs. In 1903 they received the Davy medal of the Royal Society of London. In 1904, they shared with Becquerel the Nobel Prize in Physics. Pierre was made a full Professor at the Sorbonne and in 1905 was elected to the French Academy of Sciences. In 1911 Madame Curie received the Nobel Prize in Chemistry, the only person ever to have been so honored a second time, and was also elected to the French Academy. In 1922 she was elected to the Academy of Medicine of Paris.

She returned to Warsaw for the first time in 1913, receiving signal honors. She visited the United States first in 1921 and again in 1929, each time being overwhelmed with ovations and laudatory introductions at receptions in her honor. Honorary degrees were bestowed upon her by Columbia, Yale, Northwestern and St. Lawrence Universities, the Universities of Pennsylvania and Pittsburgh, Woman's Medical College and Wellesley and Smith Colleges. In 1926 she delivered a series of lectures in South America, receiving the usual acclaim as a leader of scientific endeavor.

The strain of her many travels, the demands of the public, her teaching, and the private experiments she refused to give up, at last began to take their toll of physical strength. She was so tired following her first visit to the United States that she became seriously ill. In April, 1932, she was gravely injured by a fall in her laboratory and a surgical operation was necessary. In accordance with her wishes her last illness was unknown except to the family and a few intimate friends. After five weeks in a Paris hospital, she was removed for a few days to a Sanitarium in Sallanches on the upper Savoy, where the world's foremost woman scientist died July 4th, 1934. Her personal physicians attributed her death to a wasting anemia, produced, or at least aggravated, by the long contact with powerful radiation. That her frail frame had so long endured was no doubt due to the dominance of her forceful mind.

Neither of the Curies deigned to profit from their discoveries. No patents were obtained, no secrets regarding methods or processes kept, and all knowledge or theory was freely given to their fellow scientists. Prizes and awards of money were turned to scientific experiments or given to hospitals for the treatment of cancer. Madame Curie never owned a particle of the radium she produced until 1931, when she accepted a gift of $100,000 presented by President Harding from the women of America for the purchase of a gram of radium for experimental work. This gram was obtained from 500 tons of carnotite ore. However, she rented that radium for $3,500 a year and gave the money to a cancer hospital in Warsaw. The purchase of a second gram of radium was made possible by the donation of $50,000 on her second visit to America, President Hoover this time making the presentation. On the occasion of the twenty-fifth anniversary of the discovery of radium the French government unanimously voted her an annual pension of 40,000 francs. In honor of Marie, her daughter Irene was given an endowment of over $56,000 by American women to enable her to pursue her scientific research.

Despite financial rewards, and countless more had been hers for the asking, Marie Curie continued to live simply and frugally, giving freely of her surplus to science and to charity. By her own direction even after death there was no extravagant display or pomp or ceremony, and the people she so loved respected her last wish. But no power could lessen the intense feeling of gratitude and reverence the world holds for the great woman who has just passed from us. No nobler example of unselfish sacrifice for science and humanity has ever been presented. How oft her frail body must have ached and her tired mind yearned for quiet rest with her family! How oft must she, as human as the rest of us, have been tempted to enjoy in luxury of later years the fruits of earlier toil! Her devotion to her science was extreme, it vied only with her devotion to her family and her husband. Pierre was ever her inspiration and she unselfishly deferred to him honors intended for

her and gave him credit as a co-discoverer of radium along with many other achievements. His precept—"Make life a dream, make dreaming a reality"—was carried out by both of them on the highest possible plane.

We of the radiological world are peculiarly moved by the death of Madame Curie. In life she was one of us, a leader in our cause of conquering the enemies of life through advancement of scientific knowledge. Of all her achievements it is said she was most proud of having found a new agent for alleviating human ills. We are grateful to her for the opportunity to use this agent in our work as radiologists. We sorrow with the scientific world that so brilliant a mind, with its rich store of knowledge and wisdom should be halted in its activity. We rejoice with humanity that Madame Curie was permitted so many years of invaluable service to mankind. We are fortunate to have been contemporaries of such a pioneer—future generations will no doubt envy us the personal contact that has been the privilege of so many of us. Poland may justly feel proud as the native land where she received her early training; France may, with pardonable pride, take credit for the great work carried on within her borders; the world in general rejoices that so great a scientist lived to such good purpose. However, only those who combine pure science with personal service to humanity can fully appreciate the life of sacrifice and service which was hers.

In 1923, in a most impressive biography, Madame Curie[2] told in simple language the life story of her husband, who, in spite of poverty and hardship, had adhered to his altruistic deeds and as a comrade shared with her both his home and his work. Her story of how she discovered radium is a saga to those who are carrying out scientific investigations under unfavorable circumstances. To quote: "I had decided on a theme for my doctorate. My attention had been drawn to the interesting experiments of Henri Becquerel on the salts of the rare metal uranium. Becquerel had shown that by placing some uranium salt on a photographic plate covered with black paper, the plate would be affected as if light had fallen on it. The effect is produced by special rays which are emitted by the uranium salt and are different from ordinary luminous rays as they can pass through black paper. Becquerel also showed that these rays can discharge an electroscope. He at first thought that the uranium rays were produced as a result of exposing the uranium salt to light, but experiment showed that salt kept for several months in the dark continued the peculiar rays," and again "I then thought to find out, if there were other substances possessing the remarkable property of uranium, and soon found that substances containing thorium behaved in a similar way." A certain number of other minerals was examined, and to quote again: "A few of them showed activity; they were those containing uranium and thorium. The activity of these minerals would have had nothing astonishing about it, if it had been in proportion to the quantities of uranium or thorium contained in them. But it was not so; some of these minerals revealed an activity 3,004 times greater than that of uranium. I verified this surprising fact carefully, and could not doubt its truth. Speculating about the reason for this, there seemed to be but one explanation. There must be, I thought, some unknown substance, very active, in these minerals. My husband agreed with me and I urged that we search at once for this hypothetical substance, thinking that, with joined effort, a result would be quickly obtained."

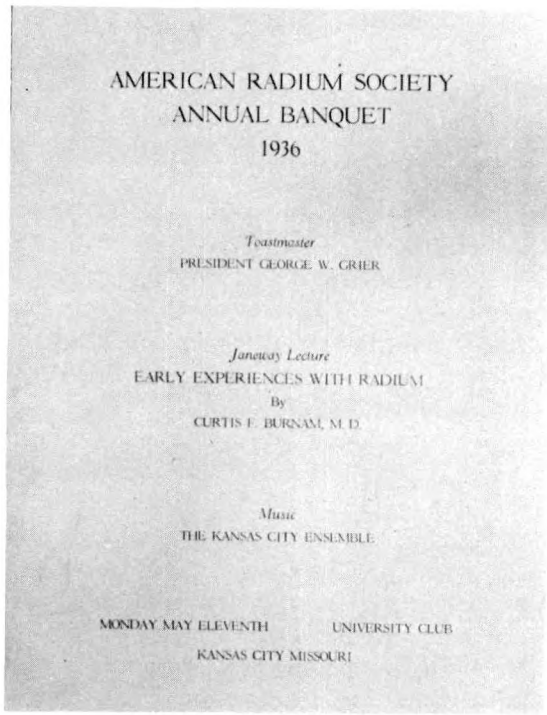
The discovery of polonium and "*radium*" followed.

The years 1936 and 1937 undoubtedly witnessed the most important events in the history of the American Radium Society.

At the Annual Meeting, May 11 and 12, 1936, in Kansas City, Missouri,[3] through the untiring efforts of Edward H. Skinner, the Janeway Medal was introduced at the

[2] Curie, Marie. Pierre Curie. Contains 242 pages. The Macmillan Co., New York, 1923.

[3] American Radium Society. Twentieth Annual Meeting, May 11 and 12, 1936. Am. J. Roentgenol. & Rad. Therapy, 1936, *35*, 551; also 824–825.



AMERICAN RADIUM SOCIETY
ANNUAL BANQUET
1936

Toastmaster
PRESIDENT GEORGE W. GRIER

Janeway Lecture
EARLY EXPERIENCES WITH RADIUM
By
CURTIS F. BURNAM, M.D.

Music
THE KANSAS CITY ENSEMBLE

MONDAY MAY ELEVENTH UNIVERSITY CLUB
KANSAS CITY MISSOURI

Fig. 2.

evening Banquet held at the University Club (Fig. 2; and 3). The medal was designed by Miss Stephanie Prince of Kansas City and executed by Tiffany of New York (Fig. 4). However it was not struck until the following year.

In 1937, on September 15th, the officers and invited foreign members of the Fifth International Congress of Radiology were the guests of the American Radium Society at the banquet in the Red Laquer Room of the Palmer House, Chicago.[4] Each of the Janeway Lecturers from 1933 to 1937 was present upon the platform and received his inscribed medal with appropriate citations. They were: Dr. James Ewing, New York City; Dr. Frances Carter Wood, New York

[4] Program of The Fifth International Congress of Radiology. Am. J. Roentgenol. & Rad. Therapy, 1937, *38*, 373-376.

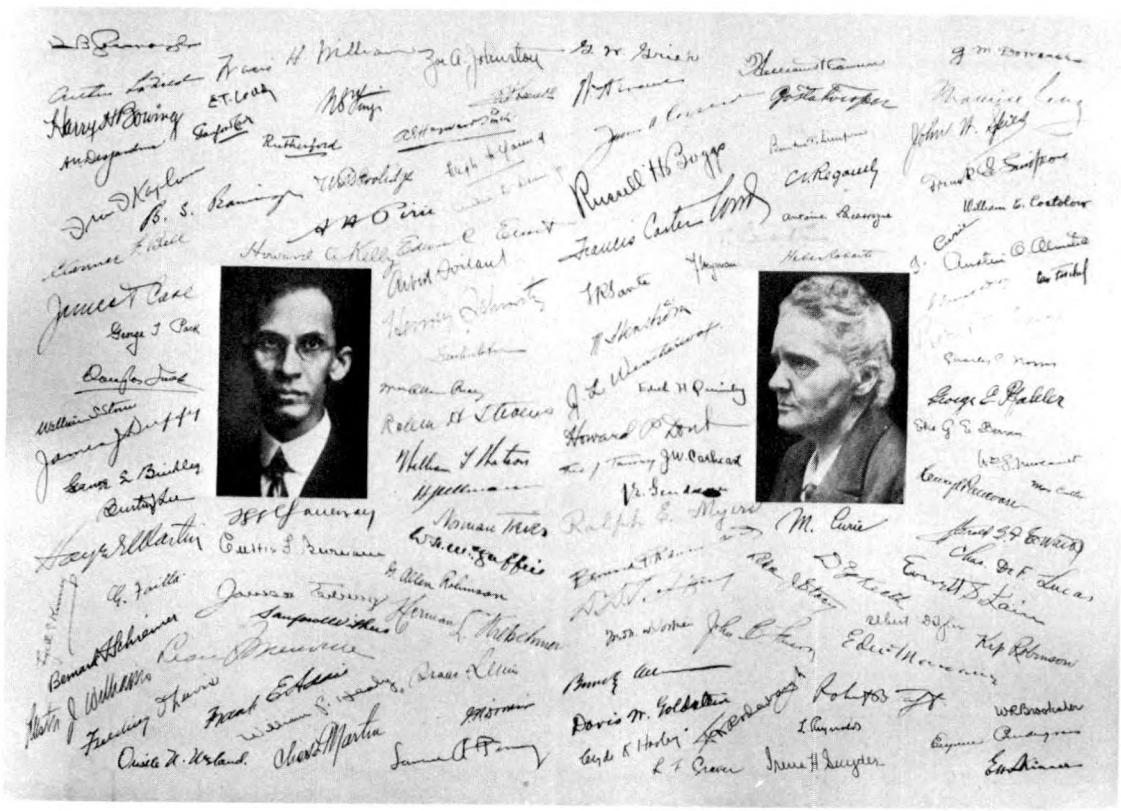


Fig. 3.



Fig. 4. (Reproduced from: Edward H. Skinner.[6] The Henry Harrington Janeway Lecture and Medal. Am. J. Roentgenol. & Rad. Therapy, 1938, *40*, p. 627.)

City; Dr. George E. Pfahler, Philadelphia, Pennsylvania; and Dr. Curtis F. Burnam, Baltimore, Maryland. The Janeway Lecturer at this meeting was Dr. Douglas Quick[5] of New York City, the only assistant of Dr. Janeway.

From 1937 to the present the Janeway Lecture and the presentation of the Janeway Medal continue to remain the highlights of the Annual Meetings of the American Radium Society.

Thus, historically, the term "radium," and, dosimetrically, the terms "radium and radium-equivalent," became symbolic standards of the specification of nearly all forms of radiation in therapy. And, to quote Skinner,[6] "it does not take much flight of the imagination to translate the significance of these mythical items (Odin and his two Ravens) into the Ren of radium lore."

The American Radium Society was honored recently by being embodied in the Ceremonial Mace of the American College of Radiology. This Mace was presented to the College in 1973 at its Golden Anniversary by Eugene P. Pendergrass,[7] who, inspired by the "*Pomp and Ceremony*" of the ritual in 1965 of the "*Bicentennial of the Medical Education in the United States*," spent 8 years in collecting and developing the details of this prototype "symbol of the leadership, scholarship and dignity in the case of Universities and Learned Societies." The Mace was designed and crafted by Mr. Henry P. Hopkins, a famous silversmith, of Baltimore, Maryland. Its wings and serpents are made of silver recovered from "fixing" solutions of x-ray films, by the sludge and electrolytic systems. The head of the Mace is made of sterling silver and the metal is goldplated to prevent tarnishing. The main pear-shaped portion of the head, containing 6 panels with silhouettes of early and modern x-ray tubes, is symbolic of the "*Lamp of Wisdom*," reminiscent of the mythological Odin at Mimir's well and his two ravens. At the bottom section of the head, there are four chased leaves and above that a six-sided section with engraved sterling silver seals. *One of these sections commemorates The American Radium Society*.

The editorial policy of this Journal was stated by Lawrence Reynolds,[8] its world-renowned Editor for more than 3 decades, on the occasion of the Golden Anniversary of its foundation, as follows: "The Founders and first Editor of the Journal, Doctor Hickey, blazed the trail of a high standard of scientific endeavor, from which the Journal has not deviated."

Based on this scientific endeavor, 3 major successive changes were made in the historic title of the Journal, reflecting the

[5] Quick, Douglas. Carcinoma of larynx. Janeway Memorial Lecture, 1937. Am. J. Roentgenol. & Rad. Therapy, 1937, *38*, 821–853.

[6] Skinner, Edward H. The Henry Harrington Janeway Lecture and Medal. Am. J. Roentgenol. & Rad. Therapy, 1938, *40*, 626–628.

[7] Pendergrass, Eugene P. The ceremonial mace of The American College of Radiology. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 705–708.

[8] Reynolds, Lawrence. Fiftieth Anniversary (1906–1956). Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *75*, 3–6.

successive phases in the astounding dynamic progress made in the science of Radiology.

The first change occurred in 1913, when the QUARTERLY was changed into a monthly publication with the title AMERICAN JOURNAL OF ROENTGENOLOGY.[8]

The second change was made in January 1923, when the name "Radium Therapy" was added. The American Radium Society had been founded in 1916 by a pioneer group of active physicians "to promote the scientific study of radium and other sources of radiation in relation to their physical properties and their therapeutic application." Since most of the papers read before this Society were published in the AMERICAN JOURNAL OF ROENTGENOLOGY, at the Fifth Annual Meeting, held in New Orleans, Louisiana, in April 1920, it was decided to make the AMERICAN JOURNAL OF ROENTGENOLOGY the official organ of the American Radium Society. This suggestion was accepted in September, 1922, by the American Roentgen Ray Society at the Annual Meeting held in Los Angeles, California and in January, 1923, the name of the Journal became THE AMERICAN JOURNAL OF ROENTGENOLOGY & RADIUM THERAPY.[8]

The third change was made by acceptance of the designation "Nuclear Medicine"[9] instead of "Atomic Medicine" at the Fifty-second Annual Meeting of the American Roentgen Ray Society held in Washington, D. C., September 25–28, 1951, in recognition of the importance of radioisotopes and the rapidity with which they have been employed within recent years not only in diagnostic procedures but also as therapeutic agents.

The final title of THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY & NUCLEAR MEDICINE[10] was adopted and has been consistently used for nearly a quarter century.

TRAIAN LEUCUTIA, M.D.

[9] LEUCUTIA, TRAIAN, M.D. Editorial. Nuclear Medicine; a historic connotation. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 845–846.

[10] Addition to the title of the Journal. Editorial. AM. J. ROENTGENOL. & RAD. THERAPY, 1952, *67*, 114.

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

Harold O. Peterson, M.D., *Director*

Richard G. Lester, M.D., *Associate Director*

Titles and Abstracts of Courses Offered
Seventy-Fifth Annual Meeting
The San Francisco Hilton Hotel
San Francisco, California
September 23–27, 1974

The Instruction Courses for the Seventy-fifth Annual Meeting of the American Roentgen Ray Society will be held on Monday, Tuesday, Wednesday and Thursday afternoons, September 23, 24, 25, 26 at 3:00 P.M. and Friday *morning*, September 27, at *11:00 A.M.*

Each course will last about 90 minutes, during which time there will be no other activity scheduled. Please note the change in time on Friday . . . courses will start at 11:00 A.M. and conclude at 12:30 P.M.

A special registration desk for the courses will be located near the general registration area. Complete information as well as tickets for the courses will be available on Sunday, September 22, from 2:00 P.M. to 4:30 P.M., and daily the rest of the week.

Ten courses will be given on Monday, 15 on Tuesday and Wednesday, 14 on Thursday, and 9 on Friday. Please note that some of the courses will be given in 2 sessions. A complete list of the faculty and abstracts for each course are shown on the following pages, along with the registration form.

Tickets will be mailed to everyone whose registration (Pink Sheet) reaches the office of the Director before September 14, 1974. Please watch for your tickets which will be mailed in an envelope marked: "Section on Instruction, American Roentgen Ray Society—Tickets Enclosed." The first mailing will be about September 5, 1974.

HOW TO REGISTER AND OBTAIN TICKETS

Review the abstracts on the following pages; make 3 choices for each day; fill out the pink order sheet with the course number and name of instructor for each choice. For each course selected a $3.00 registration fee is required from nonmembers. If you select 5 courses, one each day, the cost will be $15.00.

Members of the American Roentgen Ray Society, graduate students or residents in Radiology, and nonmembers who are contributing to the program either by way of an instruction course, a paper or *scientific* exhibit, are not required to pay for these courses but must fill out a pink order form indicating their choices.

If your advance registration reaches the office of the Director after September 14, 1974, you may obtain your tickets at the registration desk of the Section on Instruction at the hotel on Sunday, September 22, 1974, between 2:00 and 4:30 P.M.

Please mail your orders to Dr. Harold O. Peterson, 1995 West County Road "B", St. Paul, Mn. 55113.

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Maurice M. Reeder, M.D., LTC, MC, Chief, Department of Radiology, Walter Reed Army Medical Center, Washington, D. C.

David F. Reese, M.D., Diagnostic Roentgenology, Mayo Clinic, Rochester, Mn.

Reed P. Rice, M.D., Associate Professor of Radiology, Duke University Medical Center, Durham, N. C.

Gerhart S. Schwarz, M.D., Radiologist, New York Eye & Ear Infirmary, New York, N. Y.

Charles E. Shopfner, M.D., Chairman and Professor, Department of Radiology, University of South Alabama Medical School, Mobile General Hospital, Mobile, Al.

Stanley S. Siegelman, M.D., Professor of Radiology, Johns Hopkins University School of Medicine; Director of Diagnostic Radiology, Johns Hopkins Medical Center, Baltimore, Md.

Edward B. Singleton, M.D., Director of Radiology, St. Luke's Episcopal Hospital, Texas Children's Hospital, Texas Heart Institute; Clinical Associate Professor of Radiology, Baylor University College of Medicine, Houston, Tx.

Jovitas Skucas, M.D., Assistant Professor of Radiology, Department of Radiology, University of Rochester School of Medicine, Rochester, N. Y.

Edward V. Staab, M.D., Professor and Director, Division of Nuclear Medicine, North Carolina Medical Center, Chapel Hill, N. C.

Howard L. Steinbach, M.D., Chief of Radiology, French Hospital; Clinical Professor of Radiology and Medicine, University of California Medical School, San Francisco; Clinical Professor of Radiology, Stanford University Medical School, San Francisco, Ca.

Philip Strax, M.D., Medical Director, Guttman Institute and Director of Radiology, LaGuardia Hospital, New York, N. Y.

Leonard E. Swischuk, M.D., Professor of Radiology and Pediatrics, The University of Texas Medical Branch, Galveston, Tx.

Elias G. Theros, M.D., Chief and Registrar, Registry of Radiologic Pathology, The American Registry of Pathology, Armed Forces Institute of Pathology, Washington, D. C.

John F. Weigen, M.D., Department of Radiology, Palo Alto Medical Clinic; Clinical Assistant Professor of Radiology, Stanford University, Palo Alto, Ca.

Joseph P. Whalen, M.D., Professor, Department of Radiology, The New York Hospital-Cornell Medical Center, New York, N. Y.

Robert I. White, Jr., M.D., Associate Professor of Radiology; Director, Cardiovascular Diagnostic Laboratory, The Johns Hopkins Medical Institute, Baltimore, Md.

Robert H. Wilkinson, Jr., M.D., Associate Professor of Radiology, Division of Nuclear Medicine, Duke University Medical Center, Durham, N. C.

John N. Wolfe, M.D., Chief, Department of Radiology, Hutzel Hospital, Detroit, Mi.

DESCRIPTION OF COURSES

COURSE 101

Monday

RICHARD H. MARSHAK, M.D.
New York, New York

The Small Intestine

1. Technique.
2. Immuno-deficiency diseases. These include dysgammaglobulinemia, giardiasis, intestinal lymphangectasia, amyloidosis, etc.
3. Lymphosarcoma and Hodgkin's disease.
4. Miscellaneous small bowel diseases related to malabsorption. If time permits, some ischemic lesions of the small bowel will be discussed.

COURSE 102

Monday

WILLIAM MARTEL, M.D.
Ann Arbor, Michigan

Commonly Confused Skeletal Lesions

A series of cases will be presented which illustrate various errors commonly made in the diagnosis of skeletal lesions. Some conditions represent problems in diagnosis both for the radiologist and pathologist.

The discussion will encompass disorders of varying etiologies; and lesions of bones, joints and soft tissues will be shown.

COURSE 103

Monday

A. S. MACMILLAN, JR., M.D.
Boston, Massachusetts

The Temporal Bone

This course is devoted to the region of the temporal bone. It will include plain film roentgenography, linear and hypocycloidal tomography, pantopaque studies plus P.E.G.s, nuclear scans, arteriography, venography, xeroradiography, and C.A.T. scans.

COURSE 104
and
COURSE 204

Monday and Tuesday

JOHN N. WOLFE, M.D.
Detroit, Michigan

Mammography

The course is designed to present some general highlights of mammography. This will consume about the first 30 minutes. The balance of the 2 sessions will be spent concentrating on the diagnosis of carcinoma of the breast and then problems in diagnosis and suggestions for their solution. An outline is as follows:

1. general remarks about mammography, touching on positioning and selection of technique and equipment;
2. signs of carcinoma of the breast with appropriate illustrations;
3. the problems of breast calcifications and their differential diagnosis;
4. the problem of the uncalcified mass and its differential diagnosis;
5. subtle signs of carcinoma and their utilization in an attempt to make the correct diagnosis in cancers which are frequently misdiagnosed.

Both conventional film mammograms and xeroradiographs of the breast will be used as slide material.

COURSE 105
and
COURSE 205

Monday and Tuesday

JOHN A. GEHWEILER, M.D.
Durham, North Carolina

RAYMOND L. OSBORNE, M.D.
Durham, North Carolina

Pitfalls in the Diagnosis of Vertebral Trauma, Part I and Part II

"His spine is broken!" The very idea invokes terror in both patient and relatives.

There is no greater challenge to the proficiency of the radiologist than the evaluation of the injured patient since trauma is the leading cause of death in the first 40 years of life. Are you fully prepared to make a diagnosis quickly and accurately?

In this 2 day course, we will examine roentgenograms of patients having sustained vertebral injuries. Each case will be analyzed as to the problems of differential diagnosis; gross morphology of the vertebrae and the related joints and ligaments; mechanisms of injury and the expected roentgen patterns; and how the radiologist plays a major role in evaluating the acutely injured patient, thus contributing to the proper treatment.

COURSE 106

Monday

ROBERT G. FRASER, M.D.
Montreal, P. Q., Canada

Immunologic Diseases of the Thorax with Emphasis on Their Roentgenographic Manifestations

Although anaphylaxis and allergy were recognized around the turn of the century, it has only been within the last few years that some order appears to be developing out of the chaos of immunologic diseases. In 1968, Coombs and Gell recognized and defined 4 types of immunologic response, each of which is associated with specific morphologic and roentgenologic manifestations within the thorax.

This course will be devoted to a consideration of these 4 types of immunologic reaction, their mechanisms, and the diseases with which each is associated.

The following is a list of the pulmonary diseases to be discussed, together with the type of immune reaction responsible:

Type I. Extrinsic asthma

Type II. Goodpasture's syndrome
Idiopathic pulmonary hemorrhage

Type III. Allergic bronchopulmonary aspergillosis (A.P.A.)
Extrinsic allergic alveolitis
Pulmonary vasculitis: systemic lupus erythematosus, polyarteritis nodosa, Wegener's granulomatosis, drug reactions
Fibrosing alveolitis
Rheumatoid lung disease
Sjögren's syndrome

Type IV. This type probably occurs during the course of all pulmonary infections.

COURSE 107

Monday

ROBERT J. COWAN, M.D.
Winston-Salem, North Carolina

C. DOUGLAS MAYNARD
Winston-Salem, North Carolina

Dynamic and Static Plain Imaging

This course will review the fundamental concepts and clinical applications of dynamic and static brain imaging including: radiopharmaceuticals, patient preparation, technique of the procedure; indications and contraindications for the study; common sources of error; interpretation, accuracy and detection of various types of pathology; and effect of timing on the study.

The role of the dynamic study as an adjunct to the standard brain image will also be considered in depth.

COURSE 108

Monday

GERHART S. SCHWARZ, M.D.
New York, New York

Ten Most Frequent Mistakes Made in Reading Paranasal Sinus Films

The most common mistake in interpreting roentgenograms of the paranasal sinuses will be illustrated. Among them will be: false absence of sinuses (reappearance at a later date); polyps that turn out to be cysts; fluid levels that are not; false fractures that are normal anatomic structures; undeveloped sinuses which stimulate information for tumor, etc.

The 4 sinus groups will be discussed separately.

COURSE 109

Monday

STEPHEN A. KIEFFER, M.D.
Syracuse, New York

Myelography and Clivography

This course will cover the following selected topics relating to the use of positive contrast media in the evaluation of the spinal canal and posterior cranial fossa:

1. *The technique of a good myelogram.* Principles which, if carefully followed, are most likely to yield an accurate examination.
2. *The herniated lumbar disk.* Identification of myelographic patterns which show a high degree of correlation with clinically significant disk herniation. Atypical defects and their significance.
3. *The herniated cervical disk and cervical spondylosis.*
4. *Clivography.* Indications for and techniques of

examination of the cisterns and ventricles of the posterior fossa. Diagnosis of acoustic neuroma, brainstem glioma, Arnold-Chiari malformation, and other posterior fossa tumors is often most easily and reliably made by this method.

Emphasis will be placed on a clear understanding of normal anatomy in each region and correlation of radiologic findings with pathologic anatomy.

COURSE 110

Monday

PHILIP STRAX, M.D.
New York, New York

Mass Screening for Earlier Detection of Breast Cancer

COURSE 201

Tuesday

RICHARD H. MARSHAK, M.D.
New York, New York

Stomach and Duodenum

The following entities will be discussed:

1. Gastric polyps and atrophic gastritis.
2. Submucosal tumors.
3. Benign prepyloric ulcers.
4. The narrowed stomach.
5. Enlarged stomach.
6. Unusual lesions of the duodenal bulb and second portion of the duodenum.

Included in the discussion will be the author's concepts both of diagnosis and treatment of these entities.

COURSE 202
and
COURSE 302

Tuesday and Wednesday

HAROLD G. JACOBSON, M.D.
Bronx, New York

STANLEY S. SIEGELMAN, M.D.
Baltimore, Maryland

Skeletal Radiology—Interesting Challenges, Parts I and II

Of all the many interesting and diverse subjects in diagnostic radiology, skeletal disorders comprise one of the most fascinating of the entire discipline. Sufficient variations exist in each diagnostic category to offer provocative challenges to those interested in testing their skills.

In these 2 courses a variety of interesting and even infrequently encountered skeletal abnormalities will be presented together with unusual manifestations of more common disorders. These entities will be organized into appropriate categories (*e.g.*, endocrine abnormalities, neoplasms of bone, etc.). The first few cases in each group will represent unusual manifestations or even esoteric examples ("mavericks") of that specific entity; these will serve as a springboard for the presentation of more frequently encountered manifestations of the abnormality under discussion. Participants in these courses will be encouraged to offer diagnostic possibilities (from their seats) for the introductory cases in each category—always on a *completely voluntary* and *anonymous* basis (the room is dark).

The radiologic criteria diagnostic for each abnormality (even for the "mavericks" in most instances) will be discussed, with emphasis on the concept that "the answer is always on the film," even in the most difficult of cases.

The learning process is closely related to *challenge*. In these 2 courses it is anticipated hopefully that the case material will be sufficiently stimulating and the gratifications inherent in volunteering the correct diagnosis sufficiently rewarding to ensure a relevant learning experience. In any event, *no one will be embarrassed* because this will not be a "quiz show." Hopefully, the courses will demonstrate that learning, related to *challenge*, can be fun and that a careful analytic evaluation of the radiologic findings generally will provide prime consideration of the correct answer even in unusual instances since the film usually speaks its own specific language.

COURSE 203

Tuesday

A. S. MACMILLAN, JR., M.D.
Boston, Massachusetts

Sinuses and Orbits

This course will cover the sinuses, orbits, and the rest of the face. Routine projections on plain films and linear and hypocycloidal tomography will be discussed. Sialography, sinography, nasopharyngography, orthopantomography, dental radiography, and xerography are included. Study of the orbit includes dacryocystography, IOFB localization and B mode ultrasonography. Chiasmal air studies with hypocycloidal tomography, nuclear scans, arteriography, venography, and C.A.T. scans have their place. Emphasis on proper plain film evaluation is still foremost.

COURSE 206

Tuesday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

MAURICE M. REEDER
Washington, D. C.

The Gamut Approach to Radiology

Successful radiologic interpretation begins with a detailed analysis of all the parameters of disturbed morphology and pathophysiology seen on the roentgenogram.

The radiologist can usually fit the visualized abnormalities into a specific pattern of disturbed morphology such as honey-comb lung, bull's eye lesion of the gastrointestinal tract, solitary epiphyseal lesion of bone, etc. Each pattern has its own gamut or comprehensive list of differential possibilities from which the correct diagnosis can be suggested with the aid of pertinent clinical information.

This course will emphasize the importance of a thorough knowledge of gamuts as an essential element in radiographic analysis, alerting the clinician to the various possible etiologies for the specific radiographic abnormality found in his patient.

Twenty representative gamuts covering bone, chest and gastrointestinal tract radiology will be discussed and distributed to registrants.

COURSE 207

Tuesday

EDWARD B. SINGLETON, M.D.
Houston, Texas

Pediatric Pulmonary Abnormalities

The course will primarily consider causes of respiratory distress in the newborn and young infant.

The following conditions will be discussed:

Primary pulmonary diseases producing respiratory distress in the newborn and young infant

Transient tachypnea of the newborn (wet lung syndrome)
Respiratory distress syndrome
Aspiration syndrome
Pulmonary hemorrhage
Intrauterine infectious pneumonia
Aspiration pneumonitis
Wilson-Mikity syndrome
Bronchopulmonary dysplasia
Pulmonary lymphangiectasia
Pulmonary agenesis

Surgical or mechanical abnormalities producing respiratory distress in the newborn and young infant

Obstruction of upper air passages
Esophageal atresia and tracheoesophageal fistula
Bronchopulmonary foregut malformations
Pulmonary sequestrations
Pneumomediastinum and pneumothorax
Congenital lobar emphysema
Congenital cystic adenomatoid malformation
Phrenic nerve paralysis and eventration
Diaphragmatic hernia
Accessory diaphragm
Vascular ring
Chylothorax
Congenital deformities of the thorax.

If time permits, some of the common and rare pulmonary infections encountered in the young infant will be included.

COURSE 208

Tuesday

NORMAN E. CHASE, M.D.
New York, New York

IRWIN I. KRICHEFF, M.D.
New York, New York

The Angiographic Diagnosis of Intracranial Mass Lesions

This course will include a logical approach to the study and differential diagnosis of supratentorial intracranial mass lesions with an analysis of localization by shift, vascular displacements and physiologic changes.

The techniques of angiography and supplemental studies will also be included.

COURSE 209

Tuesday

DAVID H. BAKER, M.D.
New York, New York

WALTER E. BERDON, M.D.
New York, New York

Gastrointestinal Radiology in Infants and Children, Other than Newborn Intestinal Obstruction

A practical approach will be offered in the gastrointestinal examination of infants and children. The logical choice of plain films, the various approaches to the upper gastrointestinal study, as well as our method for barium enema examination, will be covered.

Numerous entities with their usual, as well as their extraordinary manifestations will be reviewed. These will include: inflammatory disease (appendicitis, ileitis, colitis), gastrointestinal bleeding states (intussusception, ulcer disease) with considerable attention to the indications and contraindications of intussusception reduction, the manifestations of neoplastic

and immunologic disease states, plus late manifestations of some congenital lesions.

COURSE 210

Tuesday

RICHARD H. GREENSPAN, M.D.
New Haven, Connecticut

Emphysema and Other Conditions Associated with Air Trapping

The radiologic diagnosis of emphysema and other forms of chronic obstructive pulmonary disease is insensitive and inaccurate. Attempts to relate the radiographic image with pathologic and morphologic changes within the lung have led to confusion, and various classifications derived from pathologic study of the lung are inappropriate for radiologic diagnosis.

Clinically, the screening method most commonly used in the detection of emphysema involves a comparison of the one second forced expiratory volume to the total vital capacity. We have adapted this method to radiography in an attempt to make the x-ray a more sensitive tool in detection of air trapping. The method has value in detecting generalized and localized air trapping within the thorax.

This course will concern itself primarily with the detection of air trapping within the thorax in various conditions, including pulmonary emphysema and other forms of chronic obstructive pulmonary disease.

COURSE 211

Tuesday

LEONARD E. SWISCHUK, M.D.
Galveston, Texas

Neonatal Chest Problems

The radiologist plays a key role in the diagnosis of neonatal chest disease. His basic job is to determine whether a surgical or medical condition exists, but many times this can be problematic. It is not so much that one isn't familiar with such conditions as hyaline membrane disease, TRDN (transient tachypnea), congenital diaphragmatic hernia, meconium aspiration, etc., but rather that one is often not able to come up with a useful differential diagnosis for certain abnormal roentgenographic patterns. For example: (1) What does one think of when one is presented with a large, opaque hemithorax? (2) A large, hyperlucent hemithorax? (3) A small, densely opaque hemithorax? (4) A small, slightly opaque hemithorax? (5) Bilateral fluffy infiltrates? (6) With bilateral bubbly lungs? (7) With bilateral granular lungs? (8) With bilateral parahilar, streaky, congested appearing lungs?

Each disease will be dealt with in enough detail so as to allow differentiation of one from the other. Grouping according to abnormal pattern is the place to start.

COURSE 212

Tuesday

JAMES J. CONWAY, M.D.
Chicago, Illinois

Pediatric Nuclear Medicine: The Central Nervous System

The application of nuclear medicine to the pediatric patient has now passed its infancy and with youthful vigor is assisting in the diagnosis and treatment of many childhood disorders. Perhaps the central nervous system has received the most attention to date and so a discussion of radionuclide brain imaging emphasizing pediatric abnormalities as opposed to well-recognized adult disorders is in order. The author wishes to convey to the participants of this course his experience of over 2,000 central nervous system studies in children. The contents will include:

A. Sedation, injection, and handling techniques for infants and children
B. Normal variants
   1. Cranium bifidum
   2. Biparietal foramen
   3. Fontanelles
C. Congenital anomalies
   1. Posterior fossa
      a. Dandy-Walker malformation
      b. Arachnoid cyst of the posterior fossa
      c. Arnold-Chiari malformation, Type II
      d. Congenital aqueductal stenosis
   2. Supratentorial
      a. Unilateral occlusion of the foramen of Monro
      b. Arachnoid cyst
      c. Porencephaly
      d. Plagiocephaly
D. Microcephaly *vs.* hydrocephaly
E. Neoplasia
   1. Supratentorial midline lesions
   2. Supratentorial cerebral lesions
   3. Supratentorial peripheral lesions
   4. Posterior fossa midline lesions
   5. Posterior fossa cerebellar lesions
   6. Miscellaneous lesions
      a. Tuberous sclerosis
      b. Dermal sinus
F. Trauma
   1. Extracranial lesions
   2. Cranial lesions
   3. Intracranial lesions
   4. Miscellaneous abnormalities
G. Inflammation
   1. Encephalitis

2. Ventriculitis
3. Meningitis
4. Chemical (chemotherapy)

H. Vascular abnormalities
1. Hypervascular lesions
   a. Aneurysm of the vena Galen
   b. Sturge-Weber syndrome
2. Hypovascular lesions
   a. Birth anoxia
   b. Sickle cell disease
   c. Brain death

COURSE 213

Tuesday

ANTHONY F. LALLI, M.D.
Cleveland, Ohio

Destructive Lesions of the Renal Parenchyma

The purpose of this course is to discuss the causes for the variations of renal size.

The primary reasons for variations of renal size are the destruction of renal parenchyma and the failure of the kidney to reach normal adult size.

The various reasons for these phenomena will be dealt with, outlining diagnostic aids in the determination of the basic pathology and the urographic and angiographic means to use in establishing the diagnosis.

Primary disease entities for consideration will include: renal papillary necrosis; medullary sponge kidney disease; pyelonephritis; renal ischemia and infarction; and renal insult and "failure to thrive."

COURSE 214

Tuesday

JOHN W. LANE, M.D.
Little Rock, Arkansas

EDWARD V. STAAB
Chapel Hill, North Carolina

Urinary Tract and Placental Nuclear Medicine Studies

Recent advances have changed Renal Nuclear Medicine studies from screening to definitive examinations in many instances.

Newer radiopharmaceuticals that increase the resolution in imaging studies make these techniques in selective instances the ones of choice for evaluating patients with potential space occupying lesions.

New ways to use many of the radiopharmaceuticals for evaluating patients with genitourinary abnormalities have been developed and evaluated clinically.

The combination of the sensitive physiologic measures that are obtained with radiopharmaceuticals and simultaneous imaging with camera systems has improved the specificity of older techniques.

This portion of the course will primarily concern itself with the clinical usefulness of Renal Nuclear Medicine in: (1) renal failure; (2) space occupying lesions; (3) urinary tract obstructions; (4) trauma; (5) children (neonates); (6) hypertension; (7) reflux; (8) residual bladder contents; (9) transplantation; (10) kidney preservation.

A short review of the major methods of radionuclide placentography will also be presented. The major emphasis will be upon practical studies available to the average Nuclear Medicine Department and methods, techniques, tips and helpful hints, as well as correlation with clinical findings and studies shall be covered. In conclusion, newer methods and thoughts on future directions in radionuclide placentography will be briefly considered.

COURSE 215

Tuesday

HERMAN GROSSMAN, M.D.
Durham, North Carolina

Renal Masses in Children

A child with an abdominal mass requires a rational approach to diagnostic evaluation in order to prevent wasted time, unnecessary trauma and expense to the patient and his family. Radiographic and other imaging examinations (*i.e.*, ultrasound and radioactive isotope scanning) should be ordered in logical sequence for the particular findings in the patient.

In the evaluation of renal masses in children emphasis will be on excretory urography and the associated studies (total body opacification and tomography) that can be obtained as needed during the urographic examination. Ultrasonography in conjunction with urography is very accurate in separating cystic and solid tumors of the kidney. These noninvasive methods give sufficient information for proper surgical management in children.

Pseudotumors of the kidneys are important to recognize. Although nephrotomography and angiography are satisfactory diagnostic methods, the advantage to renal scanning will be demonstrated.

Renal cystic diseases will be described and a simple method for grouping these diseases will be suggested.

COURSE 301

Wednesday

CHARLES E. SHOPFNER, M.D.
Mobile, Alabama

Current Status of Pediatric Radiology

Changes in physicians' practice and personal behavioral patterns come slowly. However, distinct changes have occurred after 10 years of continued

clinical study and investigation of pediatric urinary tract problems. These are in the areas of indications, methodology, motivation, normal anatomy and interpretation of uroradiologic examinations. Uroradiology remains as the most important, single clinical examination for the child with urinary tract infection, when validly indicated, properly performed, patient motivated and when interpreted according to cause and effect, postulating to show beyond a doubt the cause and effect of roentgen pathology.

Clinical material is used exclusively to illustrate specific clinical uroradiologic aphorisms and also generally to report clinical investigations.

COURSE 303

Wednesday

C. BENJAMIN GRAHAM, M.D.
Seattle, Washington

Persistent Myths which Confuse and Complicate Children's Radiology

Many misconceptions exposed here are longstanding and well entrenched in medical lore. The corresponding truths are almost equally old, but unfortunately they remain generally poorly known or understood. Healthy and sick children alike will profit if their radiologists recognize that:

1. Skeletal maturation assessment and growth potential prediction are rather imprecise and greatly overutilized.
2. The normal bone age range is surprisingly wide, even encompassing most short boys and tall girls.
3. Periosteal new bone, metaphyseal bands and beaks, and irregular or fragmented ossification centers are predominantly normal developmental phenomena.
4. The only radiographic sign in the newborn of so-called congenitally dislocated hip is actual subluxation.
5. Lumbosacral spina bifida occulta is very common and represents no more than an anatomic variation.
6. Vertebral beaking is a greatly misunderstood deformity associated with certain disorders, coming in a confusing but meaningful array of sizes and shapes.
7. Wormian bones and J-shaped sella turcica are to be expected in the infant skull and persist longer in immature individuals.
8. Normal newborn kidneys are strikingly large and usually function relatively poorly.
9. The infant urinary tract requires, and its systems allow, significantly higher urographic doses for optimum demonstration.
10. Developmental abnormalities of bladder outlet and posterior urethra, excluding valves in males, are virtually nonexistent.
11. Diaphragm level alone is a most inexact method for evaluating degree of inspiration or aortic catheter position.
12. The normal thymus may be huge, interfering with cardiac evaluation and masquerading as mediastinal disease in later childhood.
13. Neonatal atelectasis, aspiration, and pneumonia remain much overdiagnosed, while widespread misunderstandings regarding the more prevalent newborn respiratory problems continue.

It is hoped that, as "truths" prevail over "myths," there will be time for spirited discussion.

COURSE 304

Wednesday

ROBERT L. EGAN, M.D.
Atlanta, Georgia

Detection of Early Breast Cancer

A review of mammography, and other modalities it has engendered, and the study of breast diseases will be presented. The value of mammography, its pitfalls and how to obtain the most from the procedure will be stressed. Also frankly discussed will be mammographic equipment, radiographic detail and dosage, compression, screen-film combinations, processing, collimation and requirements for an occasional as well as a busy mammographic room. If desired, discussion can extend into thermography and modified radiographic techniques for breast examination.

COURSE 305

Wednesday

LEWIS E. ETTER, M.D., F.A.C.R.
Pittsburgh, Pennsylvania

Plain Film Examination of the Skull

Proven roentgen anatomic features of the skull in various positions with some pathologic alterations of interest to the practicing radiologist will be demonstrated.

As many pathologic alterations of the normal roentgen anatomy will be shown as time will allow.

COURSE 306

Wednesday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Pattern Recognition of Pulmonary Lesions

Everyone has difficulty in distinguishing among alveolar, interstitial, and other patterns of pulmonary involvement.

The purpose of this course is to help you decide when you can, and when you can't make these distinctions.

COURSE 307

Wednesday

HYMER L. FRIEDELL, M.D.
Cleveland, Ohio

Radiologic Information—Are there Trade-offs?

The quest for radiologic information is inexhaustible as to completeness, reliability and accuracy. Absolute accuracy and reliability, however, can never be achieved if there is any uncertainty in the system. The only method of reducing the impact of uncertainty (noise) is by using some form of redundancy (more procedures). It can be shown, therefore, *that more is always better.*

Since increased information and reliability are accompanied by increased radiation to the patient, any increased radiation hazard will have to be balanced against the value of more radiologic information. Similarly the cost factor comes into play.

How can these opposing elements be reconciled?

(a) A trade-off must develop between the number and type of examination done (cost) and the value of the information obtained.
(b) The extent and reliability of the information must be balanced against the increased radiologic hazard.

The nature of the problems, as well as some specific data and possible solutions, will be presented.

COURSE 308

Wednesday

DAVID F. REESE, M.D.
Rochester, Minnesota

Computerized Trans-Axial Tomography

The 3 basic forms of radiologic imaging consist of the screen-film combination, electronic methods as used in the image intensifier, and radioisotope organ visualization. To these, computerized trans-axial tomography (CTT) must now be added.

This course will deal with an experience of 2,000 scans made by the EMI scanner. The mechanics of the machine will be covered; neuroanatomy in the transverse axial projection will be explained and correlated with the computer displays.

Our clinical experience with tumors, trauma and degenerative lesions will be presented and the impact of the method on the more conventional neuroradiologic diagnostic techniques will be discussed.

COURSE 309

Wednesday

ROBERT W. McCONNELL, M.D.
Dallas, Texas

ROBERT H. WILKINSON, JR., M.D.
Durham, North Carolina

Radionuclide Imaging Studies for Evaluating the Liver, Spleen, Pancreas and Skeletal System and for Tumor Localization

The liver, spleen and pancreas lend themselves to imaging and scanning rather easily, since each will concentrate a radiopharmaceutical in detectable amounts. Making some sense out of these scans or images, relating them to anatomy and physiology, and correlating them with other diagnostic studies and the clinical work-up will be the thrust of this portion of the refresher course.

A survey of current radiopharmaceuticals, instrumentation and methodology employed in bone imaging as well as the clinical applications of this examination will be given. A comparative evaluation of current radionuclide bone imaging procedures and bone roentgenograms in terms of their sensitivity and specificity will also be discussed.

The detection of soft tissue neoplasms and/or inflammatory lesions using radiopharmaceuticals such as gallium 67 citrate and nuclear medicine imaging instrumentation has generated considerable interest; opinions as to the clinical value of these procedures will be presented and discussed.

COURSE 310

Wednesday

A. EVERETTE JAMES, JR., M.D.
Baltimore, Maryland

ROBERT I. WHITE, JR., M.D.
Baltimore Maryland

The Correlation of Radiographic and Radioisotopic Imaging: with Particular Emphasis on Chest Disease

The correlation of data acquired by radioisotopic and radiographic imaging of altered pulmonary physiology has proven valuable, sensitive and accurate. Characteristic radiographic appearances and radiopharmaceutical distribution patterns in pulmonary embolism, chronic lung disease, pulmonary edema, inflammatory conditions, and bronchogenic carcinoma will be demonstrated. Plain film chest roentgenograms, perfusion lung scans, gas inhalation images, and pulmonary angiograms will be considered with regard to their role in the evaluation of all types of lung disease. Sensitivity and specificity of each diagnostic study will be discussed. We will also present a diagnostic approach to pulmonary pathology to aid in clinical practice.

COURSE 311
and
COURSE 411

Wednesday and Thursday

ARNOLD CHAIT, M.D.
Philadelphia, Pennsylvania

Abdominal Angiography, Part I and Part II

Following a decade of explosive expansion, abdominal angiography has now taken its place among the more expensive, but no longer very "special" radiologic procedures. Its applications are well understood and its limitations recognized.

This course will include a brief review of instrumentation, techniques, and hazards, but will concentrate on a demonstration of the angiographic patterns of disease of the abdominal aorta and the viscera perfused by its branches, the liver, pancreas, spleen and gut, as well as the omentum and mesentery, in adults and children.

There will be a discussion of neoplastic, inflammatory, traumatic and vascular diseases, and a review of current experience in the angiographic diagnosis and pharmacologic control of gastrointestinal bleeding.

COURSE 312
and
COURSE 412

Wednesday and Thursday

JEREMY J. KAYE, M.D.
New York, New York

Arthrography—Part I, Shoulder and Hip
Arthrography—Part II, Knee and Ankle

Course 312. I.

A. *Shoulder Arthrography*

1. Brief Historic Review
2. Indications for Shoulder Arthrography; Contraindications
3. Anatomy of the Shoulder Pertinent to Arthrography
4. Detailed Technique of Shoulder Arthrography
5. The Normal Shoulder Arthrogram
6. The Abnormal Shoulder Arthrogram:
   a. Complete Rotator Cuff Tears
   b. Partial Rotator Cuff Tears
   c. The Frozen Shoulder; Adhesive Capsulitis
   d. Post-Dislocation Capsular Deformity
   e. Synovial Abnormalities and Loose Bodies
7. Summary and Conclusions: Why Do Shoulder Arthrography?

B. *Hip Arthrography*

1. Brief Historical Review
2. Anatomy of the Hip Pertinent to Arthrography
3. Detailed Techniques of Hip Arthrography:
   a. No Prosthesis Present, Adult or Child
   b. Prosthesis Present
4. The Normal Hip Arthrogram:
   a. Infant or Child
   b. Adult
   c. Adult With Prosthesis
5. The Abnormal Hip Arthrogram:
   a. Congenital Dislocation of the Hip
   b. Infectious Arthritis of the Hip in Infant or Child
   c. Legg-Calve-Perthes' Disease
   d. Total Hip Prostheses
   e. Miscellaneous Abnormalities
6. Summary and Conclusions: Why Do Hip Arthrography?

Course 412. II.

A. *Knee Arthrography*

1. Brief Historic Review
2. Indications for Knee Arthrography; Contraindications
3. Detailed Anatomy of the Knee As It Pertains to Arthrography
4. Techniques of Knee Arthrography in Detail:
   a. Fluoroscopic Technique
   b. Horizontal-Beam Technique
5. The Normal Knee Arthrogram
6. The Abnormal Knee Arthrogram:
   a. Meniscus Lesions
   b. Articular Cartilage Abnormalities
   c. Cruciate Ligament Tears
   d. Collateral Ligament and Capsular Tears
   e. Synovial Lesions and Loose Bodies
   f. Combined Abnormalities
7. Accuracy of Knee Arthrography:
   a. Statistical Data
   b. Potential Causes of Errors
8. Complications of Arthrography
9. Summary: Why Do Knee Arthrography?

B. *Ankle Arthrography*

1. Brief Historic Review
2. Indications for Ankle Arthrography; Contraindications
3. Detailed Anatomy of the Ankle Pertinent to Arthrography
4. Technique of Ankle Arthrography
5. The Normal Ankle Arthrogram
6. The Abnormal Ankle Arthrogram:
   a. Anterior Talo-Fibular Ligament Tears
   b. Calcaneo-Fibular Ligament Tears
   c. Combined Lateral Ligament Tears
   d. Deltoid Ligament Tears
   e. Synovial Abnormalities and Loose Bodies
7. Summary and Conclusions: Why Do Ankle Arthrography?

COURSE 313
and
COURSE 413

Wednesday and Thursday

DONALD L. KING, M.D.
New York, New York

Course 313. Abdominal and Pelvic Ultrasonography—Part I

Course 413. Abdominal and Pelvic Ultrasonography—Part II

The objective of this course will be to review the physical and technical principles of ultrasonic imaging and to present examples of current clinical applications.

Emphasis will be placed upon procedure, pitfalls and limitations of routine applications, such as placental localization, fetal cephalometry and the "large for dates" patient. Differentiation of various types of adnexal masses will be reviewed. The use of ultrasonography for evaluation of renal masses will be discussed with illustration of various gross pathologic patterns. The visualization of upper abdominal organs and masses will be presented. Real-time and stop-action imaging of the heart will also be discussed and illustrated.

COURSE 314

Wednesday

MICHAEL L. JOHNSON, M.D.
Durham, North Carolina

Cardiac Ultrasonography

Echocardiography is an extremely exciting diagnostic technique using pulsed, reflected, ultrasound which is nontraumatic, accurate and reproducible. It is diagnostic in many cardiac abnormalities including pericardial effusion, mitral stenosis, prolapsing mitral valve, IHSS, and left atrial tumors. It is helpful in assessing ventricular chamber size and function, and in the detection of mitral insufficiency, aortic insufficiency, aortic stenosis, bacterial vegetations and many congenital cardiac abnormalities.

The basic principles of cardiac ultrasonography and the echocardiographic findings in normal and diseased states will be presented. Newer techniques, such as real-time, two-dimensional, sector scanning and cardiac gated cross sectional imaging of the heart will be discussed.

COURSE 315

Wednesday

RAY A. BRINKER, M.D.
New York, New York

JOVITAS SKUCAS, M.D.
Rochester, New York

Radiology Special Procedure Room

The ultimate design of the Special Procedure Room must fall to the radiologist. For the radiologist to make the best selection, he must rely on many advisors, including hospital architects, department planners, equipment manufacturers, as well as his own experience. It is very difficult to decide between reliable, tested techniques on one hand, and newer possibly more versatile equipment and design on the other.

The emphasis of the course will be on a special procedure room design for a radiologist using femoral Seldinger techniques. Although there is much overlap, the special problems concerned with cine angiography and pediatric angiocardiography will not be covered.

Topics will include the room itself, x-ray generation equipment, tables, injectors, film-recording methods and finally a consideration of the give and take between old standard techniques and some newer approaches.

COURSE 401

Thursday

JOSEPH P. WHALEN, M.D.
New York, New York

Radiology of the Abdomen: An Anatomic Approach

This course will first briefly review the anatomy of the abdomen by means of sectional anatomy, stressing the relationship of organ systems. Further, it will discuss how the anatomy reflects the roentgen image in the normal and then how pathologic processes, specifically abdominal tumors and inflammation, alter this normal.

Emphasis will be placed on first localizing the abnormality which in turn leads to a differential diagnosis. The questions then asked are: "Is it normal?" If not, "Where is the abnormality?" Finally, with a knowledge of the localization, "What is the differential diagnosis?"

COURSE 402

Thursday

JOHN E. MARTIN, M.D., F.A.C.R.
Houston, Texas

Mammography

Breast disease is best understood when gross specimens and microhistology are correlated with comparative mammographic images.

The similarities of mammograms and gross specimens will be demonstrated.

Concepts of benign and malignant diseases of the breast will be discussed.

COURSE 403

Thursday

KENNETH D. DOLAN, M.D.
Iowa City, Iowa

Sinuses and Orbits

The emphasis in this presentation will be directed toward evaluation of paranasal sinus and maxillofacial skeletal abnormalities and how these abnormalities may affect the orbits.

This course will be divided into 3 parts:

1. A consideration of normal radiographic anatomy and normal variations in appearance of the orbits;
2. A demonstration of maxillofacial injuries which involve the orbit. They will include consideration of the local orbital, tripod, blowout, Le-Fort II and III and smash injuries which alter the orbital appearance; and
3. A presentation of the radiologic assessment of lesions which produce unilateral exophthalmos.

COURSE 404

Thursday

E. ROBERT HEITZMAN, M.D.
Syracuse, New York

Chronic Interstitial Pneumonia

Chronic disease of the pulmonary interstitium occurs frequently and such cases produce very difficult problems in differential diagnosis for the radiologist.

This presentation will focus on one aspect of interstitial disease of the lung—chronic interstitial pneumonia. The following points will be covered:

1. The anatomy of the pulmonary interstitium.
2. A definition of interstitial pneumonia.
3. A classification of interstitial pneumonia.
4. Theories of pathogenesis of interstitial pneumonia.
5. Radiographic appearances of interstitial pneumonia, with a major emphasis on differential considerations. This portion of the discussion will be based on roentgen anatomic and pathologic correlations.

A number of specific diseases, including the adult respiratory distress syndrome or so-called "shock lung" will be considered. Comments concerning the forms of interstitial pneumonia characterized primarily by infiltration of the interstitium with lymphocytes and eosinophils will be included.

COURSE 405

Thursday

D. GORDON POTTS, M.D.
New York, New York

Brain Anatomy for the Radiologist

Angiography demonstrates the anatomy of the brain because the major blood vessels have a relatively constant relationship to the brain. Air studies demonstrate directly parts of the surface of the brain. Computerized transaxial tomography demonstrates the brain anatomy, including the gyri and sulci, even more directly. The anatomic knowledge required by a radiologist differs from that required by an anatomist, a neurologist or a neurosurgeon. Certain regions must be described in more detail for the radiologist and other regions in less detail.

This course will use various radiologic and anatomic techniques to display the brain anatomy.

Concentration will be on the following areas of special importance to the radiologist:

(a) the cerebellum and its fissures
(b) brain stem
(c) cerebral sulci and gyri
(d) basal ganglia
(e) the long tracts and the cranial nerves.

COURSE 406
and
COURSE 506

Thursday and Friday

HOWARD L. STEINBACH, M.D.
San Francisco, California

Metabolic, Endocrine and Related Diseases

There will be a comprehensive discussion of roentgen findings and recent clinical and laboratory investigations of importance to radiologists in the interpretation of osteoporosis, osteomalacia, rickets, hyperparathyroidism, renal osteodystrophy, types of abnormal calcification, Paget's disease, acromegaly and other related abnormalities. The roentgen response of the skeletal system to modern treatment will be presented.

The first session will consider the various forms of osteoporosis which may sometimes be differentiated roentgenologically. This includes senile, "postmenopausal," Cushing's, congenital and transient forms. The quantitative measurements for establishing the diagnosis and following response to therapy will be discussed. The many roentgenographically distinct types of osteomalacia and rickets (vitamin D deficient, vitamin D dependent, vitamin D resistant), including the familial congenital and juvenile sporadic forms and others will be reviewed in light of new theories of vitamin D metabolism.

The second session will review in detail primary, secondary and tertiary hyperparathyroidism. The exciting recent findings in renal osteodystrophy including various forms of soft tissue calcification and their diagnostic significance will be presented including a discussion of pertinent recent clinical and physiologic information. Paget's disease will then be considered, as well as its early diagnosis and response to calcitonin and other drugs.

If time permits, a review will be made of the roentgen appearance of interesting diseases such as pseudohypoparathyroidism, hypoparathyroidism, hydrothyroidism, hypophosphatasia and others.

COURSE 407

Thursday

JOHN F. WEIGEN, M.D.
Palo Alto, California

Complications of Diagnostic Radiology

Most of the procedures in diagnostic radiology inherently challenge the body's defenses. In spite of this, they have become an essential part of the practice of medicine because the current treatment of the seriously ill requires accurate diagnosis.

The purpose of the course will be to review the types and mechanisms of adverse reactions with emphasis on a plan of treatment for each. Complications of intravascular contrast media will be emphasized and those of other studies will be discussed as time and the interest of the audience dictate.

COURSE 408

Thursday

JAMES T. T. CHEN, M.D.
Durham, North Carolina

Essentials of Cardiac Fluoroscopy

Adequate radiologic evaluation of heart disease depends on 2 basic procedures, namely, roentgenography and fluoroscopy. One deals with the anatomic details of the heart by rapid filming; the other explores the dynamic alterations that are discernible only in motion.

This course is designed to emphasize the importance of cardiac fluoroscopy in the diagnosis of heart disease. Video-tapes, slides, and cineangiocardiograms will be used to illustrate the technique of cardiac fluoroscopy, diseases that are diagnosed mainly by fluoroscopy, the rationale of their ready detectability by fluoroscopy, the correlation between fluoroscopy and other laboratory data, and the future of cardiac fluoroscopy.

Ample examples on tape, both common and uncommon, will be shown.

COURSE 409

Thursday

WILLIAM L. ASHBURN, M.D.
San Diego, California

Nuclear Imaging-Evaluation of Cardiac Diseases

A variety of new radionuclide imaging techniques has been developed which can aid in the evaluation of patients with certain cardiac disorders. Greatest progress has been made in applying these tests in the case of children with suspected congenital intracardiac shunts and in adults with ischemic heart disease. Accordingly, emphasis will be placed on these areas.

The following topics will be discussed: radionuclide angiography in the detection of cardiac shunts; quantitation of left-to-right shunts; myocardial imaging by noninvasive (Rb^{81}) means; nontraumatic determination of cardiac output and ejection fraction; myocardial perfusion imaging following the intracoronary administration of labeled particles; calculation of regional myocardial blood flow indices by the intracoronary inert gas washout technique; and "infarct" imaging in patients with acute myocardial infarction.

The techniques will be described in sufficient detail to allow those interested in applying these tests to begin. Necessary items of recording and processing equipment including the use of computers will be discussed.

COURSE 410

Thursday

C. CRAIG HARRIS, M.D.
Durham, North Carolina

The Choice, Management and Clinical Use of Radionuclide Imaging Instrumentation

In the selection of clinical nuclear imaging equipment, the following should be considered:

1. the specific procedures to be performed;
2. the necessary class of instrument required for these studies (*i.e.*, rectilinear scanner or camera);
3. within the chosen class, the virtues and limitations of a given system with regard to how easily and reproducibly it makes images of the quality desired;
4. the reputation of a system for reliability and availability of competent, restorative service.

Once a system has been acquired, its capability to perform satisfactorily will depend on proper insta[-lation, competent initial operation, cogent and disciplined operational use, periodic performance checks and solicitous "care and feeding" (environment and maintenance) to maintain its "health."

Reproducibly satisfactory clinical radionuclide images depend (over and above competent instrument performance) on physician and technologist cooperation and agreement on such factors as system choice for a given procedure, proper patient positions, location of collimator focus with respect to the target organ, spectrometer base-line and window settings, basic photorecording factors, contrast enhancement and statistical requirements on image

count density, and on maintenance of the agreed-upon parameters in each study.

The basic tenets of instrument choice, "care and feeding" and image production will be set forth in this course.

COURSE 414

Thursday

LLOYD E. HAWES, M.D.
Boston, Massachusetts

The 20 Significant X-Ray Signs of Pulmonary Disease

Only 20 x-ray signs constitute the basis for 70 per cent of all chest diagnoses. Each sign will be described in detail.

The signs range from small localized changes, such as metallic nodules, to widely distributed patterns. The latter include: alveolar air obliteration; exaggerated normal pulmonary reticulum; bronchial wall change; pleural changes; obliteration of normal architecture; intrusion of neoplasia; etc.

Using these signs, many cases, both common and rare, will be demonstrated and the value of these signs in differential diagnosis will be emphasized.

COURSE 501

Friday

J. SCOTT DUNBAR, M.D.
Vancouver, B.C., Canada

Bronchial Disease in Pediatric Radiology

Etiology, investigation and diagnosis of bronchial disease in Pediatrics will be discussed, including indications and contraindications for bronchographic studies.

COURSE 502

Friday

GERALD D. DODD, M.D.
Houston, Texas

Thermography of the Breast

There have been significant improvements in thermographic equipment during the past 5 years. Clinical requirements have been taken into consideration in the development of design parameters and information concerning tumor thermogenesis, interpretative criteria and the reliability of the technique is gradually accumulating.

The following topics, based upon laboratory and clinical trials of the newer types of equipment, will be discussed:

1. The development and present status of thermographic equipment with particular reference to clinical requirements
2. Normal thermographic patterns of the female breast
3. The genesis of abnormal thermographic signals and the heat transfer mechanics involved in the production of the abnormal breast thermogram
4. The accuracy of thermography in the diagnosis of breast cancer with emphasis upon the incidence and causes of false negative and false positive examinations
5. The current place of thermography as a screening device
6. Pending and future developments in thermographic equipment and interpretative techniques.

COURSE 503

Friday

ALEXANDER R. MARGULIS, M.D.
San Francisco, California

Inflammatory Diseases of the Colon

The radiologic appearance of inflammatory diseases of the colon will be discussed, such as the differences between ulcerative colitis, granulomatous colitis, tuberculosis, amebic colitis, other infections and ischemic disease.

The emphasis will be on correlation of gross pathologic findings with roentgen appearance.

The relationship of toxic megacolon and cancer to inflammatory disease will be discussed, as will diverticulitis and its similarities in appearance to carcinoma of the colon and granulomatous colitis.

COURSE 504

Friday

GUY D. POTTER, M.D.
New York, New York

Tomography of the Temporal Bone

A practical guide to tomography of the ear will be presented.

The normal anatomy will be shown, using normal tomograms, specimens and line drawings. The tomographic anatomy will be correlated with the anatomy as seen on routine mastoid roentgenograms.

The tomographic projections which are most useful in various clinical situations will be discussed.

Abnormal cases will be presented with the emphasis on which structures are most likely to be affected, and which tomographic projections will best show these changes.

COURSE 505

Friday

ELIAS G. THEROS, M.D.
Washington, D. C.

JAMES C. REED, M.D.
Washington, D. C.

Fundamental Patterns in Chest Roentgenology: From Nodules to Masses

The sequence of events in the development of pulmonary nodules and masses will be explored with the aid of correlating gross and histologic pathology. This may help elucidate not only the roentgen features but also the possible end results in such lesions such as cavitation, calcification, or progressive fibrosis.

A number of cases will then be analyzed in order to find distinctive roentgen features which can on occasion lead to an exact histologic diagnosis and more frequently to the most likely differential possibilities.

COURSE 507

Friday

REED P. RICE, M.D.
Durham, North Carolina

Radiologic Evaluation of the Acute Abdomen

This course is designed to illustrate the value of radiology in the evaluation of the patient with acute abdominal symptomatology.

This will include the value of the plain abdominal roentgenogram in the specific diagnosis of diseases, but will also provide a guide to the contrast examination of choice in appropriate situations.

An aggressive approach to the use of simple contrast examinations can result in significantly earlier diagnosis.

COURSE 508

Friday

JOSEPH P. KRISS, M.D.
Stanford, California

Current Nuclear Medicine Studies for the Evaluation of Thyroid Disease

A discussion of current diagnostic procedures employed in the evaluation of thyroid disease will be given. Particular emphasis will be directed toward those *in vivo* and *in vitro* procedures which use a radiopharmaceutical. Comparison of the clinical value and the reliability of these procedures will be reviewed where such comparisons are indicated.

As time permits current concepts in the treatment of hyperthyroidism with emphasis on treatment with radioiodine will be reviewed.

COURSE 509

Friday

NORMAN E. LEEDS, M.D.
Bronx, New York

Circulatory Changes During Cerebral Angiography as a Guide to Localization and Diagnosis of Intracranial Space Occupying Lesions

"Abnormalities in circulatory dynamics" such as delayed filling arteries, delayed filling veins, early filling veins and tumor circulation with and without early filling veins occur in 92 per cent of patients examined *via* cerebral angiography and are, therefore, most important. Normal circulatory patterns will be discussed and then the various abnormal vascular patterns will be presented as a guide to localization and histologic diagnosis.

Tumor blushes in the common lesions such as glioma, meningioma and metastases will be analyzed in an attempt to point out the differential patterns, so that one may discriminate between these lesions.

The "luxury perfusion syndrome" will be presented and the phenomena explained. The multiple pathologic entities (including infarcts, trauma, inflammatory disease, etc.) that produce this syndrome will be demonstrated, and the radiographic changes that permit differentiating these vascular blushes from tumor will be considered.

The changes in blood vessel walls such as arterial narrowing, arterial irregularity and vasodilatation will be presented and the probable causes and pathogenesis will be discussed.

CONDENSED SCHEDULE OF COURSES

MONDAY 3:00 to 4:30 P.M. **September 23, 1974**

101—Marshak
The Small Intestine

102—Martel
Commonly Confused Skeletal Lesions

103—Macmillan
The Temporal Bone

104—Wolfe
Mammography—Part I

105—Gehweiler
Osborne
Pitfalls in the Diagnosis of Vertebral Trauma—Part I

106—Fraser
Immunologic Diseases of the Thorax with Emphasis on Their Roentgenographic Manifestations

107—Cowan
Maynard
Dynamic and Static Plain Imaging

108—Schwarz
Ten Most Frequent Mistakes Made in Reading Paranasal Sinus Films

109—Kieffer
Myelography and Clivography

110—Strax
Mass Screening for Earlier Detection of Breast Cancer

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **September 24, 1974**

201—Marshak
Stomach and Duodenum

202—Jacobson
Siegelman
Skeletal Radiology—Interesting Challenges—Part I

203—Macmillan
Sinuses and Orbits

204—Wolfe
Mammography—Part II

205—Gehweiler
Osborne
Pitfalls in the Diagnosis of Vertebral Trauma—Part II

206—Felson
Reeder
The Gamut Approach to Radiology

207—Singleton
Pediatric Pulmonary Abnormalities

208—Chase
Kricheff
The Angiographic Diagnosis of Intracranial Mass Lesions

209—Baker
Berdon
Gastrointestinal Radiology in Infants and Children, Other than Newborn Intestinal Obstruction

210—Greenspan
Emphysema and Other Conditions Associated with Air Trapping

211—Swischuk
Neonatal Chest Problems

212—Conway
Pediatric Nuclear Medicine: The Central Nervous System

213—Lalli
Destructive Lesions of the Renal Parenchyma

214—Lane
Staab
Urinary Tract and Placental Nuclear Medicine Studies

215—Grossman
Renal Masses in Children

CONDENSED SCHEDULE OF COURSES

WEDNESDAY **3:00 to 4:30 P.M.** **September 25, 1974**

301—Shopfner
Current Status of Pediatric Radiology

302—Jacobson
Siegelman
Skeletal Radiology—Interesting Challenges—Part II

303—Graham
Persistent Myths which Confuse and Complicate Children's Radiology

304—Egan
Detection of Early Breast Cancer

305—Etter
Plain Film Examination of the Skull

306—Felson
Pattern Recognition of Pulmonary Lesions

307—Friedell
Radiologic Information—Are There Trade-offs?

308—Reese
Computerized Trans-Axial Tomography

309—McConnell
Wilkinson
Radionuclide Imaging Studies for Evaluating the Liver, Spleen, Pancreas and Skeletal System and for Tumor Localization

310—James
White
The Correlation of Radiographic and Radioisotopic Imaging: with Particular Emphasis on Chest Disease

311—Chait
Abdominal Angiography
Part I

312—Kaye
Arthrography—Part I
Shoulder and Hip

313—King
Abdominal and Pelvic Ultrasonography
Part I

314—Johnson
Cardiac Ultrasonography

315—Brinker
Skucas
Radiology Special Procedure Room

SECTION ON INSTRUCTION
ORDER SHEET

It is important to register for the Instruction Courses as early as possible since the number admitted to each course will be limited. *It is also very important that first, second and third choices be listed for each period.* All orders for tickets will be filled according to the postmark on the envelopes.

Nonmembers of the American Roentgen Ray Society will pay $3.00 for each course-period. *Nonmembers' fees must accompany this Order Sheet and will not be returned unless cancellation is received before September 14, 1974.*

Members of the American Roentgen Ray Society, residents in Radiology, and nonmembers who are contributing to the program, either by way of an instruction course, a paper or a *scientific* exhibit, are not required to pay for these courses but must fill out these Pink Order Sheets, indicating their choices.

Tickets for courses will be mailed to each registrant whose order reaches the office of the Director on or before September 14, 1974. They will be sent to the address below unless registrant directs otherwise

Please fill out the following (type or print):

....
Last Name First Name or Initials

.....
Street Address

....
City State Zip Code

Check
- ☐ Member
- ☐ Guest
- ☐ Resident in Radiology at: .....
- ☐ Scientific Exhibitor/Paper

Those whose advance registration reaches the office of the Director after September 14, 1974 may obtain their tickets at the Registration Desk of the Section on Instruction on Monday morning, Sept. 23 and thereafter. At this time tickets may also be obtained for courses not sold out.

For convenience in selecting your courses consult the condensed schedule of courses.

BE SURE TO FILL OUT SECOND AND THIRD CHOICES FOR EACH PERIOD

Period	First Choice		Second Choice		Third Choice	
	Course No.	Instructor	Course No.	Instructor	Course No.	Instructor
Monday						
Tuesday						
Wednesday						
Thursday						
Friday						

Send this order to:
DR. HAROLD O. PETERSON, 1995 West County Road "B", St. Paul, Minnesota 55113
OR if you are mailing late, send to:
DR. HAROLD O. PETERSON, The San Francisco Hilton Hotel,
Manson and O'Farrell Sts.
San Francisco, CA 94109

CONDENSED SCHEDULE OF COURSES

THURSDAY **3:00 to 4:30 P.M.** **September 26, 1974**

401—Whalen
Radiology of the Abdomen: An Anatomic Approach

402—Martin
Mammography

403—Dolan
Sinuses and Orbits

404—Heitzman
Chronic Interstitial Pneumonia

405—Potts
Brain Anatomy for the Radiologist

406—Steinbach
Metabolic, Endocrine and Related Diseases

407—Weigen
Complications of Diagnostic Radiology

408—Chen
Essentials of Cardiac Fluoroscopy

409—Ashburn
Nuclear Imaging—Evaluation of Cardiac Diseases

410—Harris
The Choice, Management, and Clinical Use of Radionuclide Imaging Instrumentation

411—Chait
Abdominal Angiography
Part II

412—Kaye
Arthrography—Part II
Knee and Ankle

413—King
Abdominal and Pelvic Ultrasonography
Part II

414—Hawes
The 20 Significant X-Ray Signs of Pulmonary Disease

CONDENSED SCHEDULE OF COURSES		
FRIDAY	**11:00 A.M. to 12:30 P.M.**	**September 27, 1974**
501—Dunbar Bronchial Disease in Pediatric Radiology		
502—Dodd Thermography of the Breast		
503—Margulis Inflammatory Diseases of the Colon		
504—Potter Tomography of the Temporal Bone		
505—Theros **Reed** Fundamental Patterns in Chest Roentgenology: from Nodules to Masses		
506—Steinbach Metabolic, Endocrine and Related Diseases		
507—Rice Radiologic Evaluation of the Acute Abdomen		
508—Kriss Current Nuclear Medicine Studies for the Evaluation of Thyroid Disease		
509—Leeds Circulatory Changes During Cerebral Angiography as a Guide to Localization and Diagnosis of Intracranial Space Occupying Lesions		

NEWS ITEMS

AMERICAN RADIUM SOCIETY ELECTS NEW OFFICERS

Following are the elected officers of the American Radium Society for 1974–75: Victor Marcial, *President;* Felix N. Rutledge, *President-Elect;* Alfred Ketcham, *First Vice-President;* Morris Wizenberg, *Second Vice-President;* Richard Jesse, *Secretary;* Luther W. Brady, *Treasurer,* The Hahnemann Medical College & Hospital of Philadelphia, 230 North Broad Street, Philadelphia, Pennsylvania 19102.

The Board members are: John V. Blady, Chairman, Antolin Raventos and Jerome M. Vaeth.

SOCIETY OF GASTROINTESTINAL RADIOLOGISTS

The Society of Gastrointestinal Radiologists will have their Annual Meeting in conjunction with the American Roentgen Ray Society Meeting.

The Meeting will be held on September 21, 1974, at the Hilton Hotel in San Francisco, California.

The Officers for the coming year are: *President,* William B. Seaman, M.D.; *President-Elect,* H. Joachim Burhenne, M.D.; *Secretary-Treasurer,* John R. Amber, M.D., Vanderbilt University Hospital, Nashville, Tennessee 37232; *Immediate Past President,* Alexander Margulis, M.D.; *Councillors,* Bernard Wolf, M.D., and Sidney Nelson, M.D.

SYMPOSIUM ON POLYTOMOGRAPHY OF THE TEMPORAL BONE

The 10th two-day Symposium on Polytomography of the Temporal Bone will be given under the auspices of The Wright Institute of Otology at Community Hospital, Indianapolis, Indiana on September 28 and 29, 1974.

The subjects to be covered are: "Basic Anatomy of the Temporal Bone" and "Technique of Polytomography of the Temporal Bone" with demonstrations of normal tomograms. Pathologic conditions revealed by polytomography, such as cholesteatoma, ossicular chain problems, otosclerosis, fractures, foreign bodies, tumors, and congenital anomalies are shown on original tomograms and the clinical applications discussed.

The number of registrants is limited to 18.

Inquiries should be directed to: The Wright Institute of Otology, Inc., Community Hospital of Indianapolis, Inc., 1500 North Ritter Avenue, Indianapolis, Indiana 46219.

AMERICAN NUCLEAR SOCIETY (ANS) ISOTOPES AND RADIATION DIVISION (IRD)

The Isotopes and Radiation Division of the American Nuclear Society will sponsor several sessions on the applications of isotopes and radiation in the fields of nuclear medicine, therapy, and biomedicine at the ANS Winter Meeting in Washington, D. C., October 27–November 1, 1974.

The IRD is planning to provide a unified forum for doctors, physicists, engineers and health physicists on a regular basis, should this pilot conference be successful.

Participation to this meeting is invited simply by contributing summaries of papers for personal presentation.

The broad categories of subject matter will shortly be published in the ANS "Call for Papers" and will include the following: 1. Radiation Sources, Detection, Applications; 2. Isotopes and Encapsulation; 3. Isotopic Biological Applications; 4. Materials Processing Through Radiation; 5. Isotope and Radiation Safeguards; 6. Implantable Nuclear Therapeutic Systems; 7. New Applications of Stable Isotopes; 8. Use of Accelerators in Radiation Therapy; and 9. Nuclear Medicine and Dosimetry.

The summary (600 words or less) deadline for the Washington Conference is July 8, 1974.

For further information, please contact Werner K. Kern, Technical Program Chairman, IRD, Space Nuclear Systems, Mail Station F-309, U. S. Atomic Energy Com-

mission, Washington, D. C. 20545 (phone 301-973-3321).

CONGRESSUS TERTIUS SOCIETATIS RADIOLOGICAE EUROPAEAE

The 3rd Congress of the European Association of Radiology will be held in Edinburgh, Scotland, June 22–27, 1975.

A Preliminary Announcement was published earlier this year and preliminary registration cards should have been returned to the Secretariat.

In July 1974 the Provisional Program will be sent directly to all those who have returned preliminary registration cards, but others may obtain copies either from the Secretaries of National organizations or from the Secretary General of the 3rd Congress of the European Association of Radiology, P.O. Box 14, 79 Lauriston Place, Edinburgh EH1 1DB. This Program contains information on the Scientific and Social program, together with registration forms and applications for the presentation of scientific papers, exhibits and films.

The closing date for registration is October 31, 1974 and it is stressed that it may not be possible to accept requests for late registration.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY

The Second Asian and Oceanian Congress of Radiology will be held in Manila, Philippines, November 10–14, 1975. Host organization is the Philippine College of Radiology, with the cooperation of the Philippine Medical Association.

Organizers have chosen as venue Manila, the commercial, social, educational and cultural center of the Philippines, which offers facilities most conducive to a successful convention and a comfortable stay. Located centrally in Asia, Manila is easily accessible by sea and by air to Congress members from all Asian and Oceanian capitals. History has made it a unique blend of eastern and western civilizations. Nowhere in Asia, nor perhaps in the entire world, does one see such an easy and cheerful blending of Oriental and Occidental cultures. The Greater Manila area, with a population of over 3 million people, includes several adjacent towns and cities. There are modern and fashionable business and shopping districts and interesting historical spots that combine to make visitors' stay enjoyable.

Scientific papers dealing with any subject relevant to Radiology, Radiotherapy, or Nuclear Medicine will be accepted, and should reach the Secretariat before February 28, 1975.

The official language of the Congress will be English.

Scientific and commercial exhibitions near the lecture halls will be open throughout the period of the Congress. Adequate space will be provided to those wishing to take part.

Reservations have been made and Congress members will be accommodated in a number of hotels offering excellent facilities.

The Executive Committee of the Congress is Dr. Federico V. Principe, *President-Elect* and *Chairman*, Organizing Committee; Dr. Buenaventura U. V. Angtuaco, *Secretary*; Dr. Edmundo V. Villacorta, *Treasurer*; Dr. Reynaldo G. Gregorio, *President, PCR* and Dr. Leon R. Lopez.

For further information please write to: Congress Secretariat Headquarters, 2161 Taft Avenue, Manila, Philippines.

NATIONAL PROSTATIC CANCER PROJECT

The National Cancer Institute has initiated an Extramural grant Organ Site program which consists of grant-supported National Projects of planned and targeted research.

Research proposals are being solicited without limitation to specific biologic disciplines for fundamental and clinical studies listed under broad categories of Etiology/Prevention, Detection/Diagnosis and Treatment of prostatic cancer.

For further information please contact the Headquarters Office of the National Prostatic Cancer Project, located at Roswell Park Memorial Institute, 666 Elm Street, Buffalo, New York 14203.

BOOK REVIEWS

Coronary Angiography. By Harold A. Baltaxe, M.D., Associate Professor of Radiology, Cornell Medical College; Head, Division of Cardiovascular Radiology, New York Hospital, New York, N. Y.; Kurt Amplatz, M.D., Professor of Radiology, University of Minnesota Medical School; Chief, Cardiovascular Radiology, University of Minnesota Hospital, Minneapolis, Minn.; and David C. Levin, M.D., Assistant Professor of Radiology, Cornell Medical College, New York, N. Y. Cloth. Pp. 239, with many figures. Price, $22.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

The purpose of this monograph is to provide a "practical" guide to the interpretation of normal and abnormal radiographic anatomy of the coronary arteries. The authors accomplished their task and furthermore they kept the text short enough to permit reading it within 1 or 2 evenings.

After a brief history of coronary arteriography, the procedures for selectively catheterizing the coronary arteries by cut down or percutaneous techniques are described. An excellent series of line drawings of the left and right coronary artery anatomy in frontal, lateral and both oblique projections are correlated with angiograms. The angiographic reproductions from logEtronic reduction of roentgenograms and single frame cineangiograms are not always of highest quality, but otherwise this section is good.

There are chapters on congenital anomalies, the effects of atherosclerosis on the coronary arteries and the post surgical coronary angiograms. A systematic classification and demonstration of the potential collateral pathways between the left and right coronary artery will prove extremely useful. The section on left ventriculography suggests that some angiographers prefer doing the left ventriculogram immediately before coronary angiography. At the present time there is an overwhelming consensus that cine ventriculography should be done immediately prior to the angiographic evaluation of the coronary arteries, since the contrast material used for coronary angiograms depresses myocardial contractility and may yield a false impression of worse left ventricular function than really exists.

The chapter on complications, contraindications and indications quotes a recent article in which greater surface roughness of polyurethene compared to polyethylene catheters was supposed to have been demonstrated by scanning electron microscopy. Unfortunately, a subsequent letter to the Editor and retraction by the original authors in that Journal showed that in fact polyurethene had a smoother surface than polyethylene. While polyurethene may have a greater potential for initiating thrombogenesis on its surface than polyethylene, greater surface roughness of polyurethene has not been demonstrated as a potential cause for this.

All in all, this book accomplishes the purposes the authors set forth in their preface. This text should prove valuable to all "beginning" coronary angiographers and to all "old-time" angiographers and non-angiographers who desire a review of this subject.—Robert I. White, Jr., M.D.

The Aortic Arch and Its Malformations: with Emphasis on the Angiographic Features. By Wade H. Shuford, M.D., Professor of Radiology, Emory University School of Medicine; Director, Division of Diagnostic Roentgenology, Grady Memorial Hospital, Atlanta, Ga.; and Robert G. Sybers, M.D., Ph.D., Professor of Radiology, Emory University School of Medicine; Director of Radiology Teaching Program for Interns and Medical Students, Emory University School of Medicine and Grady Memorial Hospital; Radiologist, Department of Radiology, Grady Memorial Hospital, Atlanta, Ga. Medical Illustrations, Grover B. Hogan, B.S., M.S. Cloth. Pp. 264, with many illustrations. Price, $19.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

This well written, organized, and illustrated monograph on the aortic arch is very comprehensive.

It covers the aorta from a developmental viewpoint to the time of a clinical discovery of malformations in infants and adults. The excel-

lent angiograms are further elucidated by line sketches of the underlying deviation of left or right arch and double aortic arch.

The significance of the resultant opacification is adequately discussed in terms of function, arteriovenous mixing, cardiac competence and clinical presentations.

There is a wide variety of illustrations of congenital deviations of the arch and a distinct absence of traumatic and aneurysmal material.

This "dutifulfilled" book is highly recommended for budding cardiologists and roentgenologists.—Jan Nyboer, M.D.

BOOKS RECEIVED

The Hand in Radiologic Diagnosis. By Andrew K. Poznanski, M.D., Professor of Radiology, The University of Michigan; Co-Director of Pediatric Radiology, C. S. Mott Children's Hospital, The University of Michigan; Research Scientist, Center for Human Growth and Development, The University of Michigan, Ann Arbor, Mich. Cloth. Pp. 588, with many illustrations. Price, $28.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1974.

Radioactive Isotopes in Biological Research. By William R. Hendee, Associate Professor of Radiology, University of Colorado Medical Center, Denver, Colo. Cloth. Pp. 356, with some figures. John Wiley & Sons, Inc., Publishers, 605 Third Avenue, New York, N. Y. 10016, 1973.

Radioassay in Clinical Medicine. Edited by William T. Newton, M.D., Professor of Surgery, Washington University School of Medicine; Chief, Surgical Service, St. Louis Veterans Administration Hospital, St. Louis, Mo.; and Robert M. Donati, M.D., Associate Professor of Medicine; Director of the Section of Nuclear Medicine, St. Louis University School of Medicine; Chief, Nuclear Medicine Service, St. Louis Veterans Administration Hospital, St. Louis, Mo. Cloth. Pp. 187, with some figures. Price, $11.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1974.

Communication Disorders: Remedial Principles and Practices. General Editor, Stanley Dickson, State University College at Buffalo, N. Y. Cloth. Pp. 520. Scott, Foresman and Company, Glenview, Ill., 1974.

Illustrated Manual of Ultrasonography in Obstetrics and Gynecology. By Mitsunao Kobayashi, M.D., Department of Obstetrics and Gynecology, Chiba University School of Medicine, Chiba, Japan; Former Assistant Professor, Department of Obstetrics and Gynecology, State University of New York Downstate Medical Center, Brooklyn, N. Y. Cloth. Pp. 316, with 674 illustrations. Price, $32.00. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1974.

Endocrinology of the Gut. Edited by William Y. Chey, M.D., D.Sc., Professor of Medicine, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.; and Frank P. Brooks, M.D., D.Sc., Professor of Medicine and Physiology, University of Pennsylvania School of Medicine, Philadelphia, Pa. Cloth. Pp. 373, with some figures. Price, $20.00. Charles B. Slack, Inc., 6900 Grove Road, Thorofare, N. J. 08086, 1974.

The Radiologic Clinics of North America. Symposium on Obstetric and Gynecologic Radiology. Guest Editor, Melvyn H. Schreiber, M.D. April, 1974, Volume XII, No. 1. Cloth. Pp. 207, with many illustrations. Subscription price is $20.00 per year. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1974.

Fraction Size in Radiobiology and Radiotherapy. Edited by Tsutomu Sugahara, M.D., Professor, Department of Experimental Radiology, Faculty of Medicine, Kyoto University, Japan; László Révész, M.D., Professor, Department of Tumor Biology, Karolinska Institutet, Medical School, Stockholm, Sweden; and Oliver C. A. Scott, Bt., British Cancer Council, London, England. Cloth. Pp. 236, with some illustrations. Price, $23.00. The Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, exclusive U. S. agents, 1974.

The National Cancer Program and International Cancer Research. National Cancer Institute Monograph 40, February 1974. Cloth. Pp. 91, with some figures. Price, $4.70. DHEW Publication No. (NIH) 74-351. U. S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, National Cancer Institute, Bethesda, Md. 20014. For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, 1974.

Lille Médical Actualités. February, 1974, Volume 19, No. 2, Supplement 1. Paper. Pp. 136. 1974.

Notes on Radiological Emergencies. Second edition. By George Ansell, M.D., F.R.C.P., F.F.R., D.M.R.D., Consultant Radiologist in Charge, Whiston Hospital; Lecturer in Radiodiagnosis, University of Liverpool. Paper. Pp. 16. Price, $4.25. Blackwell Scientific Publications. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1974.

Dr. Turtle's Babies. By William John Turtle, M.D. Paper. Pp. 318. Price, $1.50. Popular Library, 600 Third Avenue, New York, N. Y. 10016, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

American Radium Society
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

Radiological Society of North America
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Portland-Hilton Hotel, Portland, Oregon, March 31–April 4, 1975.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Chicago, Ill., June 15–19, 1974.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next year. New York, N. Y., June 16–22, 1974, New York Hilton Hotel; and Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers June 22, 1974.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1974 was September 30, 1973.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

The third examination is scheduled for Saturday, Sept. 21, 1974. Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017. The deadline for receipt of completed applications is June 1, 1974.

American Association of Physicists in Medicine
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D. C. 20010. Annual Meeting.

American Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting: Sonesta Beach Hotel, Key Biscayne, Fla., Oct. 30–Nov. 4, 1974.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

Fourteenth International Congress of Radiology
Rio de Janeiro, Brazil in 1977.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

Eleventh Inter-American Congress of Radiology
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aéreo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

Inter-American College of Radiology
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

Third Congress of the European Association of Radiology
Meeting: June 22–27, 1975 in Edinburgh, Scotland.

Second Asian and Oceanian Congress of Radiology
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society, Chapter ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

American Thermographic Society
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting: Chicago, Ill., June 22–23, 1974.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Richard H. Greenspan, Dept. of Radiol., Yale University School of Medicine, 333 Cedar St., New Haven, Conn. 06510.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Charles J. Masters, Maj. MC, Department of the Air Force, United States Air Force Hospital, Wiesbaden (USAFE) APO New York 09220.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joshua A. Becker, Downstate Medical Clin., 450 Clarkson Ave., Brooklyn, N. Y. 11203. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Franco, 45 Alexander St., Lockport, N. Y. 14094. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alfred Brettner, 27 Hurd Circle, Auburn, N. Y. 13021. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio

CENTRAL TEXAS RADIOLOGY SOCIETY
Secretary-Treasurer, Dr. John M. Stoebner, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

CHICAGO RADIOLOGICAL SOCIETY, DIVISION OF THE ILLINOIS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

COLORADO RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

DELAWARE CHAPTER OF ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

EAST BAY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George N. Weston, 1515 Ygnacio Valley Rd., Walnut Creek, Calif. 94598. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

GEORGIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER KANSAS CITY RADIOLOGICAL SOCIETY
President-Secretary, Dr. Keith W. Gallehugh, Bethany Medical Center, Kansas City, Kans. 66102. The Society meets 6 times a year.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Richard E. Litt, 6125 S.W. 31st St., Miami, Fla. 33155. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

HAWAII RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

HEALTH PHYSICS SOCIETY
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting: Houston, Texas, July 7–11, 1974.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

IDAHO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. John A. Knote, 2600 Greenbush St., Lafayette, Ind. 47904.

IOWA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference: Century Plaza Hotel, Los Angeles, Calif., Jan. 31–Feb. 2, 1975.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Eli A. Etscovitz, 10 Stonewood Dr., Newton Centre, Mass. 02159.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Howard L. Copas, 827 South 131st St., Omaha, Neb. 68154. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

New England Roentgen Ray Society
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02119. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting: May 10, 1974.

New England Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference, New Orleans, La., Apr. 25–27, 1974.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 S. University Dr., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Mark D. Reiss, 2100 Rockwood Dr., Sacramento, Calif. 95825. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

NORTHWESTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Tearle L. Meyer, 309 E. State St., Columbus, Ohio 43215.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 22–24, 1974.

SAN ANTONIO-CIVILIAN MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President, Dr. Leland E. Kellerhouse, 2466 First Ave., San Diego, Calif. 92101. Meets the first Monday of each month at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION OF RADIOLOGY, NATIONAL MEDICAL ASSOCIATION
Chairman, Dr. Roy G. Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broad-

way, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, Vanderbilt University Hospital, Nashville, Tenn. 37232.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. 21st Annual Meeting: Town and Country Hotel, San Diego, Calif., June 10–14, 1974.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 111 Whitehall Bldg., 960 E. Third St., Chattanooga, Tenn. 37403. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada
First European Course in Chest Radiology at the Royal College of Surgeons, London, W.C.1., May 20–23, 1974.
Hon. Secretary, Dr. Ian H. Kerr, 156 Lambeth Rd., London, S.E.1.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton, St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 8952 Bellfonte Rd., Richmond, Va. 23229.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Isaiah J. Seligman, 16 Guion Pl., New Rochelle, N. Y. 10801. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1 Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32

Welbeck St., London, W1M/7PG, England. Annual General Meeting: May 15–18, 1974.

CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St. Catherine St. West, Montreal 107, Que., Canada. Thirty-eighth Annual Meeting.

EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

FACULTY OF RADIOLOGISTS
Honorary Secretary, Robert Morrison, Lincolns Inn Fields, London WC2A 3PN, England. Annual General Meeting: May 15–18, 1974.

MONTREAL RADIOLOGICAL STUDY CLUB
Secretary, Dr. W. Paul Butt, Montreal General Hospital Montreal, Que., Canada. Meets first Tuesday evening, October to April.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

SOCIÉTÉ CANADIENNE-FRANÇAISE DE RADIOLOGIE
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

THE HOSPITAL PHYSICISTS' ASSOCIATION
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

THE ROYAL AUSTRALASIAN COLLEGE OF RADIOLOGISTS
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

SOUTH AMERICA

COLÉGIO BRASILEIRO DE RADIOLOGIA
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

FEDERACIÓN ARGENTINA DE SOCIEDADES DE RADIOLOGÍA
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

SOCIEDAD ARGENTINA DE RADIOLOGÍA
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

SOCIEDAD COLOMBIANA DE RADIOLOGIA
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

SOCIEDAD ECUATORIANA DE RADIOLOGÍA
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

SOCIEDAD PERUANA DE RADIOLOGIA
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

SOCIEDAD DE RADIOLOGÍA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

SOCIEDAD DE RADIOLOGÍA DEL NORDESTE ARGENTINO
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

SOCIEDAD DE RADIOLOGÍA DE LA PLATA
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

SOCIEDADE DE RADIOLOGÍA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

SOCIEDAD DE ROENTGENOLOGÍA Y MEDICINA NUCLEAR DE LA PROVINCIA DE CÓRDOBA
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

SOCIEDAD DE RADIOLOGÍA, RADIOTERAPÉUTICA Y MEDICINA NUCLEAR, DE ROSARIO
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

SOCIEDAD SALTEÑA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

SOCIEDAD VENEZOLANA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

CONTINENTAL EUROPE

ÖSTERREICHISCHE RÖNTGEN-GESELLSCHAFT
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

SOCIÉTÉ ROYALE BELGE DE RADIOLOGIE
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

SOCIÉTÉ EUROPÉENNE DE RADIOLOGIE PÉDIATRIQUE
Secretary, Dr. J. Jääskeläinen, The Children's Hospital,

Stenväckinkatu 11, SF-00290 Helsinki 29, Finland. Annual Meeting.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting in Uppsala, Sweden in June 1974.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa 1-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

HEAD

BERRY, H. M., JR.: The lore and the lure of the lamina dura 697

NECK AND CHEST

RAM, M. D. *et al.:* Thyroidography and thyrolymphography 697
SAROSI, G. A. *et al.:* Clinical features of acute pulmonary blastomycosis 698
SPENGLER, D. M. *et al.:* Orthopaedic aspects and early diagnosis of superior sulcus tumor of lung (Pancoast) 698

ABDOMEN

ST. JOHN, D. J. B. *et al.:* The Mallory-Weiss syndrome 699
LEWICKI, A. M. *et al.:* The small bowel following pyloroplasty and vagotomy 699
ROKA, R. *et al.:* Peutz-Touraine-Jeghers syndrome 700
MEISSNER, K. *et al.:* The reliability of negative angiography in clinically suspected splenic rupture 700

GENITOURINARY SYSTEM

CZEKALA, Z.: Arteriographic symptoms of renal tuberculosis 701
LEOPOLD, G. R. *et al.:* Renal ultrasonography: an updated approach to the diagnosis of renal cyst 701
PHILLIPS, J. C.: A spectrum of radiologic abnormalities due to tubo-ovarian abscess 702

NERVOUS SYSTEM

WINTER, R. B. *et al.:* Diastematomyelia and congenital spine deformities 702

SKELETAL SYSTEM

TENNER, R., and ENGELBRECHT, E.: The artificial hip joint from the roentgenologic standpoint 702
BROOKER, A. F. *et al.:* Ectopic ossification following total hip replacement: incidence and a method of classification 703
SMITH, R. W., and SMITH, C. F.: Solitary unicameral bone cyst of the calcaneus: a review of twenty cases 703
ZAGNOLI, P., and ROMANI, F.: The syndrome of the arcuate ligament 703
GOHEL, V. K. *et al.:* The serpiginous tract: a sign of subacute osteomyelitis 704

ROENTGEN DIAGNOSIS

Head

Berry, Harrison M., Jr. The lore and the lure of the lamina dura. *Radiology*, Dec., 1973, *109*, 525–528. (Address: Department of Radiology, School of Dental Medicine, University of Pennsylvania, and the Hospital of the University of Pennsylvania, Philadelphia, Pa.)

The lamina dura is a hard bony lining of the alveolus, or socket, of a tooth. Together with the periodontal ligament and the cementum, which coats the tooth root, it forms the attachment apparatus of the tooth.

An epithelial cord, the dental lamina, proliferates from the oral epithelium into the subjacent connective tissue and differentiates into a bell-shaped enamel organ. The lower border of epithelial cells—ameloblasts—form enamel, while a specialized group of connective tissue cells—odontoblasts—line up in apposition to the ameloblasts and produce dentin. These processes take place in a chamber within the bone known as a developmental crypt, characterized radiographically as a round radiolucency with a radiopaque border. Usually at the time a crypt appears in a roentgenogram, some calcification of enamel can be seen within it. Occasionally, the crypt appearance may precede calcification and the crypt will look exactly like a cyst. As calcification of the dentin root structure progresses within the crypt, the tooth bud becomes "wrapped" in specialized circular connective tissue fibers—the dental sac. As dentin is forming apical to the cervical line of the enamel, the circularly arranged fibers of the dental sac become the periodontal ligament around the tooth root. The periodontal ligament produces the cementum which covers the root dentin and the lamina dura of the tooth socket. As the cementum and lamina dura are being produced about the forming root, fibers of the periodontal ligament become entrapped in their substance. The attachment of the periodontal ligament fibers in the lamina dura and in the cementum holds the tooth securely in the socket.

Although the lamina dura has the radiopaque quality of cortical bone in a roentgenogram, it is more precisely a cribriform (sieve-like) plate of bundle bone. The name cribriform is applied because the bone is perforated by a great number of Volkmann's canals which are conduits containing nerves and blood and lymph vessels supplying the periodontal ligament. Because of the great number of Sharpey's fibers, and because lamellae are less conspicuous, this bone is called bundle bone. Bundle bone also demonstrates differences in matrix composition.

Following the extraction of a tooth, when the lamina dura has no further function, it disappears completely and forever. The daily stresses of normal living cause portions of the enamel on the tooth crown to slowly wear away. The teeth normally "drift" to remain in contact and show further "passive eruption" to maintain occlusal contact. As the stress is decreased on one side of the tooth and increased on the other, corresponding changes may take place in the calcification of the socket walls and it may be impossible to trace the lamina dura completely around each tooth. Additionally, periodontal diseases may destroy portions of the lamina dura.

Dental roentgenograms of all areas of the jaws are essential diagnostic aids to help rule out hyperparathyroidism and other systemic diseases. Dental diagnosis of hyperparathyroidism is more accurate, when all the periodontal structures are considered rather than just the lamina dura. The periodontal ligament appears as a dark area surrounding the roots. The lamina dura is seen as a dense white line of bone around the periodontal ligament. Subperiosteal bone resorption of the cortex as seen in hyperparathyroidism, therefore, appears as loss of lamina dura. It is not as specific as subcortical resorption in phalanges. Absence of the lamina dura can also occur in Paget's disease, osteomalacia, osteoporosis, Cushing's disease, myelomatosis, leukemia and metastases, and it is not pathognomonic of hyperparathyroidism.—*Jon F. Bergstrom, M.D.*

Neck and Chest

Ram, M. D., Archer, Bernard T., and Brown, Henry W. Thyroidography and thyrolymphography. *Surg., Gynec. & Obst.*, March, 1974, *138*, 417–420. (From: The Departments of Surgery and Radiology, Huron Road Hospital, Cleveland, Ohio.)

Imaging of the total thyroid gland can be accomplished by direct injection of low viscosity oil directly into the substance of the gland. Unlike scanning procedures the appearance of the image is independent of function, and will demonstrate the extent and structure of the entire gland, and also the lymphatic drainage, which is often more extensive than has been recognized.

After skin anesthesia a 23 gauge needle is introduced into a lobe of the thyroid gland, and its position is checked by watching motion during swallowing. After aspiration, to be sure the needle is not in a blood vessel, 1 to 2 ml. of iodized oil is injected over a 5 minute interval. Fluoroscopy during injection demonstrates the part of the lobe being opacified, leak beyond the capsule, and spread through the lobe. The direction of the needle can be changed to obtain filling of the entire lobe. The procedure is repeated for the other lobe.

At 15 minutes the entire gland and isthmus were filled, and at 24 hours the lymph nodes were filled. On follow-up films only the lymph nodes were filled. Twelve patients admitted for thyroid operation were

studied by this method. Diagnoses were as follows: hyperthyroidism 3; simple goiter 2; multinodular goiter 2; single nodule 2; malignant disease 2; and Hashimoto's disease 1. In 2 of the patients with hyperthyroidism the lymphatics emptied into the jugular veins, requiring a change in position of the needle. These patients did not have any adverse effects, and the roentgenograms of the lungs did not show any changes, probably because of the small amount of contrast material. The hyperthyroid glands were enlarged, probably because of pretreatment with propyl thiouracil, and later films showed lymphadenopathy involving the cervical and mediastinal lymph nodes. In 1 an unsuspected small cyst was demonstrated. The lymphovenous connections may be due to greater secretory function. Simple goiters showed a fine reticular pattern throughout, and only the cervical lymph nodes were visualized. In multinodular goiters, some of the nodules were seen as negative defects and some filled with the opaque material. In 3 patients with Hodgkin's or malignant disease, injection was difficult and required more pressure. In 1 the reticular pattern of the lobe was lost, and an irregular and puddling appearance was demonstrated. In the presence of malignant disease the delayed roentgenograms may show the involvement of the lymph nodes before they are palpably enlarged.

In this study the thyroid gland did not show any reaction to the contrast medium, but in a study of dogs by Sterns and Doris a significant lipogranulomatous reaction was noted in 10 per cent of the animals after 100 days.—*Lois Cowan Collins, M.D.*

SAROSI, GEORGE A., HAMMERMAN, KENNETH J., TOSH, FRED E., and KRONENBERG, RICHARD S. Clinical features of acute pulmonary blastomycosis. *New England J. Med.*, March, 1974, *290*, 540–543. (Address: George A. Sarosi, M.D., Veterans Administration Hospital (111), 54th Street and 48th Avenue South, Minneapolis, Minn. 55417.)

In October and November, 1972, 18 adults and children of Bigfork, Minn. were found to have evidence of infection with *B. dermatitidis* (blastomycosis). All were members of 4 families who were engaged in building a small cabin in a wooded area. Although repeated attempts to isolate the fungus from the environs of the cabin failed, the clear cut association of all symptomatic patients with the cabin justified calling this a common source epidemic. Of the 21 persons exposed 16 had positive blastomycin skin tests, 7 of whom were symptomatic and 9 were asymptomatic. Only 4, 2 in each group, had positive complement fixation tests. In a community wide survey, there were only 7 reactors to the blastomycin skin test among 395 tested.

The symptomatic patients presented with influenza-like illness of varying severity. All had abnormal chest roentgenograms, and in 4 *B. dermatitidis* was recovered from the sputum. Of 11 patients without symptoms the chest roentgenograms were positive in 5. None of the chest roentgenograms showed evidence of pleural disease, although several had pleuritic chest pain. The pulmonary findings on roentgenologic examination were nonspecific.

None of the patients received specific antifungus therapy, because the symptomatic patients were already beginning to improve when the diagnosis was established. All had complete recovery, symptomatically and radiographically. Apparently blastomycosis may occur as an acute self limited pulmonary infection, similar to Histoplasma and Coccidioides infections.—*Lois Cowan Collins, M.D.*

SPENGLER, DAN M., KIRSH, MARVIN M., and KAUFER, HERBERT. Orthopaedic aspects and early diagnosis of superior sulcus tumor of lung (Pancoast). *J. Bone & Joint Surg.*, Dec., 1973, *55-A*, 1645–1650. (Address: Dan M. Spengler, M.D., University of Michigan Medical Center, Ann Arbor, Mich. 48014.)

The medical records of 51 patients with a mean age of 56 years were reviewed retrospectively to emphasize predominantly musculo-skeletal presenting systems of patients with superior sulcus tumor. There were 45 males and 16 females and all but 2 patients had a history of cigarette smoking. Forty-nine of the 51 patients had histologically confirmed tumor, with 20 being squamous cell carcinomas, 13 undifferentiated carcinomas and 11 adenocarcinomas. There was 1 case each of giant cell carcinoma, medullary carcinoma and fibrous mesothelioma.

The superior pulmonary sulcus is a groove in the lung formed by the subclavian artery as it crosses the apex of the lung in the cupola of the pleura. Because most apical lung tumors arise in relation to the sulcus, they are frequently called superior sulcus tumors. The superior inlet of the thorax is bounded laterally by the first rib, anteriorly by the first costal cartilage and the manubrium and posteriorly by the head of the first rib and body of the first thoracic vertebra. Most of the area is occupied by the apex of the lung. Important structures within the superior thoracic inlet are from anterior to posterior, the subclavian and jugular veins, the phrenic and vagus nerves, the subclavian and common carotid arteries, the recurrent laryngeal nerve, the 8th cervical and first thoracic nerves and the sympathetic chain and stellate ganglion. The superior sulcus tumor may involve any combination of these structures and therefore the signs and symptoms are varied and complex.

Pain was a presenting symptom in all patients and the average duration of symptoms prior to diagnosis was 6.8 months. Forty-four patients had shoulder pain, and brachialgia suggestive of brachial plexus involvement occurred in 29 patients. The intensity

of the pain was variable, but often described as constant and worse at night. Significant weight loss occurred in one-half of the patients and all but 2 had a history of cigarette smoking. Infrequently patients had an initial complaint of hoarseness, dyspnea or cough. Physical examination was normal in 21 patients; 12 patients had supraclavicular fullness; and 19 presented with Horner's syndrome which was the single most important physical finding leading to a correct diagnosis. Prominence of the superficial jugular venous system, discoloration of the upper extremity and edema characteristic of early venous occlusion were not uncommon. Chest auscultation yielded no consistent findings.

The most frequent cause for delay in diagnosis appeared to be simply failure to include superior sulcus tumor in the differential diagnosis. Initial roentgenographic studies demonstrated erosion of a rib or vertebra in 23 patients. An equal number were initially treated under presumptive diagnosis of shoulder bursitis, degenerative disk disease of the cervical spine or thoracic inlet syndrome. Four patients even underwent laminectomy and foraminotomy because of neck pain radiating to the upper extremity.

The authors wish to emphasize that the diagnosis of superior sulcus tumor must be considered whenever a patient has shoulder pain and initial roentgenograms of the shoulder or cervical spine must be carefully viewed to exclude the presence of superior sulcus tumor. Patients with persistent shoulder pain should have a full-size chest roentgenogram with lordotic views and thoracic spine roentgenograms.—*John T. Underberg, M.D.*

Abdomen

St. John, D. J. B., Masterton, J. P., Yeomans, N. D., and Dudley, H. A. F. The Mallory-Weiss syndrome. *Brit. M. J.*, Jan., 1974, *1*, 140–143. (From: Monash University Medical School, Alfred Hospital, Melbourne 3181, Australia.)

The criteria for diagnosis of the Mallory-Weiss syndrome have been widened to include cases where hematemesis has been caused by spontaneous mucosal tearing at the gastroesophageal junction regardless of the precipitating factor. In review of 121 patients admitted to Monash University Medical Hospital, Melbourne, Australia, for major upper gastrointestinal bleeding the diagnosis of Mallory-Weiss syndrome was established in 16. Endoscopy with fiber-endoscopes was performed on 90 of 121 patients within 24 hours of admission. The criterion for the diagnosis was the finding of single or multiple longitudinal mucosal tears at, or immediately below the gastroesophageal junction, irrespective of the presence or absence of a history of vomiting before hematemesis. The typical history of vomiting was obtainable in only 9 of 16 patients, but recent alcohol intake was high in another 4. Establishment of the diagnosis by esophagogastroscopy was of special benefit when surgery was needed for control of continuing blood loss and it also simplified subsequent medical management of those patients in whom bleeding stopped spontaneously.

The incidence of 13.2 per cent in this series suggests that the Mallory-Weiss syndrome may be a relatively common cause of upper gastrointestinal bleeding.—*Lois Cowan Collins, M.D.*

Lewicki, Ann M., Kleinhaus, Uriel, Brooks, John R., and Membreno, Alejandro A. The small bowel following pyloroplasty and vagotomy. *Radiology*, Dec., 1973, *109*, 539–544. (Address: Ann M. Lewicki, M.D., Department of Radiology, Harvard Medical School, 25 Shattuck Street, Boston, Mass. 02115.)

When a symptomatic patient who has undergone a pyloroplasty and vagotomy presents for radiologic examination of the upper gastrointestinal tract, the primary concern is usually the possibility of recurrent ulcer disease. Little attention has been given to the radiographically detectable extragastric alterations that these surgical procedures may cause. Recently the onset of sprue has been linked to vagotomy and a gastric drainage procedure. The authors compared the small bowel roentgenograms of 51 patients, who had undergone a pyloroplasty and vagotomy at least 2 years previously, with roentgenograms of 50 normal controls.

Vagotomy combined with a gastric drainage procedure is an accepted surgical mode of treating peptic ulcer disease, particularly in the duodenum. Denervation of the stomach is usually accomplished by sectioning and excising the major anterior and posterior vagal trunks immediately below the diaphragm, which also interrupts the parasympathetic nerve supply to all gastrointestinal organs in the abdomen except the left colon.

Even in the hands of the most experienced surgeon, vagotomy may occasionally be incomplete and pyloroplasty inadequate. Therefore, the authors decided to evaluate the individual effect of each procedure upon the small bowel. In both of the subgroups, the small bowel was dilated to the same extent as in the total postsurgical group and significantly more dilated than in the control patients. Therefore, the authors concluded that the maximal dilatation in the intestine must be produced by one of the surgical procedures alone.

Although the roentgenographic changes seen in the small bowel after vagotomy or pyloroplasty may be identical, the mechanisms behind them are almost certainly different. Parasympathetic denervation of the intestine produces uncoordinated motility rather than an actual change in peristalsis or tone. Altered motility results in a dilated small bowel with re-

sultant prolonging of transit. On the other hand, disruption of the pyloric barrier may affect gastric digestion and interfere with the admixture of food with biliary and pancreatic secretions. Thus the altered composition of the intestinal content may actually be responsible for the changes seen in the diameter and motility of the intestine.

In 2 previous roentgen studies of postvagotomy patients, prolonged transit time and widening of the intestinal lumen were observed in the immediate postoperative period, but disappeared completely within 6 months after vagotomy. In the authors' study it was shown that this widening persists into the delayed postoperative period and involves all segments of the small bowel. The widening remained even after gastric motility was restored and was also seen in patients who showed normal gastric emptying. Transit time was significantly prolonged as well. Dilatation of small bowel loops was frequently accompanied by barium precipitation in emptying segments, resulting in a roentgenographic picture which is indistinguishable from that of sprue. The authors' data indicate that in a patient in whom diarrhea develops after vagotomy and pyloroplasty, it is impossible to assess, on the basis of the roentgenographic appearance of the small bowel alone, whether sprue has developed or whether the changes were due to the surgical procedures. The authors feel that absorption studies and intestinal biopsies are mandatory in these patients, so that a proper course of therapy can be instituted, particularly in those with malabsorption.—*E. J. Schoolar, M.D.*

Roka, R., Euler-Rolle, J., Depisch, D., Mandl, W., and Priesching, A. Das Peutz-Touraine-Jeghers-Syndrom. (Peutz-Touraine-Jeghers syndrome.) *Wien. klin. Wchnschr.*, Jan., 1974, *86*, 18–20. (Address: Dr. R. Roka, I. Chirurgische Universitätsklinik, Alser Strasse 4, A-1095 Wien, Austria.)

The Peutz-Touraine-Jeghers syndrome denotes intestinal polyposis associated with mucocutaneous pigmentation. In Austria it is rarely encountered. Therefore, 2 characteristic cases are reported.

The first patient, a 24 year old woman, presented with a history of dark pigmentation around both lips since the age of 6. There was constipation and marked anemia at the age of 11. At that time, she was operated on for jejunal polyps and associated intussusception. Recently, she complained of increasing pigmentation of the perioral skin and diarrhea. A fist-size mass was palpated in the left lower abdomen. At operation separate jejunal and sigmoidal intussusceptions were found together with 4 polyps. There was a definite family history of the syndrome.

The second patient, an 18 year old girl, presented with a recent history of sudden lower abdominal pain and a severe anemia. The patient appeared very pale and in peritoneal shock. There was a stippled dark-brown pigmentation of the lips, most concentrated at the angles of the mouth. At operation, an intussusception was found at the jejunoileal junction and a second one in the ileocecal region. Polyps were present at both sites. The father and 8 sisters also exhibited characteristic pigmentations of the lips and face.

Abnormal pigmentation usually develops in the first 2 years of life, progresses until puberty, and decreases later on. When far advanced, it presents a butterfly distribution of the face, but usually is concentrated around the lips and in the buccal mucosa. Occasionally, it may also be observed on fingers, toes, knees, elbows, and in the palm of the hands and the soles of the feet. The findings are sufficiently striking to allow correlation with secondary anemia, bleeding intestinal polyps, and secondary intussusception.

The intestinal polyps vary in frequency from a solitary one to several hundreds. The diameter averages 5 cm. The size can be correlated with the frequency of bleeding and intussusceptions. The polyps are mostly located in the jejunum and ileum, but can also be found throughout the intestinal tract. Complications reach a peak in the second decade of life and rarely occur past the age of 30. Histologically, the tumors differ from simple adenomatous polyps through hypertrophy of muscle fibers from the muscularis mucosae and the muscle layers involving even the pedicles. Thus, a potentially malignant invasion is simulated.

The course of the disease, aside from intussusceptions, suggests a relatively benign nature. An intestinal carcinoma, when developing later in life, now appears unrelated to the syndrome, as it occurs at locations remote from the specific polyps. Therefore, a conservative management is indicated with the treatment confined to intestinal complications and secondary anemia.—*Ernest Kraft, M.D.*

Meissner, K., Schmoller, H. J., and Suwandschieff, N. Die Aussaugekraft der negativen Angiographie bei klinischem Verdacht auf Milzruptur. (The reliability of negative angiography in clinically suspected splenic rupture.) *Wien. klin. Wchnschr.*, Oct., 1973, *85*, 712–713. (Address: Dr. K. Meissner, Landeskrankenanstalten Salzburg, Mullner Hauptstrasse 48, A-5020 Salzburg, Austria.)

The authors report 8 cases with clinically suspected splenic ruptures, who had normal splenic arteriograms: in 5 cases, conservative treatment was followed by complete recovery; in 2 cases, the absence of splenic rupture was proven by surgery, and in 1 case, by autopsy.

The radiographic approach of obtaining a reliable angiographic diagnosis is discussed. Aside from recognition of normal anatomic variants, "false positive"

findings can be reduced by employing selective injection techniques and/or oblique projections. The diagnosis of a normal splenic arteriogram should only be made in the absence of any findings suspicious of splenic rupture.

The opinion is expressed that, in applying these strict criteria, a normal splenic arteriogram excludes splenic rupture even in cases of well supported clinical suspicion.—*Peter F. Winter, M.D.*

Genitourinary System

Czekala, Zbigniew. Arteriographische Symptome der Nierentuberkulose. (Arteriographic symptoms of renal tuberculosis.) *Radiol. clin. et biol.*, 1974, *43*, 201–209. (Address: Dr. Lidia Czekala, ul. Ligonia 48/4, 40-037 Katowice, Poland.)

Renal tuberculosis occurs in 17 to 20 per cent of all extrapulmonary forms of tuberculosis. It usually develops from a remote focus as hematogeneous seeding. Less frequently, an ascending form secondary to a genital tuberculosis is encountered. This variety has become relatively infrequent. The extent of renal tuberculosis can only be properly evaluated with the aid of angiography.

During the past 6 years, 12 cases of renal tuberculosis were recognized. The diagnosis was confirmed bacteriologically and/or surgically with histologic studies. In 4 cases, both kidneys were involved; in 8, the disease had remained unilateral. The age of the patients varied from 30 to 69 years.

The angiographic findings could be divided into 4 groups:

1. *Parenchymatous tuberculosis* showed tortuosity and caliber changes of interlobar arteries, occasionally vascular occlusion, decreased branching, weak staining of tissues, and poor demarcation of the cortex.

2. *Tuberculous infarct* disclosed slender, stretched, or slightly tortuous arteries, weak staining of tissues, and sharp demarcation of the anemic segment.

3. *Cavernous tuberculosis* displayed small, smooth, stretched, or slightly curved segmental intrarenal arteries. The main artery was narrowed in widespread cavitation.

4. *Mixed form* of tuberculosis comprised those cases in which at least 2 angiographic forms could be recognized as well as cirrhotic processes.

Selective angiography has proved superior to conventional urography in silent and small foci. In more advanced stages, the method has allowed to determine the extent of the foci. The parenchymatous form is difficult to differentiate from chronic pyelonephritis. In tuberculous infarcts, tissue staining is weak, the anemic portion is sharply demarcated, but arteries can be traced to the periphery of the kidney. The cavities, unlike cysts, present with walls of varying thickness, while the normal segments show marked contrast staining. Calcific foci present irregular borders as observed in 3 cases.

Selective angiograms facilitate a control of therapeutic results and the correct choice of surgical procedures.—*Ernest Kraft, M.D.*

Leopold, George R., Talner, Lee B., Asher, W. Michael, Gosink, Barbara B., and Gittes, Ruben F. Renal ultrasonography: an updated approach to the diagnosis of renal cyst. *Radiology*, Dec., 1973, *109*, 671–678. (Address: George R. Leopold, M.D., Department of Radiology, University Hospital, 225 West Dickinson Street, San Diego, Calif. 92103.)

With the increasing population in the geriatric age range, there has been an increase in the incidence of asymptomatic renal masses. Ultrasonic B-scanning, followed by cyst puncture in cases with cysts, is an accurate method of distinguishing solid tumors from benign cysts, and at little risk and expense to the patient.

The authors review 84 cases of renal masses with proved diagnoses in which ultrasonic B-scanning was performed.

The scannings were performed at both high and low gain settings with the patient in a prone position. Transverse scans at 1 cm. intervals were obtained and the upper and lower margins of the kidneys defined. Three features indicated a renal cyst: (1) a mass almost entirely free of internal echoes; (2) smooth and sharply defined anterior margin; and (3) increased echoes on opposite side of the mass. One or 2 strong echoes appearing within a cyst usually meant a septated cyst. Solid tumors in a kidney produced many internal echoes, and irregular anterior margins, and an absence of increased echoes on the opposite side of the mass.

In accomplishing cyst puncture, ultrasound scanning localized the lesion and determined its depth. High dose urography and fluoroscopy were employed to check position and motion of kidney with respiration. Cyst puncture was performed, using a 4 inch No. 19 Teflon-sheathed needle advanced, with respiration suspended in midexpiration. Once cyst fluid was obtained, the needle was withdrawn leaving the flexible Teflon catheter in place. More cyst fluid was aspirated and sent to the laboratory for cytology and lactic dehydrogenase analysis. Aspirated fluid was replaced with urographic contrast material and roentgenograms in multiple positions were obtained. Air contrast cystography was discontinued as being unrewarding. Peripelvic cysts were not punctured routinely because of risk of vessel injury.

Cyst puncture was successful in 31 of 32 patients. Only one complication occurred: gross hematuria which cleared spontaneously. Clear cyst fluid, normal or low lactic dehydrogenase, and negative cytology were diagnostic of benign renal cyst. Over-all diag-

nostic accuracy in distinguishing solid tumors from cysts was 94.5 per cent.—*J. M. Stoebner, M.D.*

PHILLIPS, JERRY C. A spectrum of radiologic abnormalities due to tubo-ovarian abscess. *Radiology*, Feb., 1974, *110*, 307–311. (Address: Department of Radiology, University of Tennessee Medical Units, 865 Jefferson Avenue, Memphis, Tenn. 38103.)

The excretory urographic and barium colon examination findings of 45 women with tubo-ovarian abscesses are presented.

The findings on excretory urograms included: pelvic soft tissue mass; ureteral dilatation due to pelvic mass; and medial displacement of the pelvic ureter. The latter has not been described with other causes of intraabdominal masses.

Barium colon examination findings included constricted areas in sigmoid which were more often longer than 5 cm. and often associated with serrated or spiculated margins. Extraluminal gas collections and fistula into an abscess were less frequently noted diagnostic signs.

A list of specific differential diagnostic possibilities is presented and their differentiating points are discussed. These include: periappendiceal abscess; postoperative pelvic abscess; diverticulitis; benign ovarian neoplasms; malignant ovarian neoplasms, uterine tumors; massive bladder distention; perforating carcinoma of the colon; metastases to sigmoid colon; endometriosis; and carcinoid or scirrhous carcinoma of the sigmoid colon.

The author believes that cases of unsuspected tubo-ovarian abscess can be diagnosed and differentiated from other causes of abdominal mass by the combined findings of excretory urography and barium colon examination.—*James A. Chapman, M.D.*

NERVOUS SYSTEM

WINTER, ROBERT B., HAVEN, JAMES J., MOE, JOHN H., and LAGAARD, SHELDON M. Diastematomyelia and congenital spine deformities. *J. Bone & Joint Surg.*, Jan., 1974, *56-A*, 27–39. (Address: Robert B. Winter, M.D., Gillette Children's Hospital, 1003 Ivy Avenue East, St. Paul, Minn. 55106.)

Diastematomyelia is a congenital malformation of the vertebral axis with a localized longitudinal separation of the spinal cord with an interposed septum. Usually this condition is associated with many vertebral anomalies including scoliosis. Twenty-seven patients with diastematomyelia are reported and 26 of these patients had a significant congenital spinal deformity. Sixteen of these 26 patients required a spine fusion. The septum may be osseous, cartilaginous or fibrous.

Roentgenographically, a bone septum was visualized in 15 patients. Twenty-six of the 27 patients had widening of the interpedicular distance and 25 patients had spina bifida. The widening between the pedicles was always in the area of the diastematomyelia, while the spina bifida was frequently located in other portions of the spine. In some cases, the spina bifida was extensive and involved several vertebrae, while in some patients it was a small spina bifida. Eight patients had 1 or more hemivertebrae and 15 patients had segmentation failures including fused vertebral bodies. Myelography demonstrated the septa. False positives during myelography occurred in patients with congenital kyphosis in whom the kyphotic bulging of the vertebral body in the mid line of the spinal canal caused a division of the contrast medium column. This could be avoided if the myelograms were obtained in the supine position.

The surgical treatment includes laminectomy and septum excision. Spine fusion was frequently performed for correction of the scoliosis.—*Ronald J. Ross, M.D.*

SKELETAL SYSTEM

TENNER, R., and ENGELBRECHT, E. Das künstliche Hüftgelenk aus röntgenologischer Sicht. (The artificial hip joint from the roentgenologic standpoint.) *Fortschr. a . d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Jan., 1974, *120*, 11–22. (Address: Dr. med. R. Tenner, Diagnostisches Strahleninstitut des Allgemeinen Krankenhauses St. Georg, Lohmuhlenstrasse 5, 2000 Hamburg 1, Germany.)

Based on almost 6,000 cases of artificial hip joints, the roentgenographic findings of normal and abnormal total hip arthroplasty are described. The single anteroposterior projection of the entire prosthesis and the contralateral hip was sufficient in routine cases. If malposition of the prosthesis was suspected or if the finding in the anteroposterior projection was equivocal, additional roentgenograms in lateral projection were obtained.

Infection is the most serious complication of total hip arthroplasty.

In early infection sinus tracts are usually present and sinography is helpful to determine the extent of the infection.

Late infection, about ½ to 1 year after surgery, is difficult to recognize on the roentgenogram, and the diagnosis is primarily based on clinical findings. Roentgenographic findings include an increase in the width of the lucent zone between bone and acrylic cement, and a broad sclerotic zone and/or ill defined osseous architecture of the surrounding bone. Definite signs of infection in the femur consist of irregular cortical thickening, destruction of the cortex and periosteal reaction. Sinus tracts in late infection are seen late, usually secondary to abscess formation.

Prosthetic loosening is usually caused by infection; a so-called "sterile infection" probably does not exist. Depending on the type of prosthesis inserted, periarticular calcifications of different degree are seen in 8 to 16 per cent of the cases. Osseous fracture in the region of the prosthesis is easily recognized. Direct blow almost never results in displacement of the acetabular cup of the prosthesis, but in fracture of the pubic or ischial ramus. Fracture in the region of the femur usually occurs at the end of the intramedullary stem of the prosthesis. Fracture of the prosthesis is rare.—*Peter F. Winter, M.D.*

Brooker, Andrew F., Bowerman, Jack W., Robinson, Robert A., and Riley, Lee H., Jr. Ectopic ossification following total hip replacement: incidence and a method of classification. *J. Bone & Joint Surg.*, Dec., 1973, *55-A*, 1629–1632. (Address: Andrew F. Brooker, M.D., Division of Orthopaedic Surgery, The Johns Hopkins Hospital, 601 North Broadway, Baltimore, Md. 21205.)

One-hundred consecutive patients were treated with total replacement of the hip, utilizing the technique and prosthesis of McKee and Farrar, or the technique of Charnley or Mueller, as performed by 3 members of the full time staff of the Johns Hopkins University School of Medicine. The cases were reviewed a minimum of 6 months following the operative procedure to evaluate the formation of ectopic bone about the hip. Roentgenograms of the hip were made preoperatively and a minimum of 6 months following surgery and the degree of ectopic bone formation graded according to 1 of 4 classifications as to the degree of soft tissue calcification, bony spurring reducing the space between the opposing bone surfaces, or development of apparent bony ankylosis of the hip. Correlation of the preoperative and postoperative functional status of the hip by means of the Harris hip rating scale was made.

The study revealed that ectopic bone formation without apparent bony ankylosis occurred in 19 of the 21 patients and no significant alteration in the result achieved in total arthroplasty seemed to occur.

Fourteen of the 100 patients had had previous hip surgery and 7 already had ectopic bone formation. Additionally, 14 of the 86 patients with no prior hip surgery had ectopic calcification. Postoperative evaluation of these patients revealed that the patients with previous surgical procedures had a much higher incidence of development of ectopic ossification. Review of the type of previous surgery indicated that the patients with femoral head prosthesis were more likely to develop ectopic ossification, although this did not necessarily result in lesser function.—*John T. Underberg, M.D.*

Smith, Ronald W., and Smith, Chadwick F. Solitary unicameral bone cyst of the calcaneus: a review of twenty cases. *J. Bone & Joint Surg.*, Jan., 1974, *56-A*, 49–56. (Address: Ronald W. Smith, M.D., 6180 Caminito Regla, San Diego, Calif. 92122.)

The solitary unicameral bone cyst does not occur very frequently in the calcaneus. Twenty patients with this lesion of the calcaneus were studied. The ages of these patients ranged from 5 to 38 years and there were 15 males and 5 females.

It is believed that the calcaneal solitary cysts develop from a congenital tissue remnant in the region of the primary ossification center. This tissue remnant grows and becomes cystic. There have been no pathologic fractures in the series reported and in the authors' review of previous cases of the unicameral bone cyst of the calcaneus.

Fourteen of the cysts were discovered coincidentally on roentgenograms ordered for a foot or ankle injury. Six patients had a spontaneous onset of heel pain without any history of injury.

In the lateral view of the calcaneus, the unicameral bone cyst was always present at the base of the calcaneal neck inferior to the anterior portion of the posterior facet. In the tangential view, the cyst was always located against the lateral wall of the bone and extended $\frac{1}{2}$ to $\frac{2}{3}$ the width of the calcaneus. In most of the cysts, the anterior margin was straight and vertical and the posterior margin was curvilinear. The margins were usually sclerotic and in only 2 patients was the cortex expanded.

Eleven patients had the lesions curetted and bone grafted, and 9 patients had no surgical intervention. In 6 of these 9 patients, there were no roentgenographic signs of progression and spontaneous regression occurred in 2 patients.

The normal calcaneus has a variable number of trabeculae in the neck of the calcaneus and 7 per cent of normal calcanei have a prominent lucency in this area. Twenty-two per cent have a moderate lucency. These are called pseudocysts, since they are not completely void of bone centrally, are less well demarcated and are more triangular in shape.

Unless the patients have persistent pain, nonoperative treatment is recommended for the solitary cyst of the calcaneus which has the typical roentgenographic appearance of a unicameral cyst.—*Ronald J. Ross, M.D.*

Zagnoli, Paolo, and Romani, Francesco. La sindrome del legamento arcuato. (The syndrome of the arcuate ligament.) *Radiol. med.*, Aug., 1973, *59*, 702–709. (Address: Paolo Zagnoli, Istituto di Radiologia dell'Università, 3 via del Pozzo, I-41100 Modena, Italy.)

In 1963, Harjola and Lathiharyn, and in 1965, Dunbar *et al.* described a particular celiac axis syndrome and abdominal angina produced by the compression of the celiac trunk by the arciform fibers of the aortic hiatus of the diaphragm.

The compression of the celiac trunk by the arcuate ligament can occur: (1) because the artery has a much higher origin from the aorta, in the inferior segment of the mediastinum. The trunk penetrating into the abdominal cavity passes under the inferior margin of the arcuate ligament which presses it against the aorta; and (2) in the case in which the arcuate ligament is reaching farther down than normally and compresses therefore the celiac trunk, which is in its normal position.

The diminished blood flow produces a symptomatology characterized by crampy epigastric or paraumbilical pain, during or immediately after a meal, weight loss and a systolic murmur in the epigastrium. The weight loss is due to the spontaneous limitation in the amount of food ingested by the patient who tries to eliminate this way the appearance of the symptoms.

In the past 3 years the authors have observed 6 cases presenting the syndrome of the arcuate ligament. In all cases the diagnosis was confirmed by aortography, by the Seldinger's technique. The most common angiographic findings were: (1) verticalization of the first 2–3 cm. of the celiac trunk which was flattened against the wall of the aorta; (2) slight indentation in the anterior aspect of the celiac trunk ("coupe d'ache" of the French authors), produced by the inferior border of the arcuate ligament; (3) after running normally, 2–3 cm. from its origin, the artery changed to a horizontal course or turned upward with frequent post-stenotic dilatations; and (4) presence of arteriosclerotic plaques in the abdominal aorta.

All cases were successfully operated.

Numerous and clear diagrams and illustrations enrich the article.—*A. F. Govoni, M.D.*

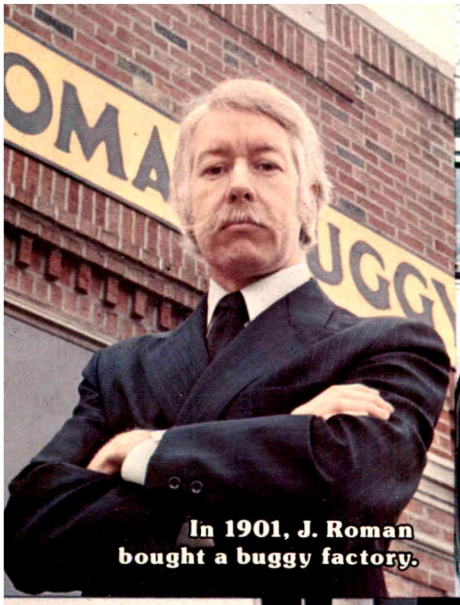
Gohel, Vijay K., Dalinka, Murray K., and Edeiken, Jack. The serpiginous tract: a sign of subacute osteomyelitis. *J. Canad. A. Radiologists*, Dec., 1973, *24*, 337–339. (Address: Vijay K. Gohel, M.D., Thomas Jefferson University Hospital, Department of Radiology, 11th and Walnut Streets, Philadelphia, Pa. 19107.)

The nutrient artery is the main route of infection in hematogenous osteomyelitis.

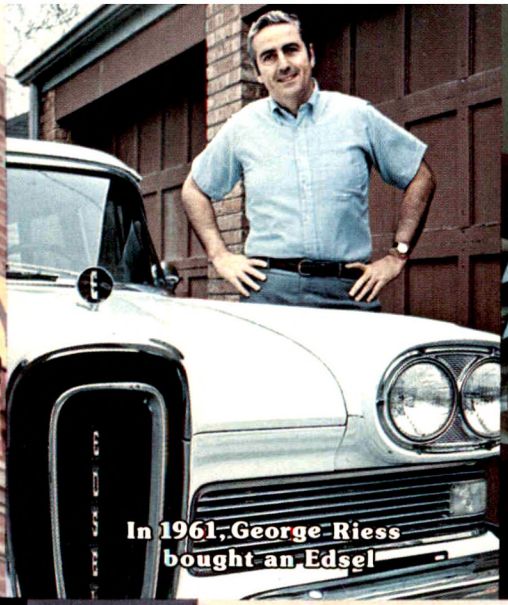
In children, once the organisms have entered bone, they lodge in the terminal capillary loops at the metaphyseal bone ends adjacent to the epiphyseal plate, which acts as a vascular barrier. The blood flow in numerous capillary loops and venous sinusoids is relatively stagnant in the metaphysis. One or several small metaphyseal abscesses may, therefore, coalesce to form a large abscess cavity. Increased pressure in the area of inflammation drives the infection into several channels of lesser tension. Since it is difficult to cross the epiphyseal plate, the most common route is the venous sinusoids. The infection may break through the cortex or be controlled with antibiotics. In the latter case a localized abscess cavity will result.

The tract along which the abscess extends to the surface in an attempt to break through the cortex appears roentgenographically as a sharply demarcated tapering lucency continuous with the abscess cavity. This characteristic appearance of the abscess cavity is particularly observed in children between 1 to 15 years. Most of the cases fall in the category of subacute or early chronic osteomyelitis. All had received antibiotic therapy.

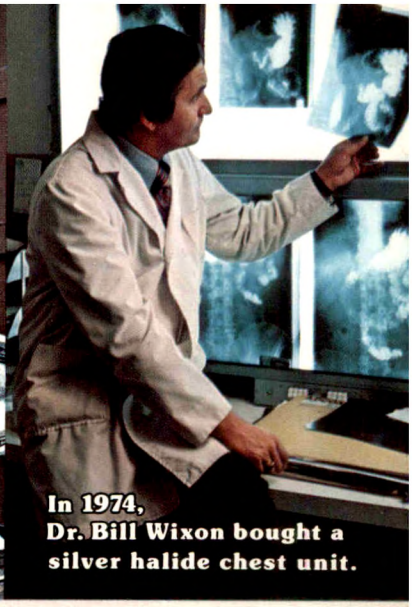
The cavity was usually eccentrically located in the metaphysis abutting on the epiphyseal plate. A serpiginous or tapering radiolucent tract extends from the main cavity toward the diaphysis. The cavity has a well defined sclerotic margin. This appearance is typical and aids in distinguishing it from similarly benign cystic lesions, such as nonossifying fibroma, chondromixoid fibroma, solitary bone cyst and chondroblastoma.—*David Morse, M.D.*



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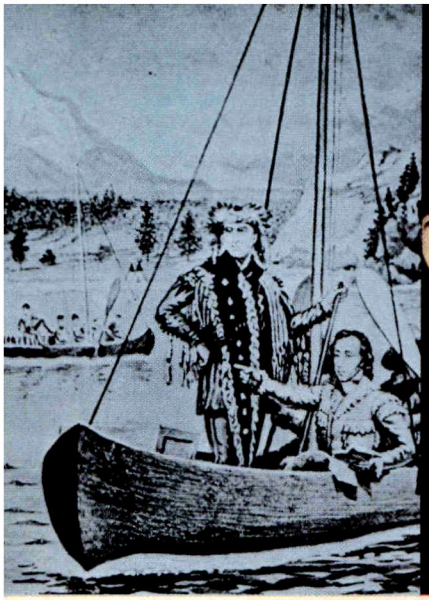
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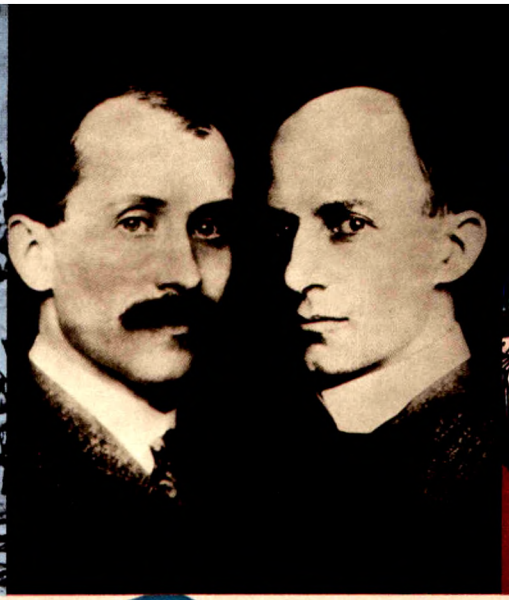
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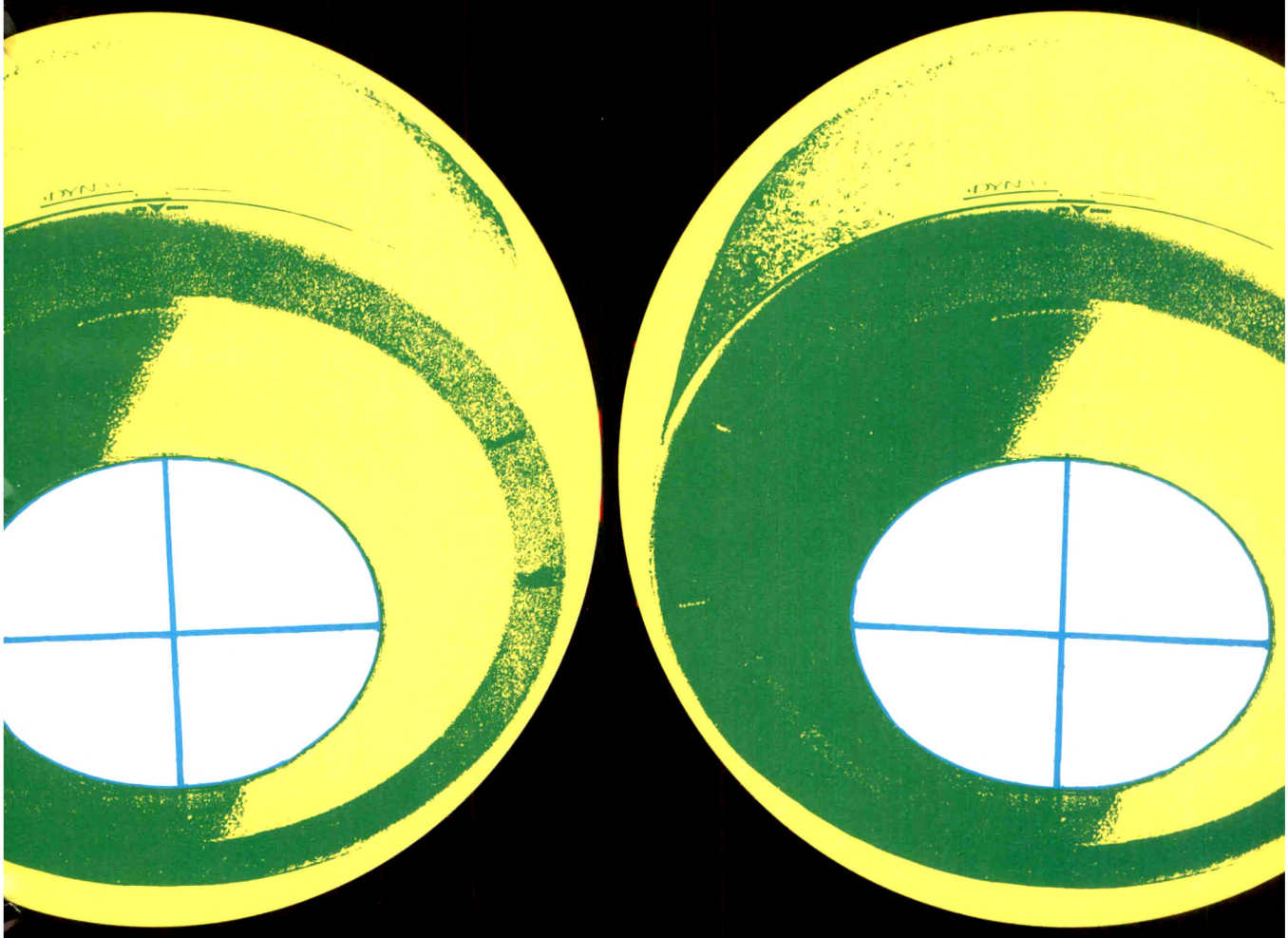
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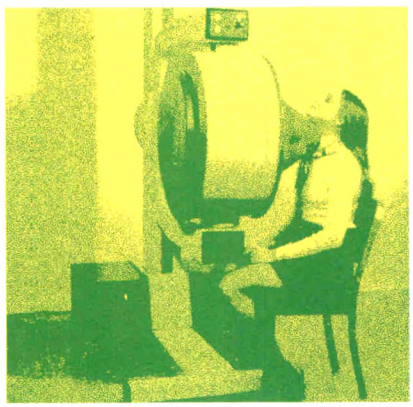
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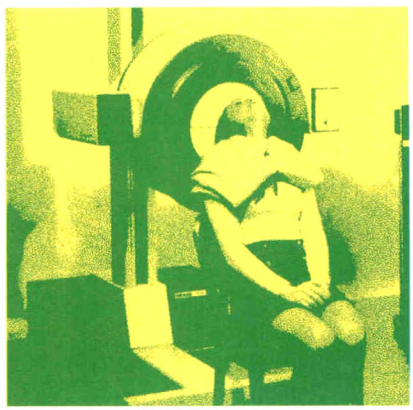
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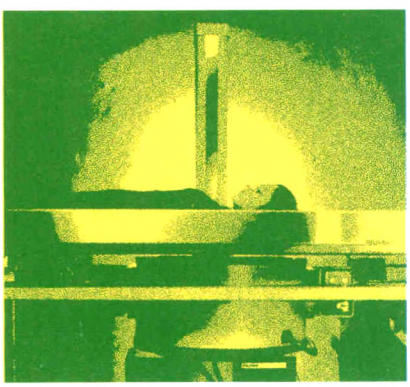


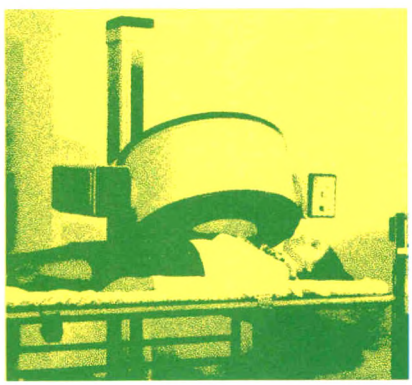


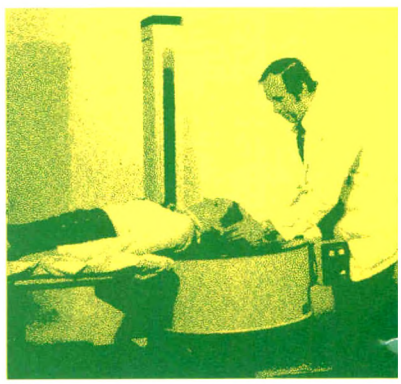
Dyna Camera 4

☐ High-resolution images, a result of advanced detector techniques producing a clear, sharp diagnostic gamma-image presentation.
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Cancer Medicine—Holland & Frei

Unquestionably the most critical and comprehensive book on cancer published in any language in the twentieth century, this work contains a vast amount of information on all aspects of oncology—etiology, pathogenesis, epidemiology, immunology, virology, diagnosis and treatment. Designed for use by everyone who is in any way involved with the disease, it is sure to become *the* standard reference. *Edited and with contributions by* JAMES F. HOLLAND, M.D., *Mt. Sinai School of Medicine of the City University of New York, New York City; and* EMIL FREI, III, M.D., *Harvard Medical School, Boston, Massachusetts. (158 Contributors). 2018 pp. (7 × 10), 589 figs., numerous charts and tables, 1973, $90.00.*

Cardiac Catheterization and Angiography. Edited by WILLIAM GROSSMAN, M.D., The University of North Carolina, Chapel Hill. A comprehensive, yet concise and up-to-date survey of the subject, including chapters on percutaneous technique and angiography. *About 450 pp., illus., Fall, 1974.*

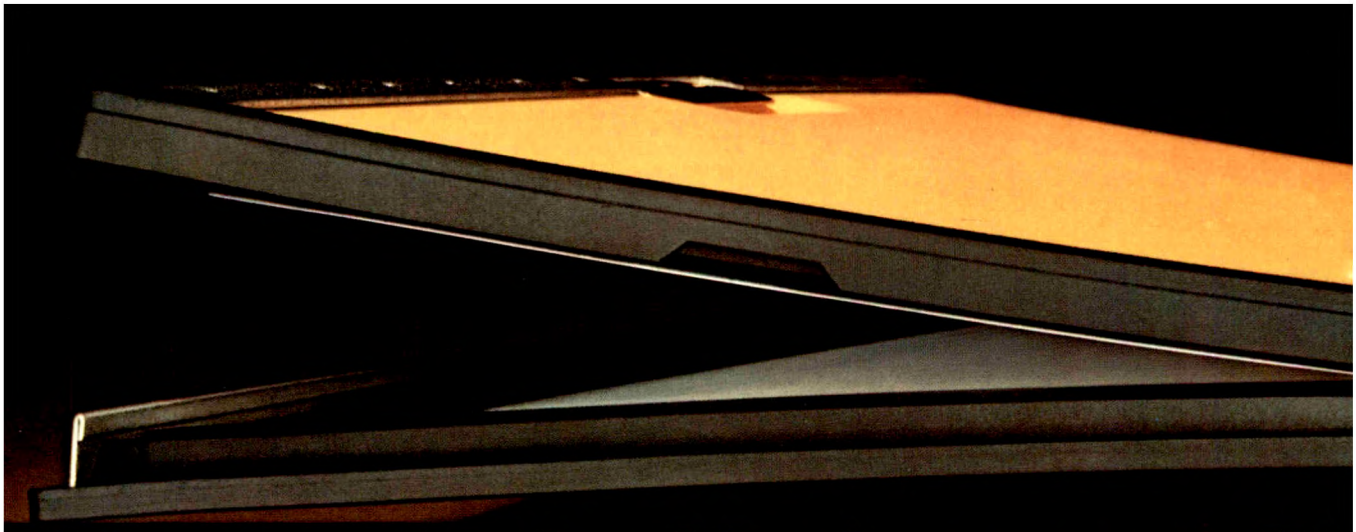
Echocardiography. By HARVEY FEIGENBAUM, M.D., Indiana University School of Medicine, Indianapolis. With the assistance of SONIA CHANG, B.S. Describes the current state of echocardiography . . . instrumentation, theory, physical properties of ultrasound, and engineering are discussed. *239 pp. (7 × 10), 193 illus., 1972, $11.00.*

An Introduction to the Physics of Diagnostic Radiology. By EDWARD E. CHRISTENSEN, M.D., THOMAS S. CURRY, III, M.D., and JAMES E. NUNNALLY, M.A., all of the University of Texas Southwestern Medical School, Dallas. Contains essential aspects of physics and information on production and recording of data from radiographic examinations. *252 pp. (7 × 10), 208 illus., 1972, $14.50.*

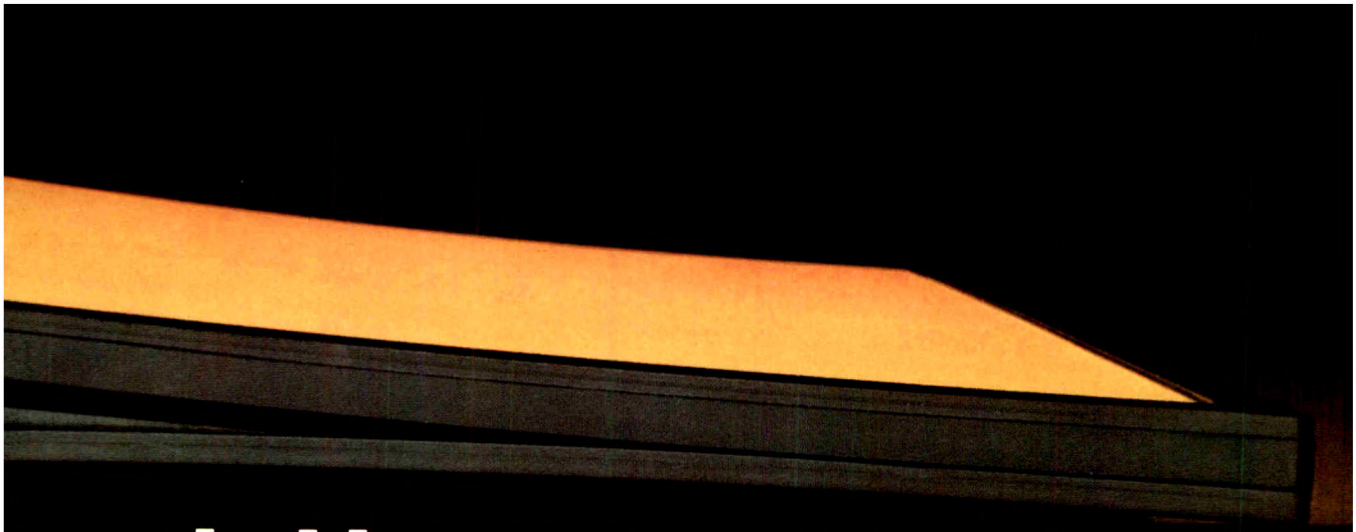
Medical Radiation Biology. By DONALD J. PIZZARELLO, Ph.D., New York University School of Medicine, New York; and RICHARD L. WITCOFSKI, Ph.D., The Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina. Emphasizes clinical aspects with applications to diagnosis, nuclear medicine and radiation therapy. *111 pp. (7 × 10), illus., 1972, $9.00.*

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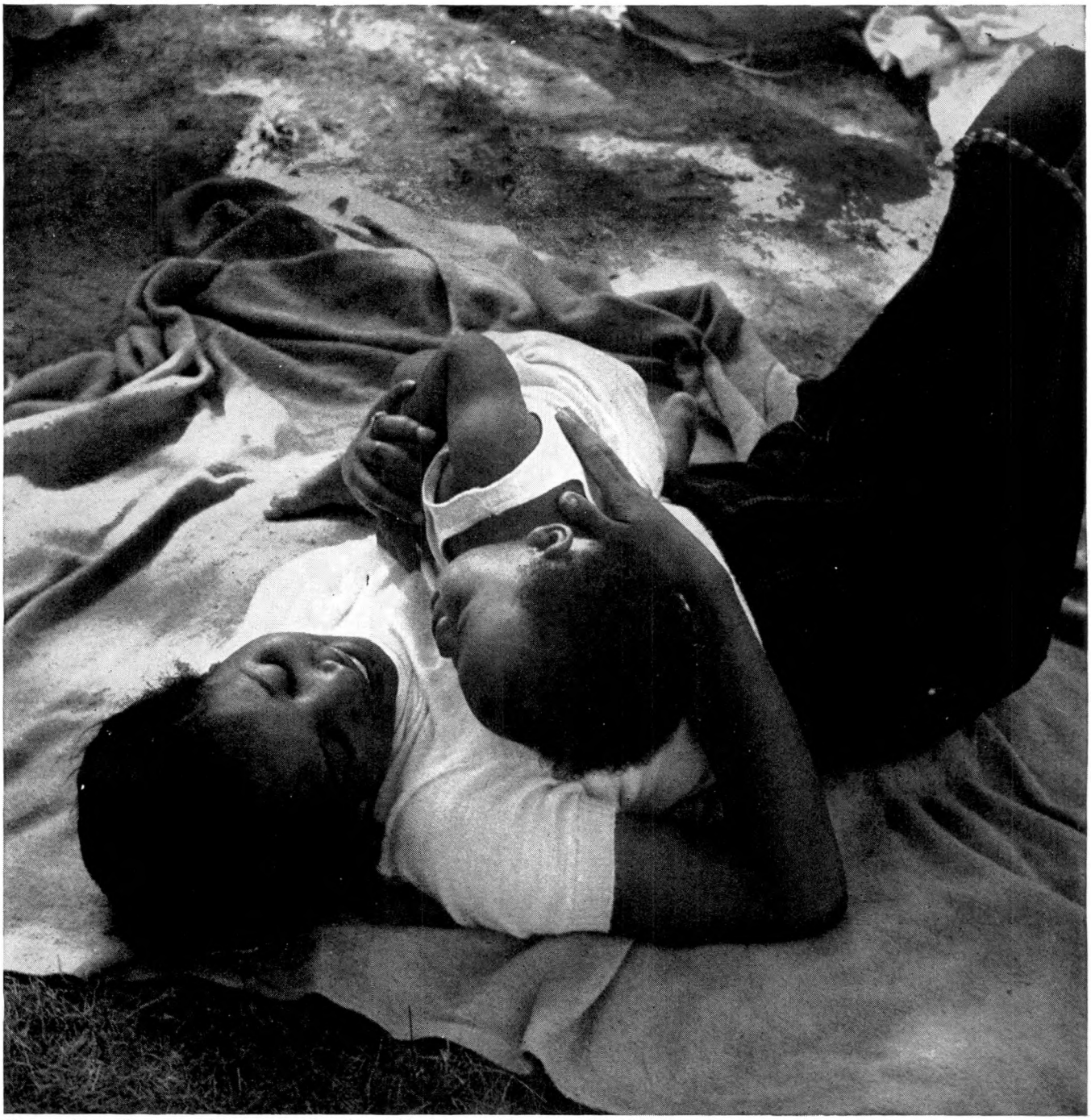
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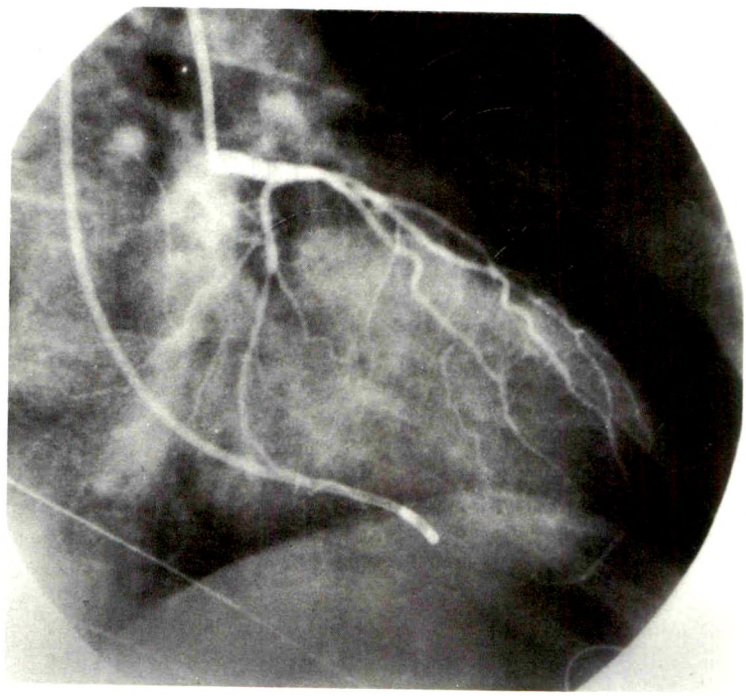
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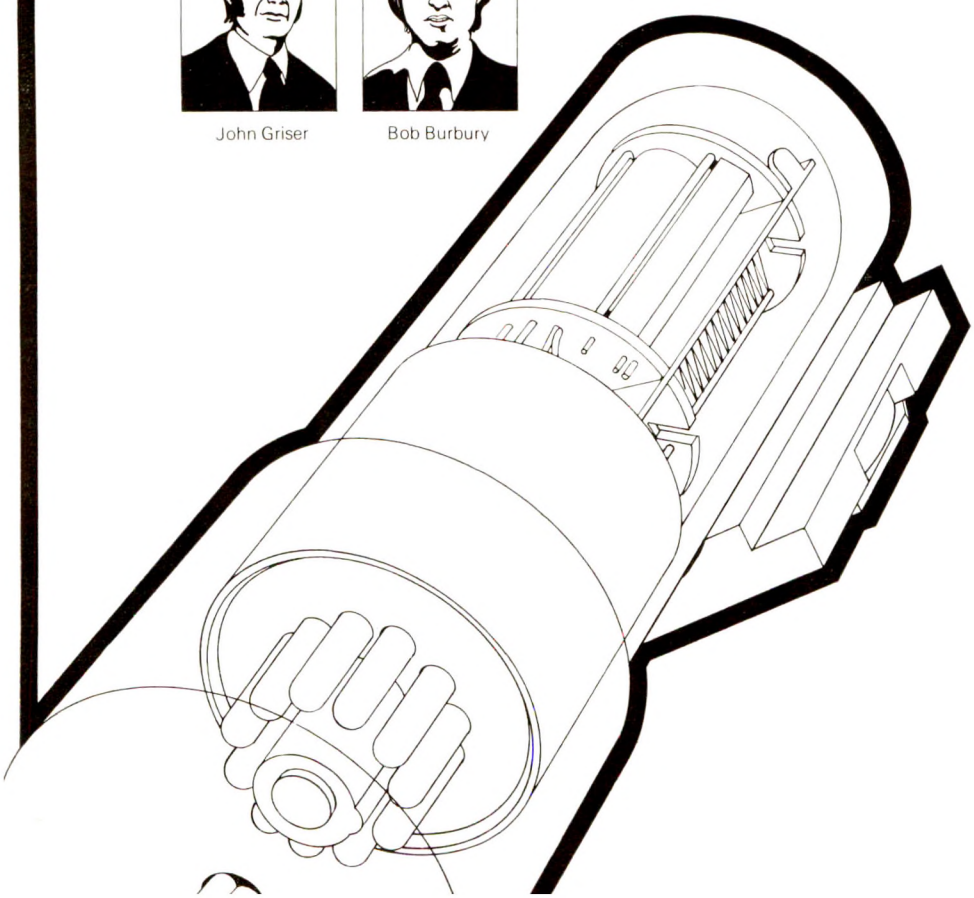
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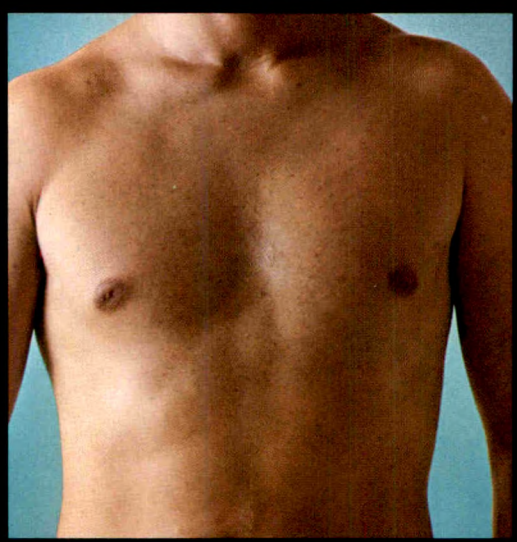
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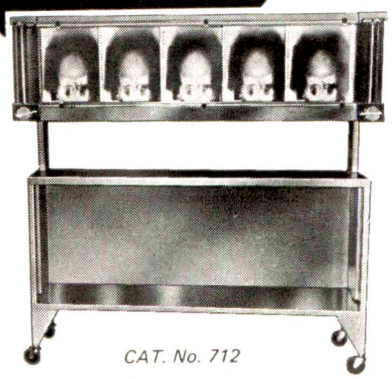
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COMPLICATIONS OF DIAGNOSTIC RADIOLOGY *by* **John F. Weigen,** *Palo Alto Medical Clinic, Palo Alto, California, and* **Sydney F. Thomas,** *Chico, California.* Diagnostic radiology, often essential if the primary physician is to treat his patient's disease logically, may, unfortunately, be associated with significant complications. This work is designed to aid all physicians who use diagnostic radiology so that they may balance the risk of a specific examination against the need for the study. *'73, 576 pp., 81 il., 53 tables, $25.75*

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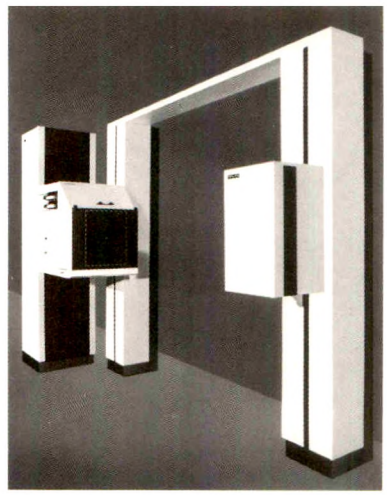
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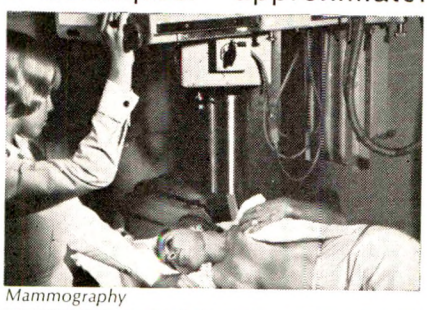
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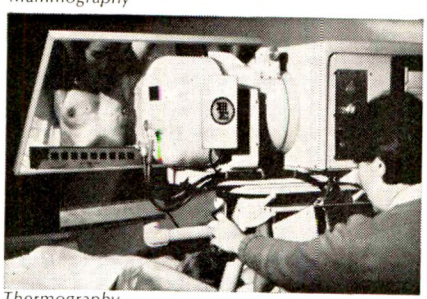
Breast Cancer: earlier warning system

Futility and frustration beset the physician confronted with breast cancer. For the last 35 years, the survival rate has not significantly changed despite intensive educational programs aimed at earlier detection, and improvement in treatment techniques.

What is the outlook? We know the key to reducing mortality from breast cancer is in the *earliest possible* diagnosis. The stage at which breast cancer is detected is *crucial* to the outcome of treatment. By the time a lump is discovered through BSE or clinical examination, critical time may have been lost.



Mammography



Thermography

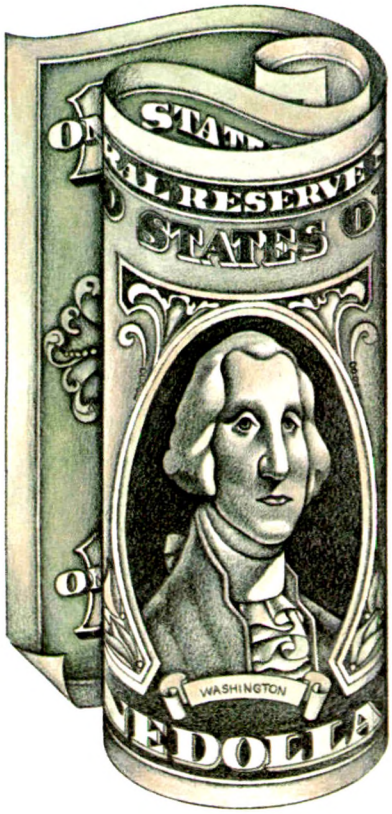
And we *do* have the means to achieve earlier diagnosis. We do have an *earlier warning system.* Mammography and thermography can detect breast cancer *before* a lump is discernible by palpation. To demonstrate that it is practical and feasible to detect breast cancer earlier by using these modalities, the American Cancer Society and the National Cancer Institute are funding a network of breast cancer® demonstration projects. Supported by grants of $2-million from the ACS and $4-million from the NCI, 20 such centers are expected to be operative across the country by the end of the year. Each will screen at no charge, approximately 5,000 women annually, in what is considered to be the ideal detection program — to include clinical examination, mammography and thermography. Each of these detection methods contributes independently to the detection of breast cancer, and none can be dispensed with in the search for early disease.

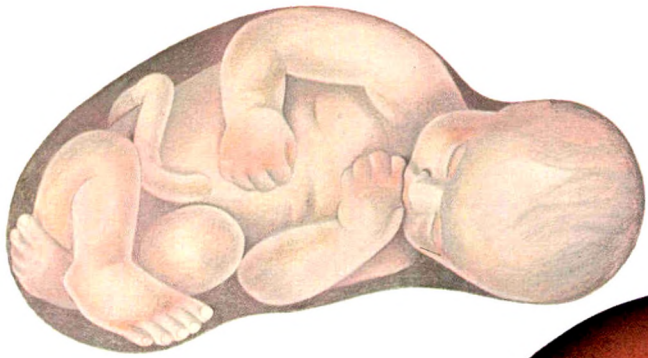
At present we cannot prevent breast cancer, but the potential for saving more lives is immense. The five-year survival rate changes dramatically from 53% when axillary nodes are positive, to 85% when the disease is localized, to nearly 100% for in-situ cancer.

We have an earlier warning system. Let's use it.

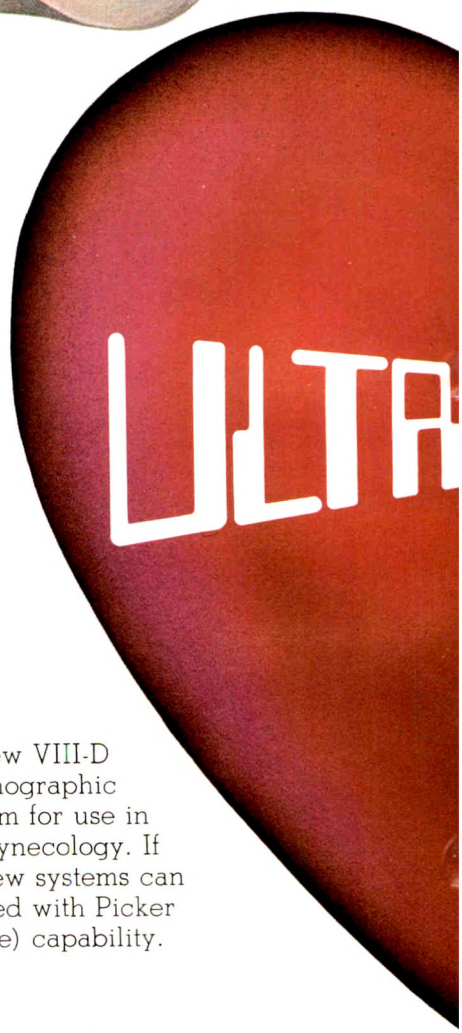
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An Economic lly





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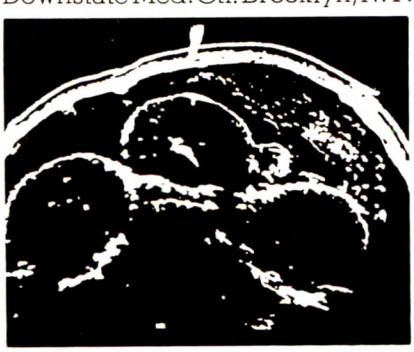


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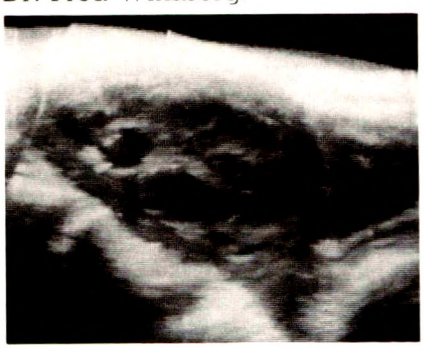


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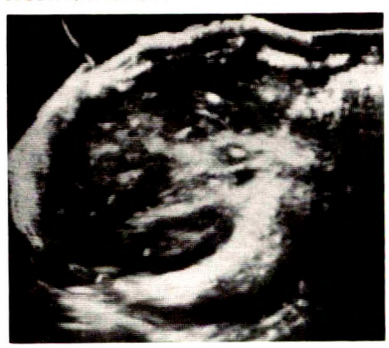
Transverse scan of triplet fetal head in third trimester. Courtesy Downstate Med. Ctr. Brooklyn, N.Y.



Picker EDC scan of 24-week missed abortion. Courtesy Dr. Fred Winsberg.



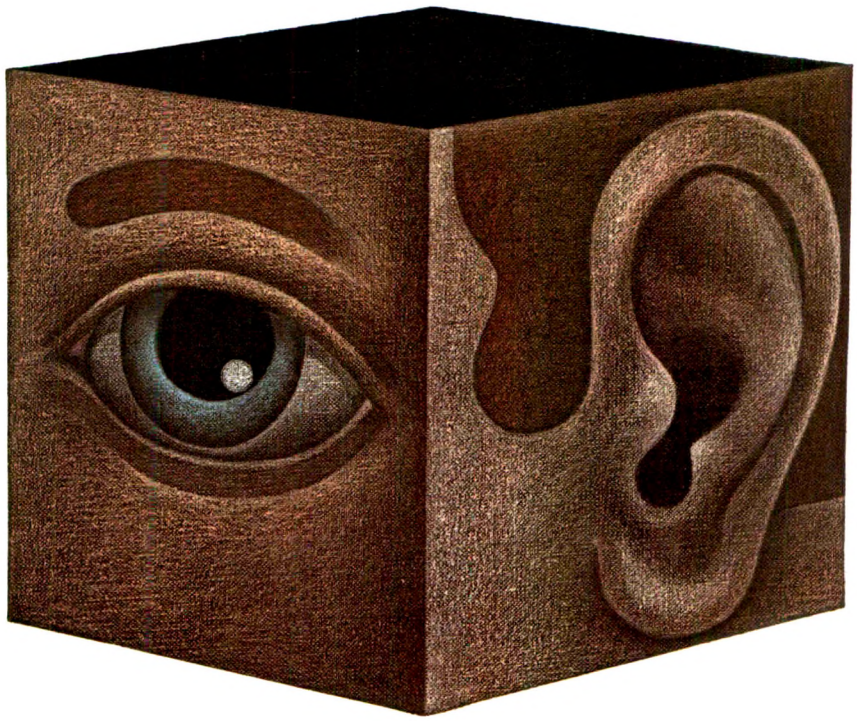
EDC transverse scan of normal liver.



Another reason to buy from...

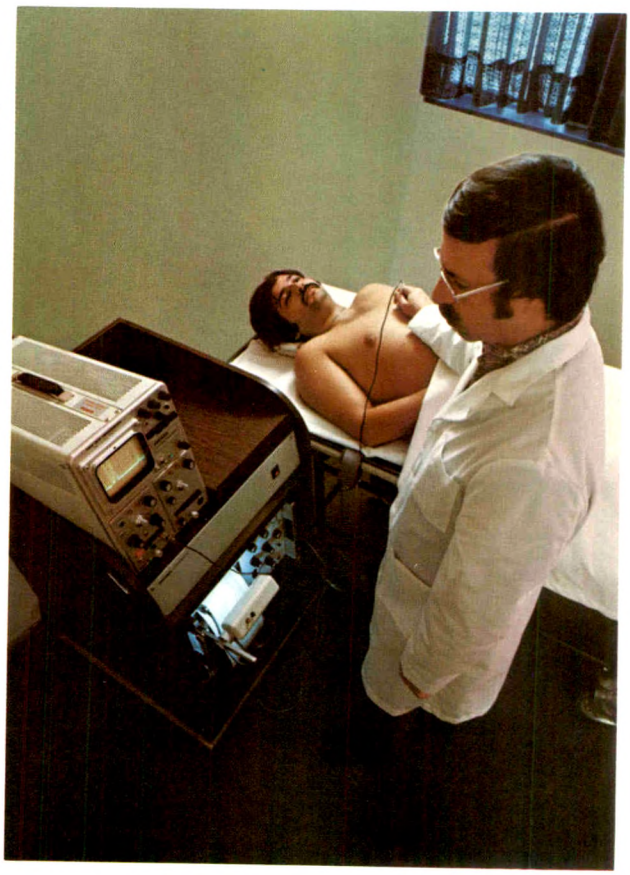
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In ultrasonography, intermittent ultrasonic signals are sent into the body, and their echoes converted into signals that are displayed as distinctive visual patterns on the screen of a cathode ray tube.

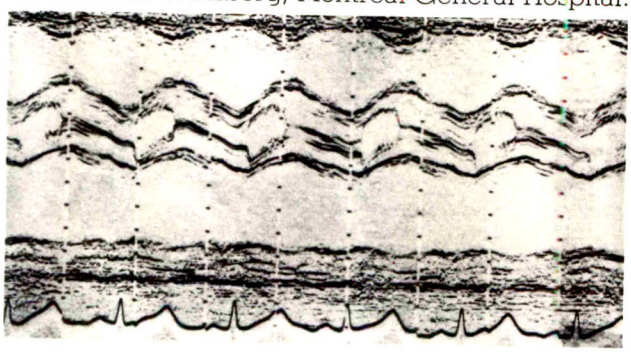




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M-mode dynamic echocardiogram shows fluttering of aortic valve cusps. Courtesy Dr. Fred Winsberg, Montreal General Hospital.



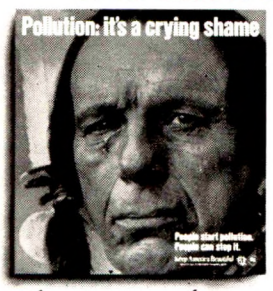
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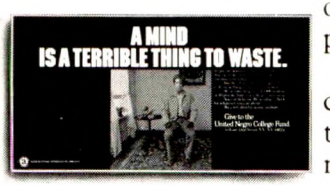




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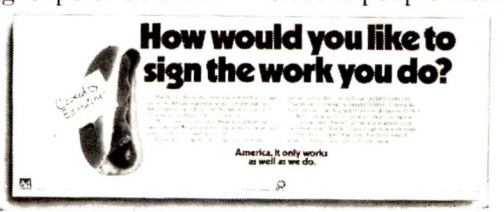
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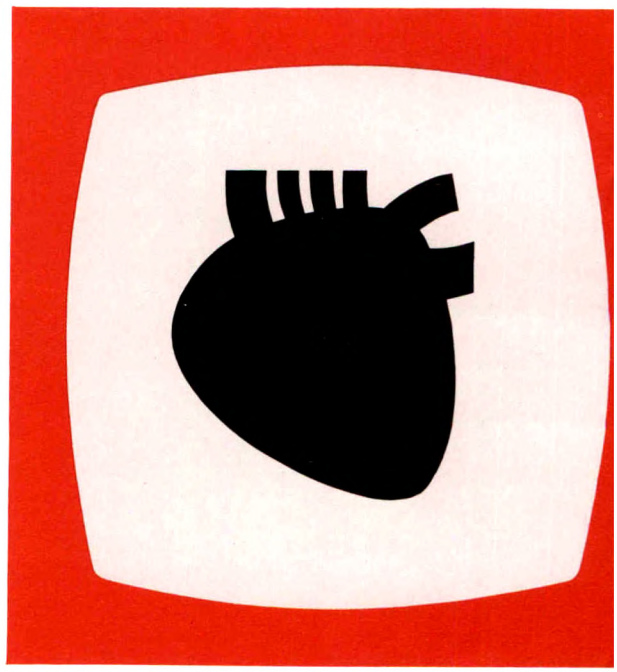
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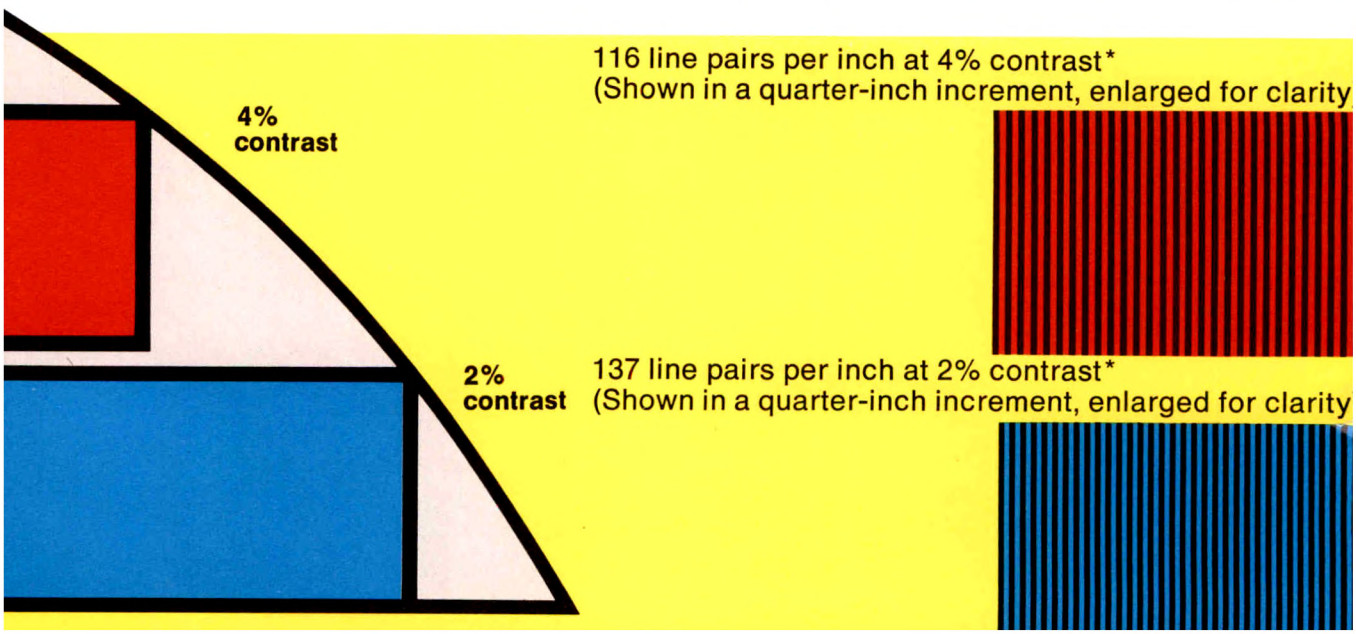
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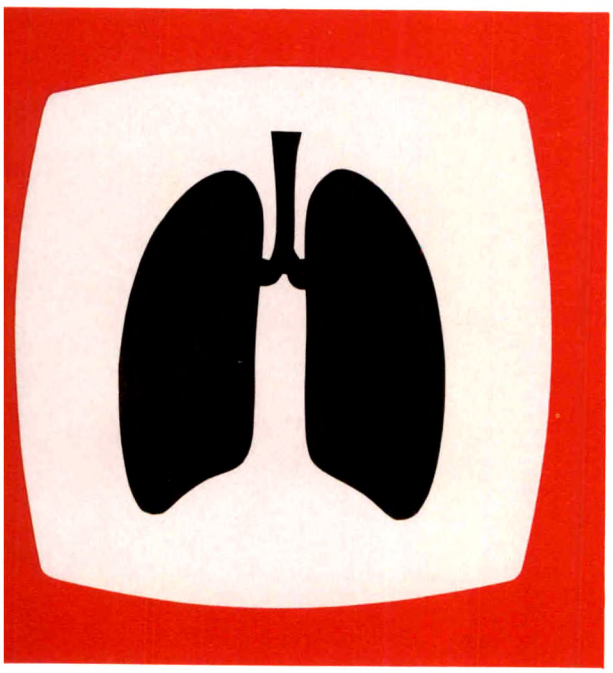
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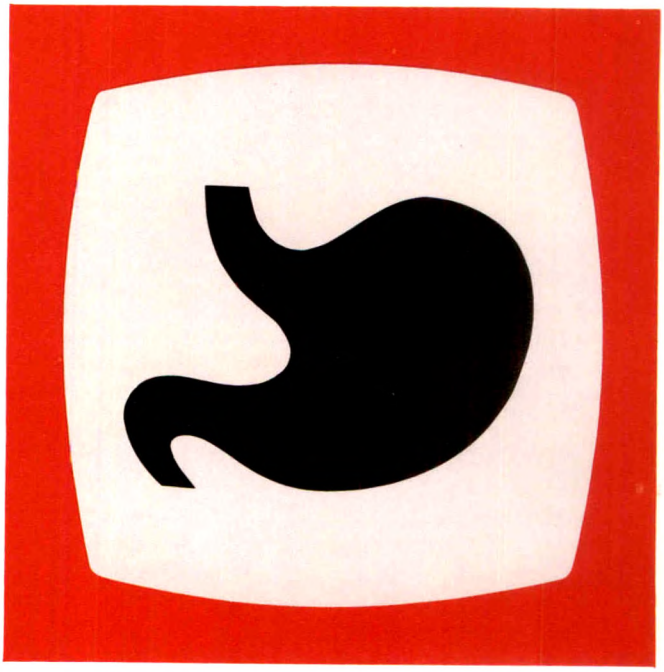
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INDEX TO ADVERTISERS

Amersham/Searle xxx

Doctor's Camera xxxvi

Eastman Kodak ..... xxviii, xxix

Elema-Schonander xvii

E-Z Em i

Flow Pharmaceuticals ..... Inside Front Cover

General Electric ..... xxxii, xxxiii

Ilford X-Ray ..... vi, vii

Lea & Febiger xxvii

Low X-Ray xx

Machlett Laboratories xi

Ortho Pharmaceuticals ..... Inside Back Cover

Philips Medical Systems (U.S.) ..... xxxviii, xxxix

Philips Medical Systems (England) ..... viii, ix

Picker Corporation ..... xxiii, xxiv, xxv, xxvi, xxxv, xlii, xliii

Procter & Gamble ..... xiv, xv, xvi

Profexray ..... xxxiv, Back Cover

Purdue-Frederick v

Radx xl

Siemens Corporation ..... xlvi, xlvii

S & S X-Ray xxxvi

3-M Company ..... xii, xiii

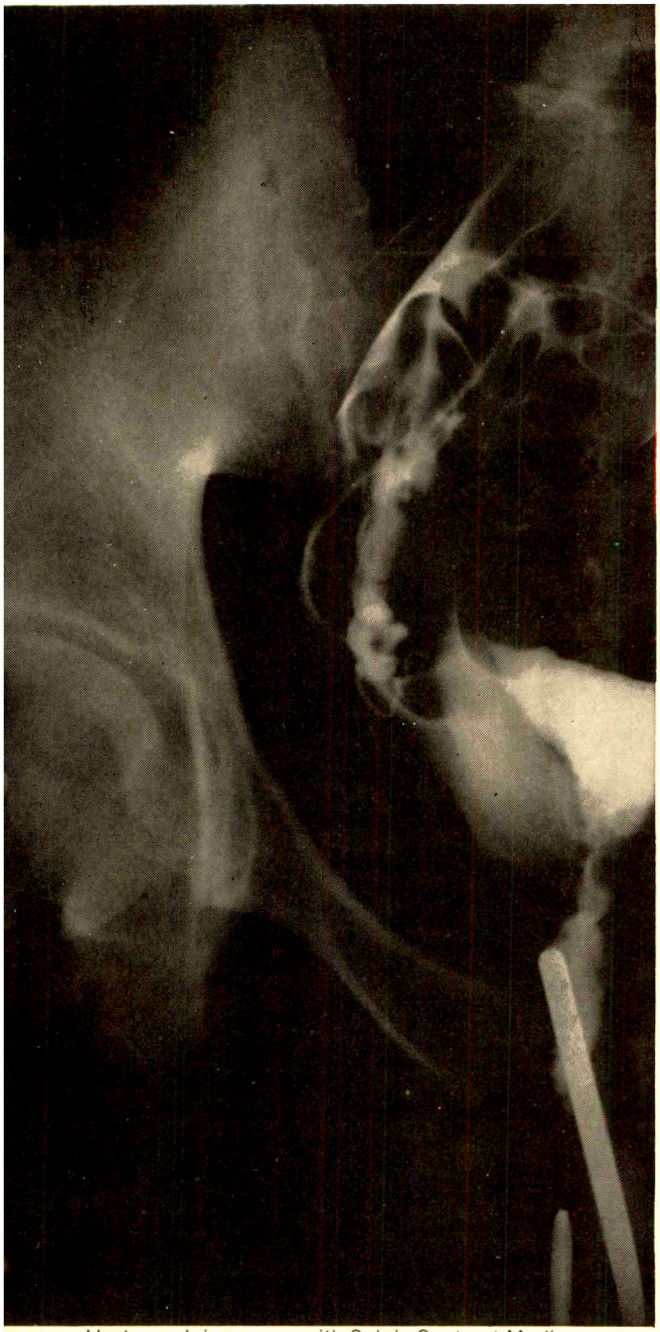
Thomas, Charles C, Publisher ..... xvi, xxxvii

Toshiba International Corporation x

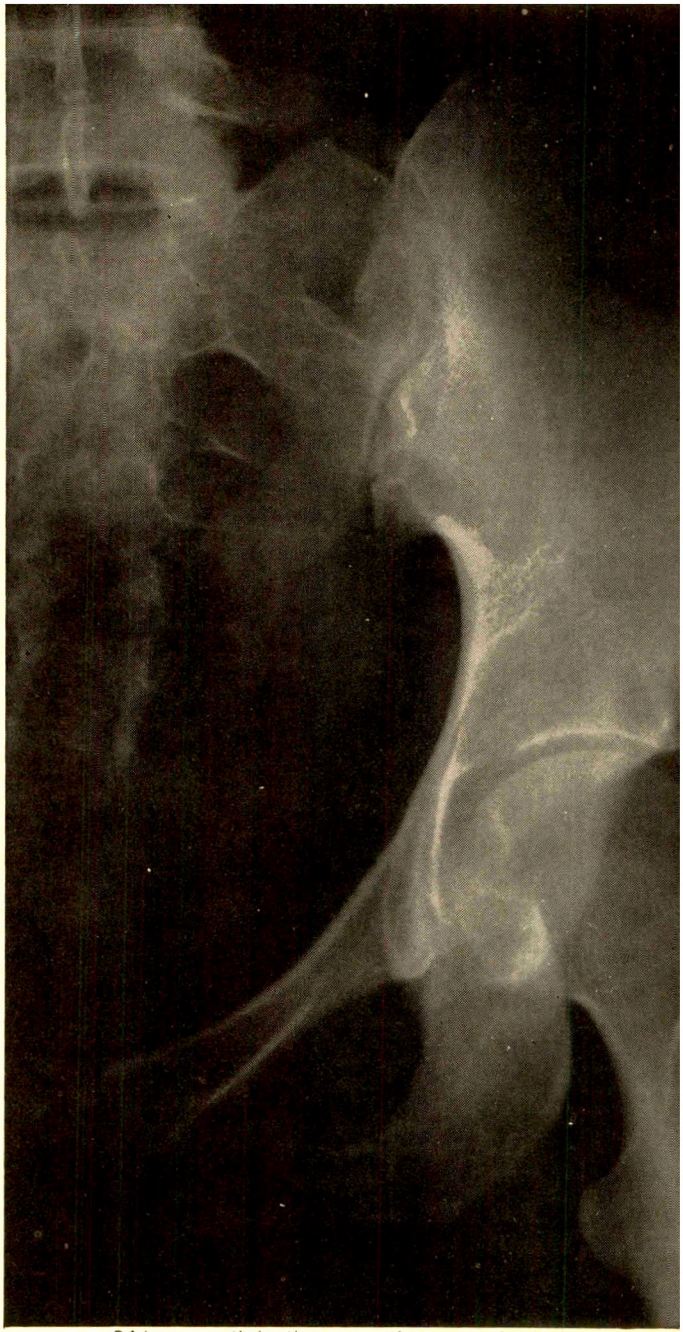
Varian Radiation Division ..... xviii, xix

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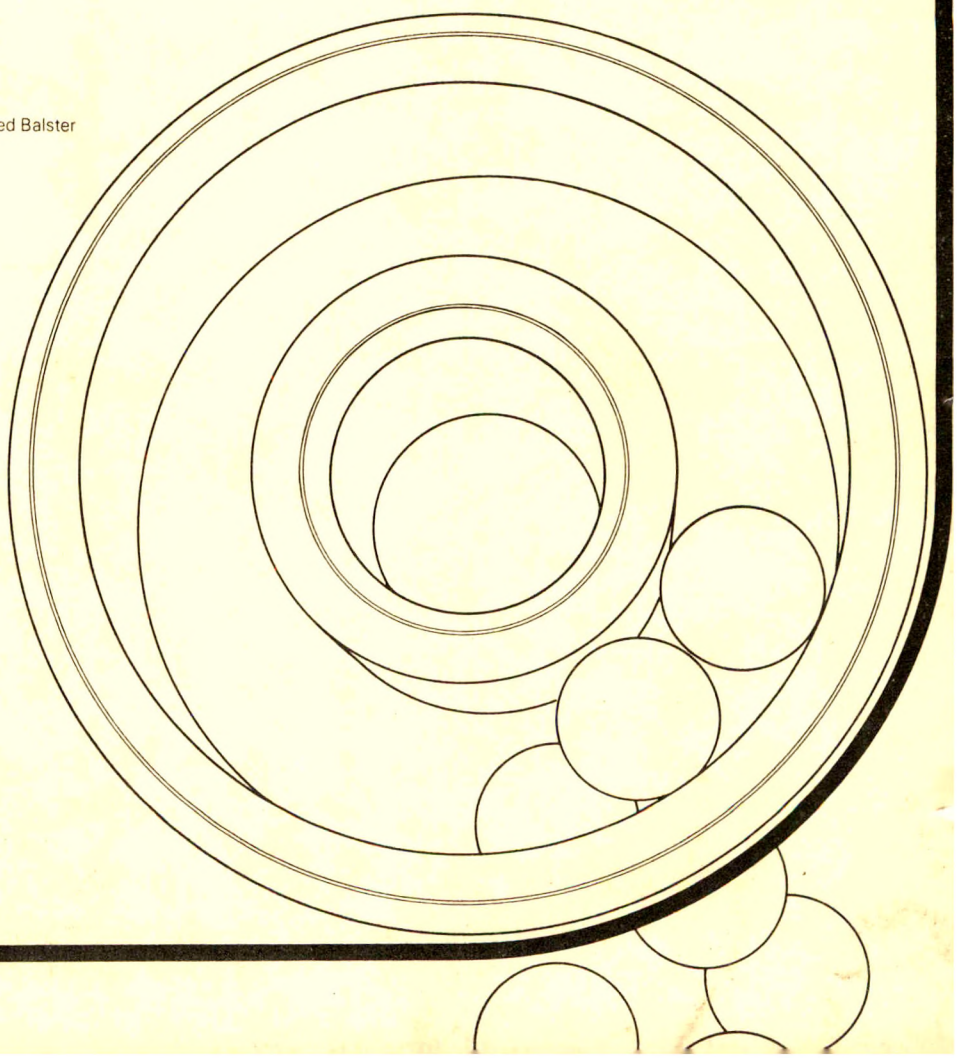
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INDEX NUMBER

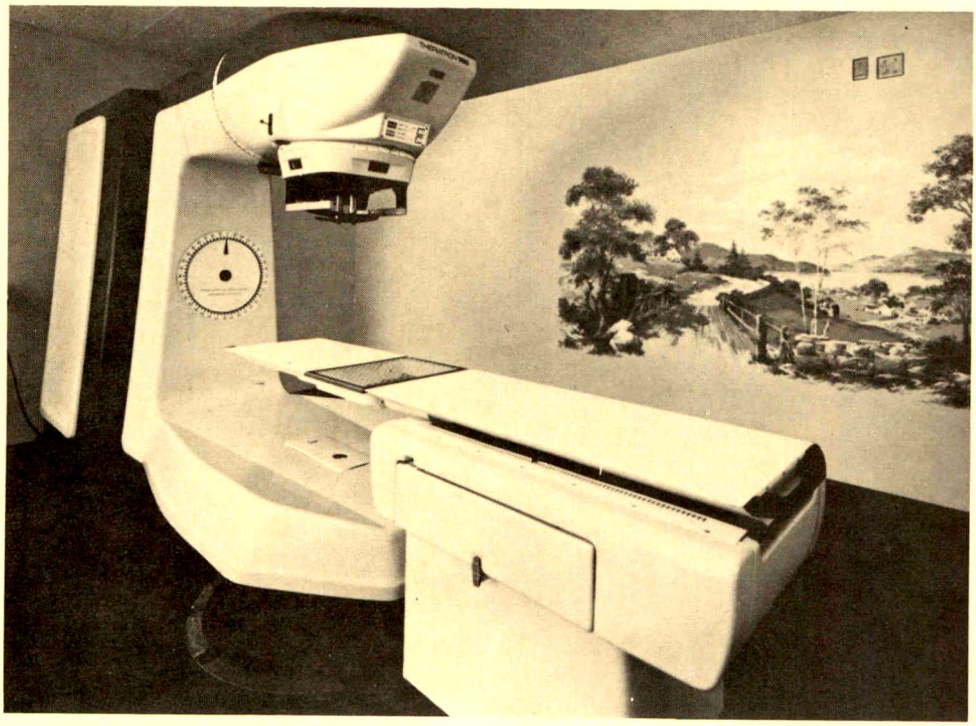
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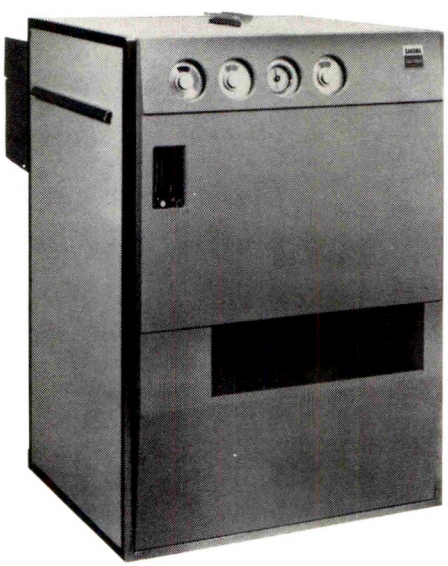
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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 121 AUGUST, 1974 No. 4

TABLE OF CONTENTS

Quantitative Thyroid Scanning: A Reliable Thyroid Function Test. L. BARRY ARKLES, M.R.A.C.P. 705

An Unusual Concentration of Radiopertechnetate in a Patient with Thyroid Cancer. JACQUES LAMOUREUX, M.D., PH.D. 714

Diagnosis of Barrett's Esophagus with Radioisotopes. FELIPE GORDON, M.D., JOSÉ RAMÍREZ-DEGOLLADO, M.D., RODRIGO MUÑOZ, M.D., ALFREDO CUAREN, M.D., AND LUIS LANDA, M.D. 716

Clinical Evaluation of Preoperative Liver Scintigraphy in Gastric Carcinoma Patients. KATSUSHI WATANABE, M.D., KENJIRO KAWAHIRA, M.D., AND KEIICHI MATSUURA, M.D. 720

The Use of Multiple Radionuclide Imaging to Differentiate the Focal Intrahepatic Lesion. LAWRENCE R. MUROFF, M.D., AND PHILIP M. JOHNSON, M.D. 728

The Value of Tc^{99m}-Diphosphonate (HEDSPA) as a Skeletal Scanning Agent. J. PAPADIMITRIOU, M.D., M. VEZERIDIS, M.D., M. CONSTANTINIDIS, PH.D., E. CHIOTELIS, PH.D., C. CONSTANDINIDIS, M.D., AND C. TOUNTAS, M.D. 735

Pitfalls in Tc^{99m} Polyphosphate Skeletal Imaging. JAMES H. THRALL, M.D., NASSAR GHAED, M.D., GEORGE E. GESLIEN, M.D., STEVEN M. PINSKY, M.D., AND MERRILL C. JOHNSON, M.D. 739

Influence of Fractionation on Acute and Late Reactions in Vocal Cord Carcinoma. R. KENNETH LOEFFLER, M.D. 748

The Results of Split-Course Radiation Therapy in Cancer of the Lung. HUGH SCRUGGS, M.D., ANAS EL-MAHDI, M.D., SC.D., RICHARD D. MARKS, JR., M.D., AND WILLIAM C. CONSTABLE, M.B., CH.B., D.M.R.T. . . . 754

Dual Camera Studies of Pulmonary Function with Computer Processing of Data. MERLE K. LOKEN, M.D., PH.D., RICHARD A. PONTO, B.S., RICHARD S. KRONENBERG, M.D., LAWRENCE E. WILLIAMS, PH.D., AND MARVIN E. GOLDBERG, M.D. 761

Extended Mantle Radiotherapy in Hodgkin's Disease and Malignant Lymphoma. J. E. MARKS, M.D., E. M. MORAN, M.D., M. L. GRIEM, M.D., AND J. E. ULTMANN, M.D. 772

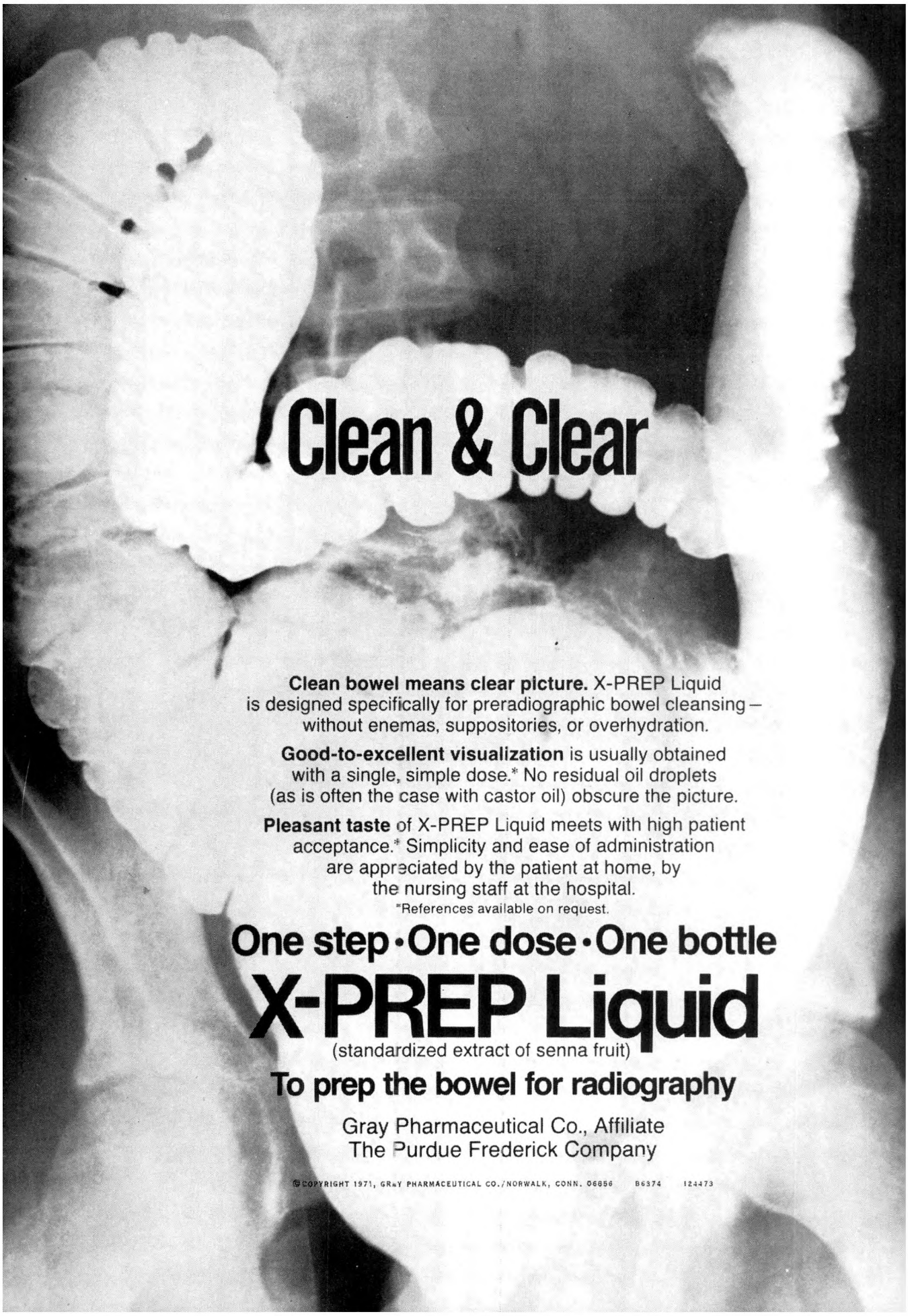
Angiography of the Liver and Spleen in Hodgkin's Disease. K. JONSSON, M.D., AND A. LUNDERQUIST, M.D. 789

Kaposi's Sarcoma: Experience with Ten Cases. STEVEN G. MANN, M.D. . 793

(*Continued on page iv*)

LIBRARY

Urographic Manifestations of Metastatic Melanoma. HARVEY M. GOLDSTEIN, M.D., STEPHEN KAMINSKY, M.D., SIDNEY WALLACE, M.D., AND DOUGLAS E. JOHNSON, M.D. 801
The Increased Incidence of Carcinoma of the Colon following Ureterosigmoidostomy. RANDALL S. PREISSIG, M.D., WILLIAM F. BARRY, JR., M.D., AND RICHARD G. LESTER, M.D. 806
Detection and Localization of an Occult Vesicoenteric Fistula. EDWARD K. PROKOP, M.D., E. ULRIC BUDDEMEYER, SC.D., H. WILLIAM STRAUSS, M.D., AND HENRY N. WAGNER, JR., M.D. 811
Inferior Vena Caval Extension of Renal Carcinoma: A Lost Cause?: Roentgenographic and Pathologic Findings in Surgical Patients. DAVID L. MCCULLOUGH, M.D., AND LEE B. TALNER, M.D. 819
Calcification in Uroepithelial Tumors of the Bladder: Report of 5 Cases and Survey of the Literature. STEPHEN W. MILLER, M.D., AND RICHARD C. PFISTER, M.D. 827
Radiation Therapy and Pelvic Node Dissection in the Management of Cancer of the Prostate. BASIL S. HILARIS, M.D., WILLET F. WHITMORE, JR., M.D., MOSTAFA A. BATATA, M.D., AND HARRY GRABSTALD, M.D. . 832
Percutaneous Needle Localization of Clustered Mammary Microcalcifications Prior to Biopsy. BARBARA THREATT, M.D., HENRY APPELMAN, M.D., RICHARD DOW, M.D., AND TERRY O'ROURKE, M.D. 839
A Device for Precision Needle Biopsy of the Breast at Mammography. ANDERS MÜHLOW 843
Analysis of 462 Breast Carcinomas. JOHN N. WOLFE, M.D. 846
A New Biopsy Needle and Its Clinical Use. L. H. LEE, M.D. 854
Thermal Enhancement of the Radiosensitivity Using Cultured Normal and Neoplastic Cells. J. H. KIM, M.D., PH.D., S. H. KIM, M.S., AND E. HAHN, PH.D. 860
Computer Generated Isodose Curves for High Energy X-ray Machines: Experimental Verification and Time-Saving Strategies. KAREN LANE, B.A., PETER BLOCH, PH.D., AND LAWRENCE W. DAVIS, M.D. 865
Design of X-ray Targets for High Energy Linear Accelerators in Radiotherapy. E. B. PODGORŠAK, PH.D., J. A. RAWLINSON, M.SC., M. I. GLAVINOVIĆ, B.SC., AND H. E. JOHNS, PH.D. 873
Three-Dimensional Imaging of Abdominal Organs with Ultrasound. S. NØRBY RASMUSSEN, M.D., S. SKOGSTAD NIELSEN, M.SC., ROYAL J. BARTRUM, JR., M.D., BENT STIGSBY, M.D., AND HANS H. HOLM, M.D. . . . 883
Officers 889
Editorial
Fifty-sixth Annual Meeting of the American Radium Society. LUTHER W. BRADY, M.D. 890
National Research Council: Picker Foundation Awards, 1974–1975 . . . 897
National Council on Radiation Protection and Measurements (NCRP); Report No. 41 900
I.A.E.A. Catalogue of Radiation Dosimetry Data 901
News Items 902
Book Reviews 905
Books Received 906
Society Proceedings 907
Abstracts of Radiologic Literature 914
Subject Index to Volume 121 924
Author Index to Volume 121 933



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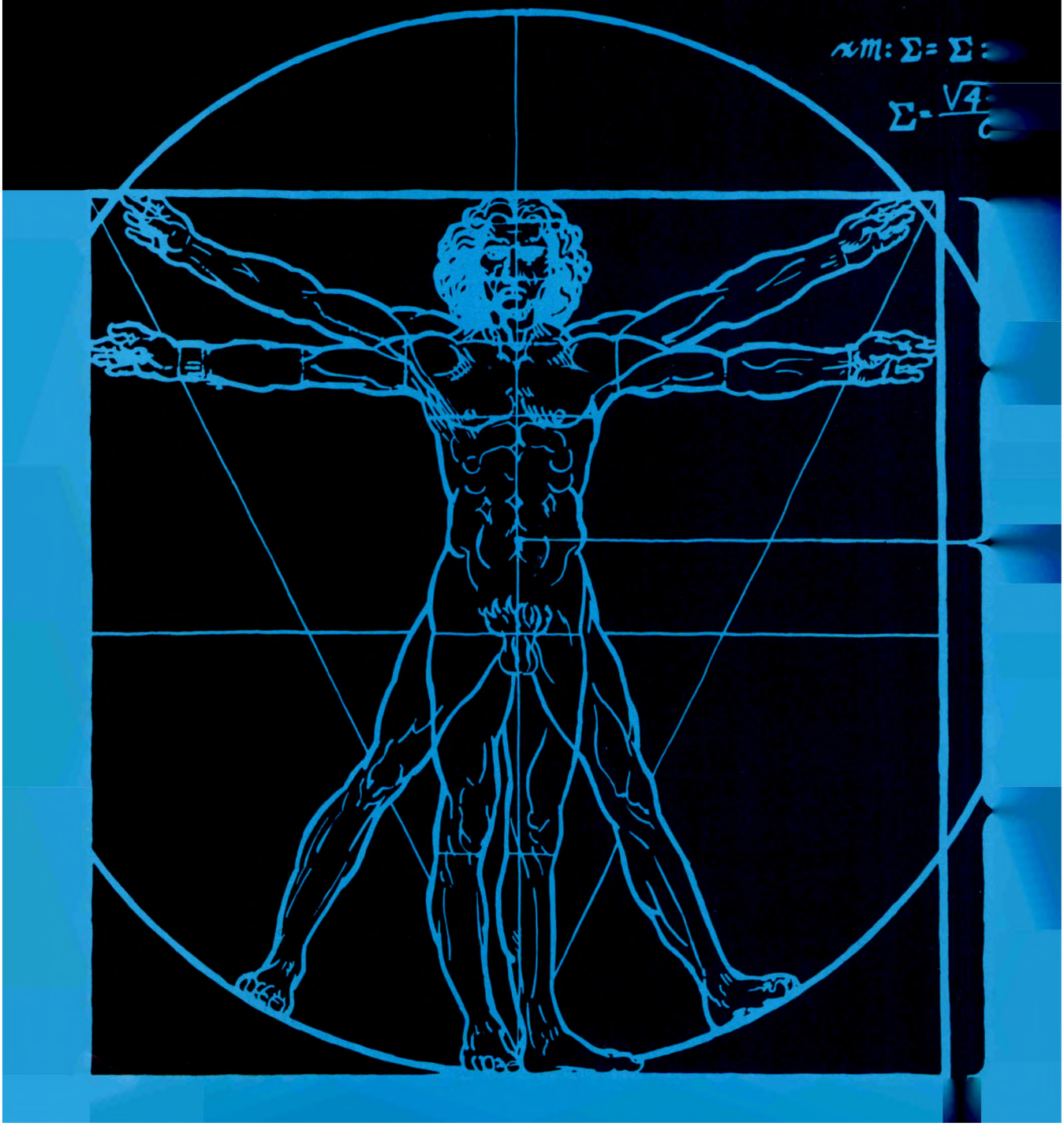
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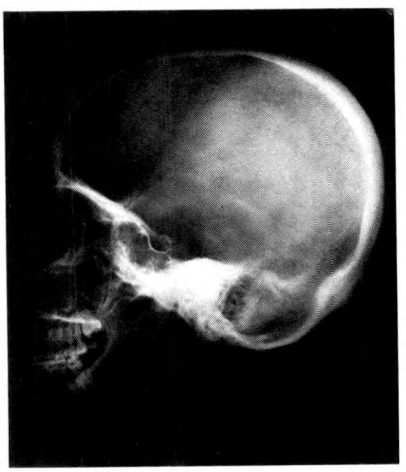
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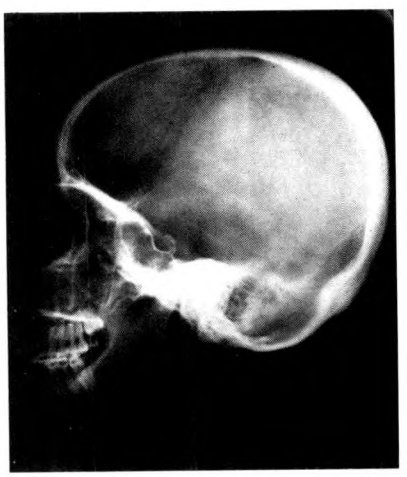
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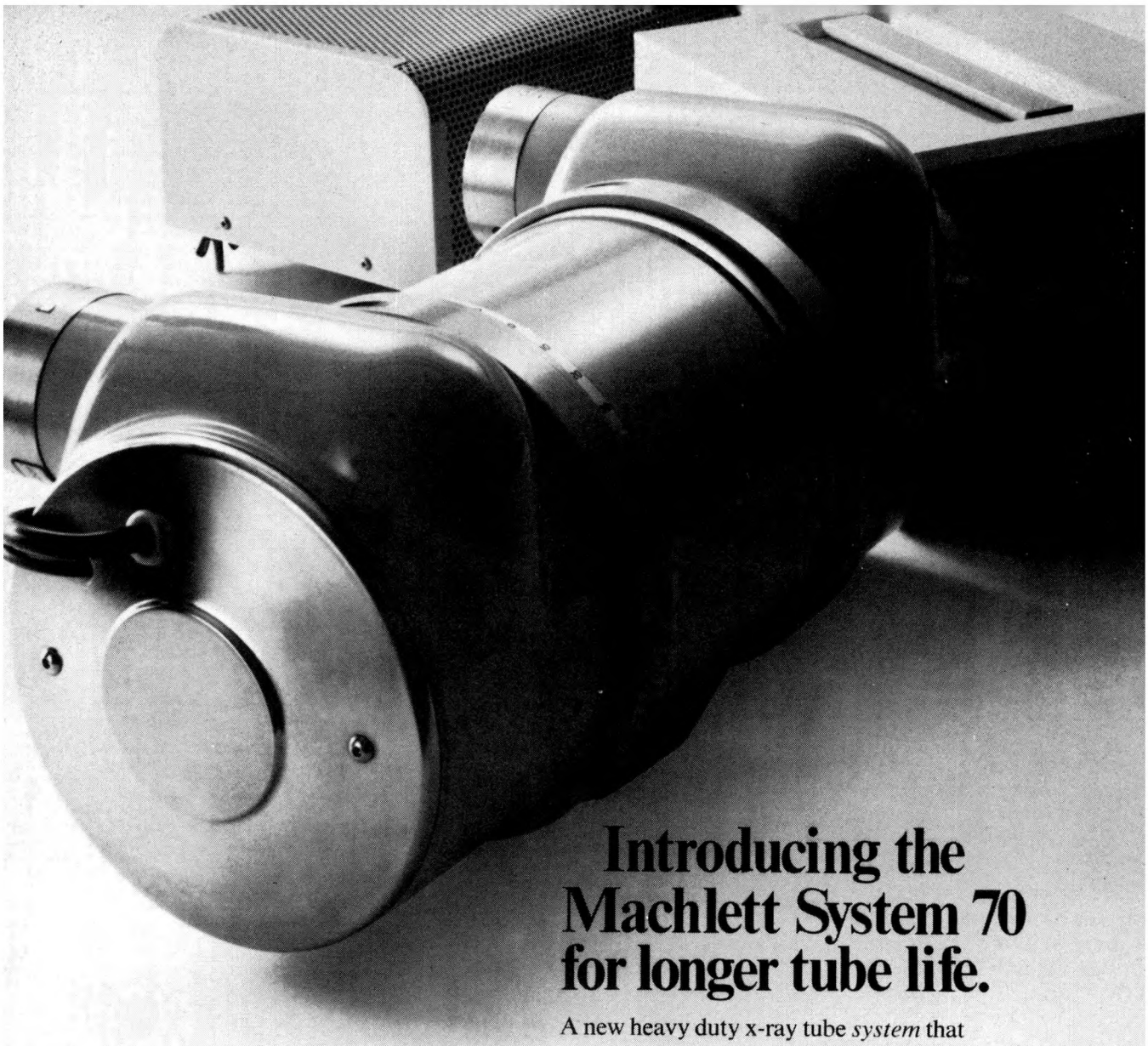
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*__Radiology__ 105: 185-190, October, 1972. X-ray exposure reduction using Rare-Earth Oxysulfide Intensifying Screens; Robert A. Buchanan, PhD., Solon I. Finkelstein, M.D., and Kenneth A. Wickersheim, PhD.

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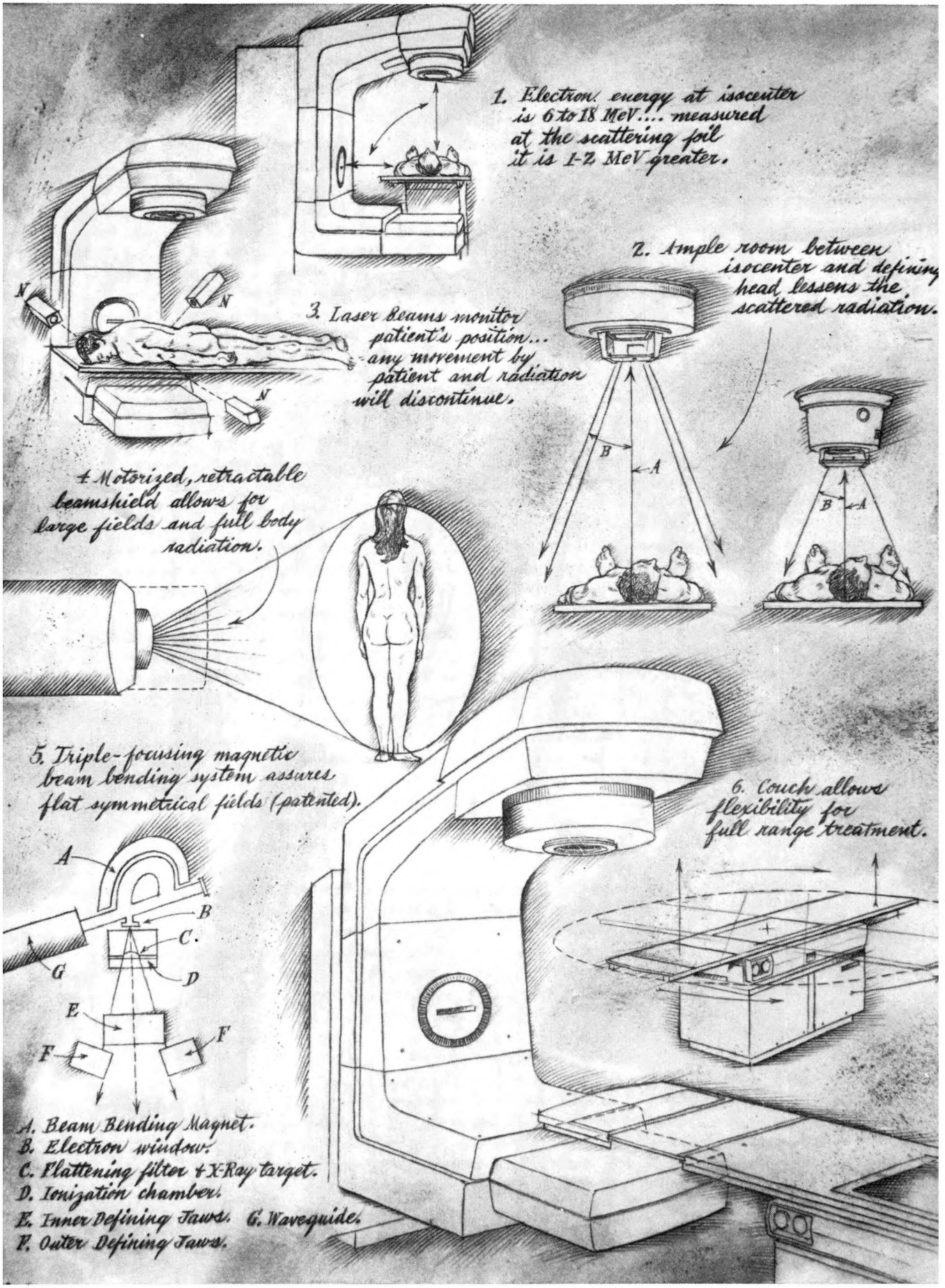
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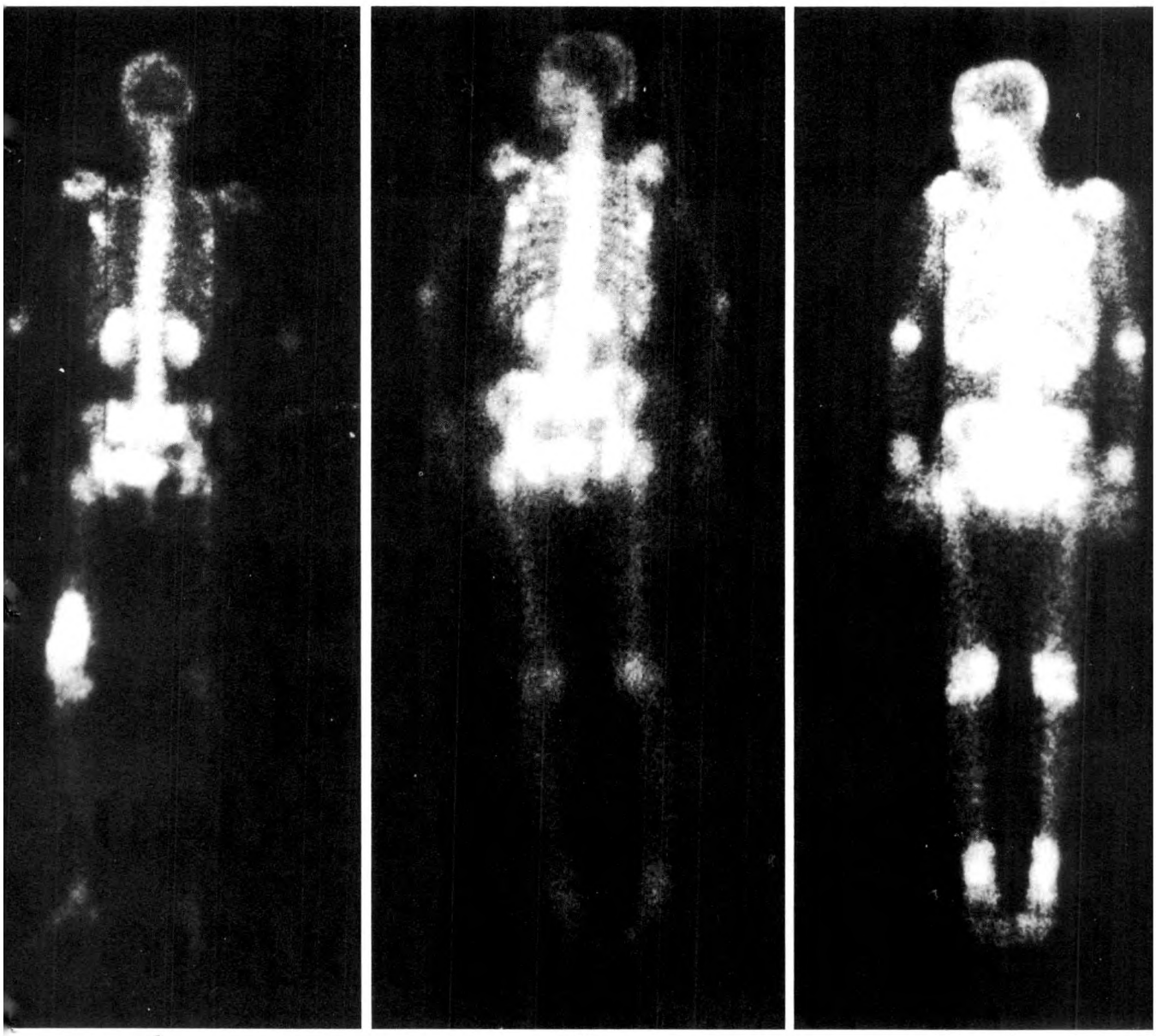
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Posterior: 961,752 counts/1054.3 sec (17.6 min)
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OSTEOSCAN

(5.9 MG DISODIUM ETIDRONATE 0.16 MG STANNOUS CHLORIDE)

SKELETAL IMAGING AGENT

See following page for brief summary of package insert.

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SKELETAL IMAGING AGENT



Brief summary of Package Insert. Before using, please consult the full Package Insert included in each kit.

DESCRIPTION

Each vial of OSTEOSCAN contains 5.9 mg disodium etidronate and 0.16 mg stannous chloride as active ingredients. Upon addition of ADDITIVE-FREE ^{99m}Tc-pertechnetate, these ingredients combine with ^{99m}Tc to form a stable soluble complex.

ACTIONS (CLINICAL PHARMACOLOGY)

When injected intravenously, ^{99m}Tc-labeled OSTEOSCAN has a specific affinity for areas of altered osteogenesis. Areas of bone which are undergoing neoplastic invasion often have an unusually high turnover rate which may be imaged with ^{99m}Tc-labeled OSTEOSCAN.

Three hours after intravenous injection of 1 ml ^{99m}Tc-labeled OSTEOSCAN, an estimated 40-50% of the injected dose has been taken up by the skeleton. At this time approximately 50% has been excreted in the urine and 6% remains in the blood. A small amount is retained by the soft tissue. The level of ^{99m}Tc-labeled OSTEOSCAN excreted in the feces is below the level detectable by routine laboratory techniques.

INDICATIONS

OSTEOSCAN is a skeletal imaging agent used to demonstrate areas of altered osteogenesis.

CONTRAINDICATIONS

None.

WARNINGS

This radiopharmaceutical should not be administered to patients who are pregnant or lactating unless the information to be gained outweighs the potential hazards.

Ideally examinations using radiopharmaceuticals, especially those elective in nature, of a woman of childbearing capability should be performed during the first few (approximately 10) days following the onset of menses.

Radiopharmaceuticals should be used only by physicians who are qualified by specific training in the safe use and handling of radionuclides produced by nuclear reactor or particle accelerator and whose experience and training have been approved by the appropriate government agency authorized to license the use of radionuclides.

The ^{99m}Tc-generator should be tested routinely for molybdenum breakthrough and aluminum. If either is detected, the eluate should not be used.

PRECAUTIONS

Both prior to and following ^{99m}Tc-labeled OSTEOSCAN administration, patients should be encouraged to drink fluids. Patients should void as often as possible after the ^{99m}Tc-labeled OSTEOSCAN injection to minimize background interference from accumulation in the bladder and unnecessary exposure to radiation.

As in the use of any other radioactive material, care should be taken to insure minimum radiation exposure to the patient, consistent with proper patient management, and to insure minimum radiation exposure to occupational workers.

ADVERSE REACTIONS

None.

DOSAGE AND ADMINISTRATION

The recommended adult dose of ^{99m}Tc-labeled OSTEOSCAN is 1 ml with a total activity range of 10-15 mCi. ^{99m}Tc-labeled OSTEOSCAN should be given intravenously by slow injection over a period of 30 seconds within three (3) hours after its preparation. Optimum scanning time is 3-4 hours postinjection.

The patient dose should be measured by a suitable radioactivity calibration system immediately prior to administration.

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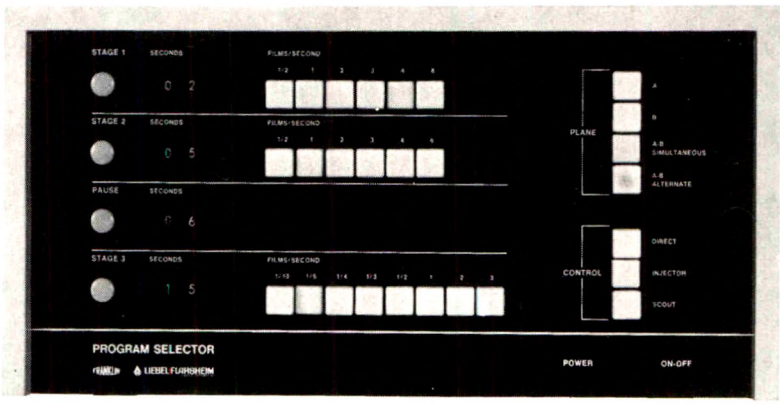
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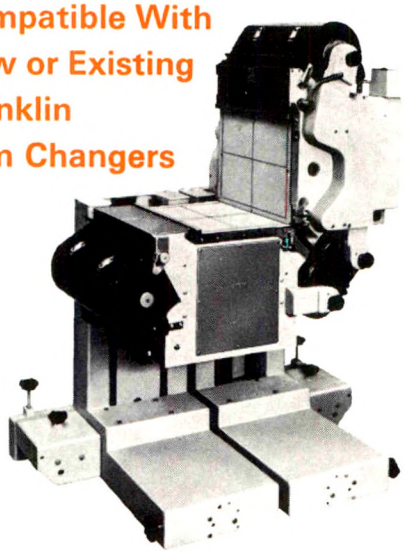
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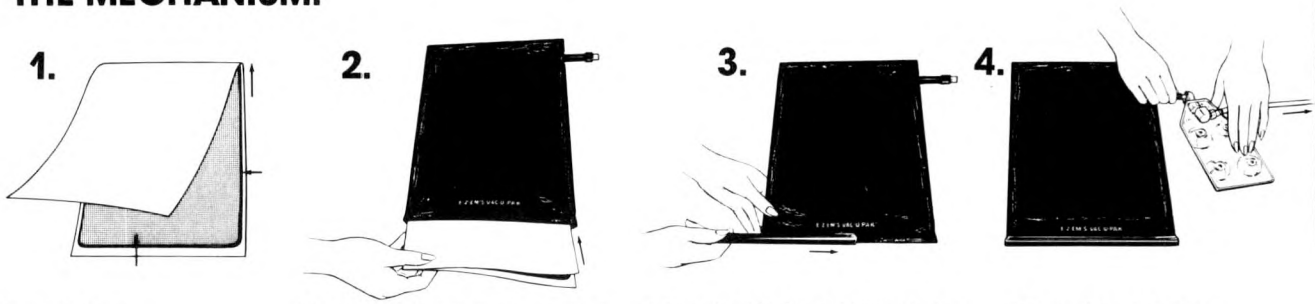
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QUANTITATIVE THYROID SCANNING: A RELIABLE THYROID FUNCTION TEST*

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MELBOURNE, AUSTRALIA

THE value of technetium pertechnetate ($Tc^{99m}O_4^-$) uptake studies as an index of thyroid function has been established.[3,13] Normal thyroid uptake levels of $Tc^{99m}O_4^-$ are of the order of 1 to 4 per cent, varying somewhat in the different series reported. One of the problems, therefore, with early $Tc^{99m}O_4^-$ uptake studies is the high extrathyroidal neck radioactivity. This has been overcome in various ways,[13,16] all of them having a degree of complexity which may involve measurements over the thyroid gland and adjacent areas in the neck, counting a reference source or phantom and taking into account the time factors involved. Despite these problems, early intravenous $Tc^{99m}O_4^-$ uptake studies continue to be more widely used and in 1972 gained recognition by a special International Atomic Energy Agency Panel.[13] The Panel approved several different methods for performing these tests but it was pointed out that a simpler method was desirable.

It was noted in the Department of Nuclear Medicine, Repatriation General Hospital, Melbourne in 1972, that the count rate over the thyroid gland in $Tc^{99m}O_4^-$ scans appeared to correlate well with the thyroid functional status. Since that period we have evolved a satisfactory method of $Tc^{99m}O_4^-$ Quantitative Thyroid Scanning (QTS). This technique is described and our experience in 137 euthyroid and 63 thyrotoxic patients is documented. The scans were evaluated in the light of the clinical thyroid status, *in vitro* thyroid function tests and I^{131} uptake studies. The results of this study indicate that calculation of extrathyroidal neck radioactivity and percentage uptake of the administered dose are not essential prerequisites for satisfactory intravenous $Tc^{99m}O_4^-$ uptake studies. QTS would appear to be a simple and reliable test in the diagnosis of thyrotoxicosis and more reliable than I^{131} in some cases of solitary toxic nodules.

MATERIAL AND METHOD

Quantitative Thyroid Scannings (QTS) performed on the first 200 patients who were shown to be either thyrotoxic or euthyroid were included in the study.

* From the Repatriation General Hospital, Heidelberg, Melbourne, Victoria, Australia.

Evaluation of thyroid status, in addition to QTS, included T3 resin uptake, serum thyroxine and free thyroxine index, 2 hour I^{131} uptake, clinical examination, and in all the thyrotoxic patients, subsequent follow-up. Patients with hypothyroidism, thyroiditis and those currently on drug therapy for thyrotoxicosis were excluded from the trial. Repeat studies performed on patients, whether thyrotoxic or euthyroid, were not included in the series. Patients with carcinoma of the thyroid invariably had additional thyroid scannings performed with I^{131} and were also excluded from the study. There were 2 patients in whom the thyroid status was equivocal at the time of writing and they were excluded from the series. This left 137 euthyroid and 63 thyrotoxic patients.

The intravenous dose of $Tc^{99m}O_4^-$ varied between 0.2 mc and 2 mc, the usual activity being 1 mc. Following initial calculation, the dose was drawn up in a mantoux syringe and checked in a Radx IV Dose Calibrator. The highest activity in c.p.s./mc in the thyroid gland at 20 minutes following injection was noted. Routine thyroid scannings were subsequently performed. The statistical quality (information density) of the scans varied but was never less than 1,000 counts per square centimeter.

Line spacing was 2 mm. and no background cut-off was used. The submandibular glands were incorporated in the field of view for the last 163 thyroid scans. In the latter part of the study, quantitative counting over the submandibular glands was routinely performed. The equipment used was a Selo D.S. 7 Digital Dual-head 3 inch Crystal Scanner, only the upper head being used. A high resolution, low energy collimator with a focal depth of 7 cm. and resolution of 9 mm. was used. Two hour I^{131} uptake studies were performed on fasting patients using a Selo Thyroid Uptake Probe, all parameters conforming to I.A.E.A. recommendations. Tetrasorb I125 and Triosorb I125 kits (Abbott Laboratories) were used for all the *in vitro* thyroid function tests performed in this Department. Twenty-three of the 63 thyrotoxic patients came from other hospitals where the methods used for the *in vitro* tests varied. Postsuppression studies were performed following a course of triiodothyronine 80 μg. daily for 1 week.

RESULTS

The mean uptake in the euthyroid group of patients was 322 c.p.s./mc (S.D. 134) compared with 1,400 c.p.s./mc (S.D. 861) in the thyrotoxic group (Fig. 1). Using 600 c.p.s./mc as the upper limit of normal (2 S.D. 54–590 c.p.s./mc) the reliability of the test was 98.5 per cent in the euthyroid group and 90.5 per cent in the thyrotoxic group, giving an over-all reliability in the 200 patients of 96 per cent. Comparative reliabilities in the thyrotoxic group were 84 per cent for 2 hour I^{131} uptakes (38 studies), 86 per cent for the T3 resin uptake (58 studies), 92 per cent for the serum thyroxine (63 studies), and 97 per cent for the

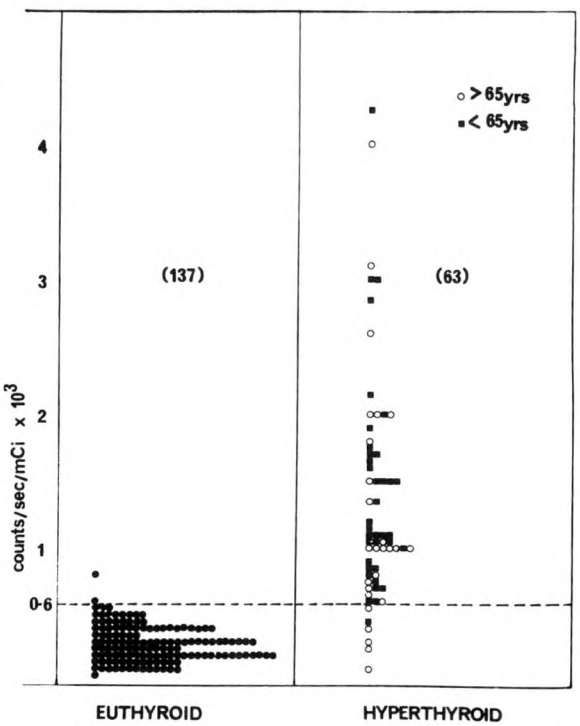


FIG. 1. Scattergram depicting intravenous $Tc^{99m}O_4^-$ 20 minute uptakes in 137 euthyroid and 63 thyrotoxic patients.

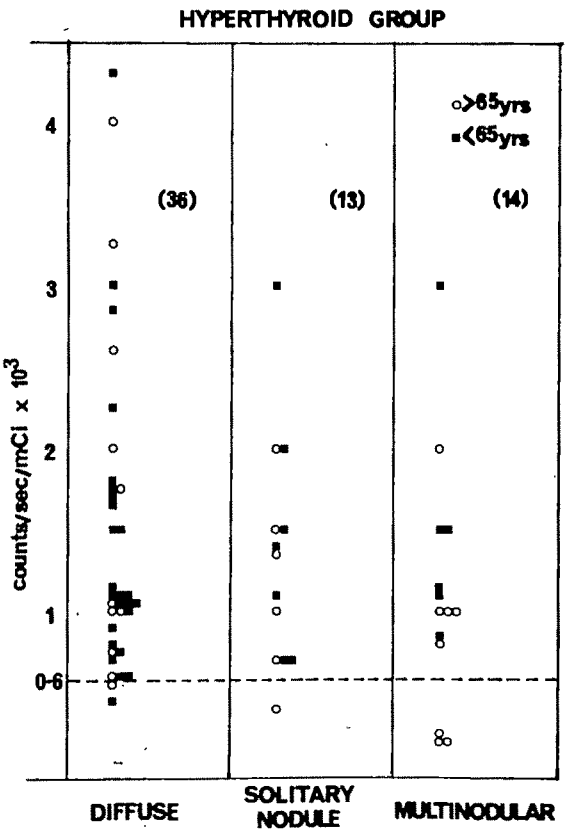


FIG. 2. Scattergram depicting intravenous $Tc^{99m}O_4^-$ 20 minute uptakes in subgroups of thyrotoxic patients.

free thyroxine index (58 studies). In the thyrotoxic group of 63 patients, 36 scans (57 per cent) showed diffuse uptake, 13 (21 per cent) a solitary hot nodule and in 14 (22 per cent) a multinodular goiter. The mean 20 minute intravenous $Tc^{99m}O_4^-$ levels were 1,580 (range 450–4,285), 1,349 (range 400–3,000) and 1,077 (range 200–3,000) c.p.s./mc, respectively (Fig. 2). The 6 thyrotoxic patients with results of less than 600 c.p.s./mc included 5 over the age of 65 years (Table I). In the 26 patients in this age group, the reliability of the test was 81 per cent in contrast to 97.3 per cent in the 37 patients under 65 years of age. There were 2 patients in whom the $Tc^{99m}O_4^-$ levels were normal and the I^{131} uptake studies were marginally elevated (Table I). Six thyrotoxic patients had I^{131} uptake levels in the normal range and 4 of these had elevated pertechnetate levels (Table II). Pre- and post-triiodothyronine suppression studies showed good correlation between I^{131} and $Tc^{99m}O_4^-$ levels (Table III). Submandibular gland quantitative counting in 50 consecutive euthyroid and 15 consecutive thyrotoxic patients showed a mean uptake of 109 c.p.s./mc (S.D. 42) in the euthyroid group and 131 c.p.s./mc (S.D. 44) in the thyrotoxic group. The mean uptake in all 65 patients was 114 c.p.s./mc (S.D. 42) (Fig. 3). The highest activity in any submandibular gland in the series was 230 c.p.s./mc. The size of the thyroid glands using the criteria of Tong and Rubenfeld[14] was within normal limits in 15 (24 per cent) of the 63 thyrotoxic patients, compared with 115 (83 per cent) of the 137 euthyroid patients. All 14 toxic multinodular glands were enlarged, whereas 10 of the 26 glands (38.5 per cent) showing diffuse uptake were within normal limits. In 8 patients (12.5 per cent) in the thyrotoxic group, the gland was retrosternal. The mean age of the thyrotoxic patients was 60.5 years with a range of 16 to 88 years.

DISCUSSION

The value of intravenous $Tc^{99m}O_4^-$ thyroid uptake studies in the diagnosis of thyrotoxicosis is now acknowledged.[1–4,6,9,10,13,15] Most important is the radiation dose to the patient's thyroid gland which with $Tc^{99m}O_4^-$ is approximately 200 mr/mc compared with a tracer dose of 5–10 μc of I^{131} resulting in a radiation dose to the thyroid of 5–10 rads. I^{123} is the most satisfactory iodine radionuclide for diagnostic purposes, but by virtue of its cyclotron production and relatively short half-life of 13 hours, is not readily available.

QTS appears to be as reliable as the $Tc^{99m}O_4^-$ uptake studies performed by the various methods previously described. As with other early $Tc^{99m}O_4^-$ uptake methods, QTS can be used to assess suppressibility of thyroid function while the patient is on Carbimazole therapy.[9,11] The ability to perform the test at short notice with the results available within 30 minutes has

TABLE I

FALSE NEGATIVE $Tc^{99m}O_4^-$ UPTAKE STUDIES

(Total Thyrotoxic Patients, 63)

Patient (Sex, Age)	Type of Gland	20 Minute $Tc^{99m}O_4^-$ Uptake (c.p.s./mc)	2 Hour I^{131} (Per Cent)	T.4 1 μg. Per Cent
1. G.C. (M) 72	Diffuse	550	—	7.4
2. A.B. (F) 79	Multinodular	200	—	7.3
3. W.T. (M) 88	Multinodular	250	15	7.8
4. E.M. (M) 57	Diffuse	450	21	10.9
5. D.L. (F) 67*	Solitary nodule	400	18	8.8
		360	16	
6. K.O'B. (F) 78	Multinodular	275	21	8.4

* Patient 5 had pre and post T3 suppression studies performed.

Normal Levels: $Tc^{99m}O_4^-$ 20 minute intravenous uptake<600 c.p.s./mc; 2 hour I^{131} uptake<20 per cent; thyroxine iodine 2.6–7.2 μg. per cent.

TABLE II

FALSE NEGATIVE 2 HOUR I^{131} UPTAKE STUDIES

(Total Thyrotoxic Patients, 38)

Patient (Sex, Age)	Type of Gland	2 Hour I^{131} (Per Cent)	20 Minute $Tc^{99m}O_4^-$ Uptake (c.p.s./mc)	T.4 1 μg. Per Cent
1. M.D. (M) 64	Solitary nodule	12	1,375	8.9
2. D.L. (F) 67*	Solitary nodule	18	400	8.8
		16	360	
3. V.M. (F) 70	Solitary nodule	19	1,364	9.9
4. R.K. (F) 76	Solitary nodule	16	1,000	8.4
5. C.H. (M) 55	Retrosternal	19	800	12.0
6. W.T. (M) 88	Multinodular	15	250	7.8

* Patient 2 had pre and post T3 suppression studies performed.

Normal Levels: $Tc^{99m}O_4^-$ 20 minute intravenous uptake<600 c.p.s./mc; 2 hour I^{131} uptake<20 per cent; thyroxine iodine 2.6–7.2 μg. per cent.

TABLE III

PRE AND POST T3 THYROID UPTAKE RESULTS*

Patient	2 Hour I^{131}		20 Minute $Tc^{99m}O_4^-$ Uptake (c.p.s./mc)		Final Status
	Pre (Per Cent)	Post (Per Cent)	Pre	Post	
1. L.V.	13	6	350	92	Euthyroid
2. A.A.	19	22	1,000	1,050	Toxic
3. R.M.	29	29	1,000	825	Toxic
4. G.M.	16	9	250	160	Euthyroid
5. D.L.	18	16	400	360	Toxic
6. O.B.	14	5	240	100	Euthyroid
7. K.V.	—	—	1,000	1,250	Toxic
8. K.J.	—	—	215	90	Euthyroid
9. S.D.	—	—	167	84	Euthyroid
10. M.M.	—	—	340	130	Euthyroid
11. B.W.	—	—	334	110	Euthyroid
12. S.W.	—	—	250	77	Euthyroid
13. I.B.	—	—	350	160	Euthyroid

* It was not felt justified to perform suppression studies using I^{131} in the younger patients.

proved very helpful in the occasional patients in whom early confirmation of thyroid status is desirable. In this situation, blood is taken for *in vitro* tests prior to the commencement of the quantitative thyroid scanning. There appears to be little justification for routinely performing I^{131} uptake studies on young patients in whom the diagnosis of thyrotoxicosis is queried. The scanning technique is not essential for uptake studies with $Tc^{99m}O_4^-$ which can be performed satisfactorily with the standard probe systems generally available or utilizing the gamma camera for carotid-thyroid transit time studies.[1,15]

$Tc^{99m}O_4^-$ has already been shown to be as reliable as I^{131} in suppression studies.[3,9,11]

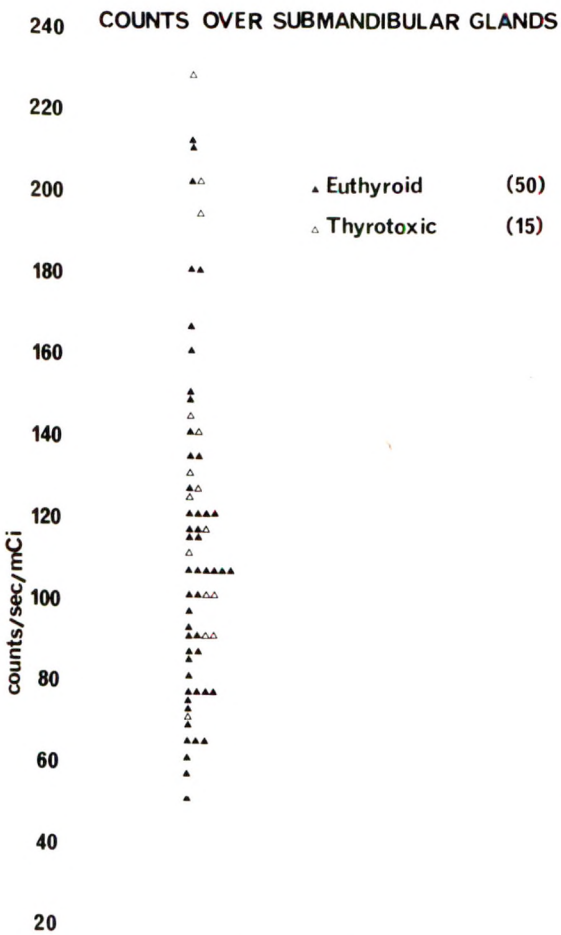


Fig. 3. Scattergram of submandibular counts depicting $Tc^{99m}O_4^-$ 20 minute uptakes in 50 euthyroid and 15 thyrotoxic patients.

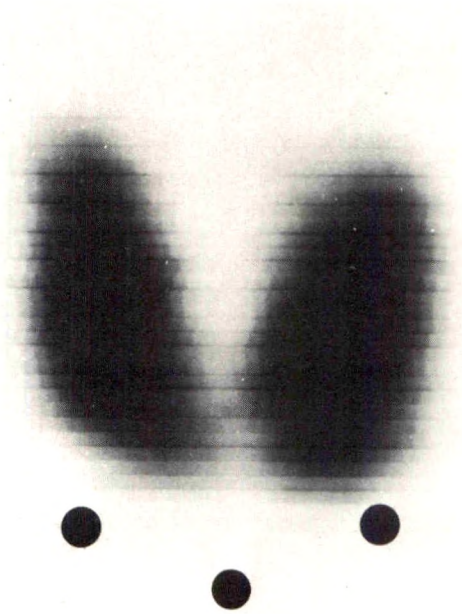


Fig. 4. Thyrotoxic gland: 3,000 c.p.s./mc.

This was confirmed in our small series and, in addition, the appearance of the thyroid scans themselves was characteristic. In the typical thyrotoxic patient no background activity was present and the submandibular glands were not visualized (Fig. 4). In contrast, in the euthyroid patients, extrathyroidal neck radioactivity and the submandibular glands were always visualized (Fig. 5). Following suppression, the activity in the submandibular glands usually exceeded the thyroid which was poorly delineated in the euthyroid group (Fig. 6, *A* and *B*), whereas in non-suppressible glands there was no significant change (Fig. 7, *A* and *B*). Quantitative estimations of the submandibular gland were performed, as it was important to confirm that the submandibular $Tc^{99m}O_4^-$ activity did not exceed the euthyroid level, although this had previously been noted in a study on a small number of patients.[7] It is helpful to incorporate the submandibular glands on the scans, as in addition to the characteristic appearances noted in different functional thyroid states, it does prevent erroneous results due to incorrect dosage

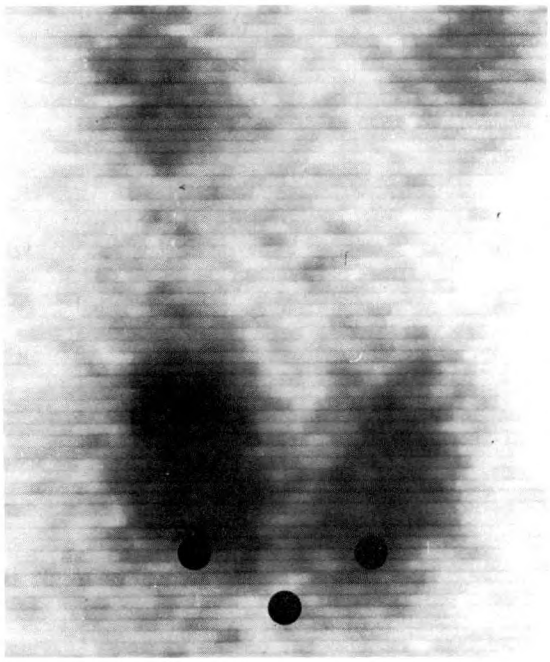


FIG. 5. Euthyroid gland: 250 c.p.s./mc.

given or misreading of the count rate. This technique provides a safeguard against these events, as the appearances of the scans are quite independent of the dose of $Tc^{99m}O_4^-$ administered to the patient. Although uptake readings were routinely taken at 20 minutes it was noted throughout the study that maximal thyroid uptake occurred between 15 and 25 minutes, with minimal change during this time, so that precise timing may not be critical.

The appearances in thyrotoxicosis factitia are striking and we have had 3 such cases (Fig. 8). QTS is a rapid and simple method of confirming this entity.

QTS is more informative than the usual uptake studies in so far as it combines a functional with a visual display of the thyroid gland and is thus comparable with dynamic thyroid studies performed on the gamma camera. In addition, the activity in different parts of the gland can be as-

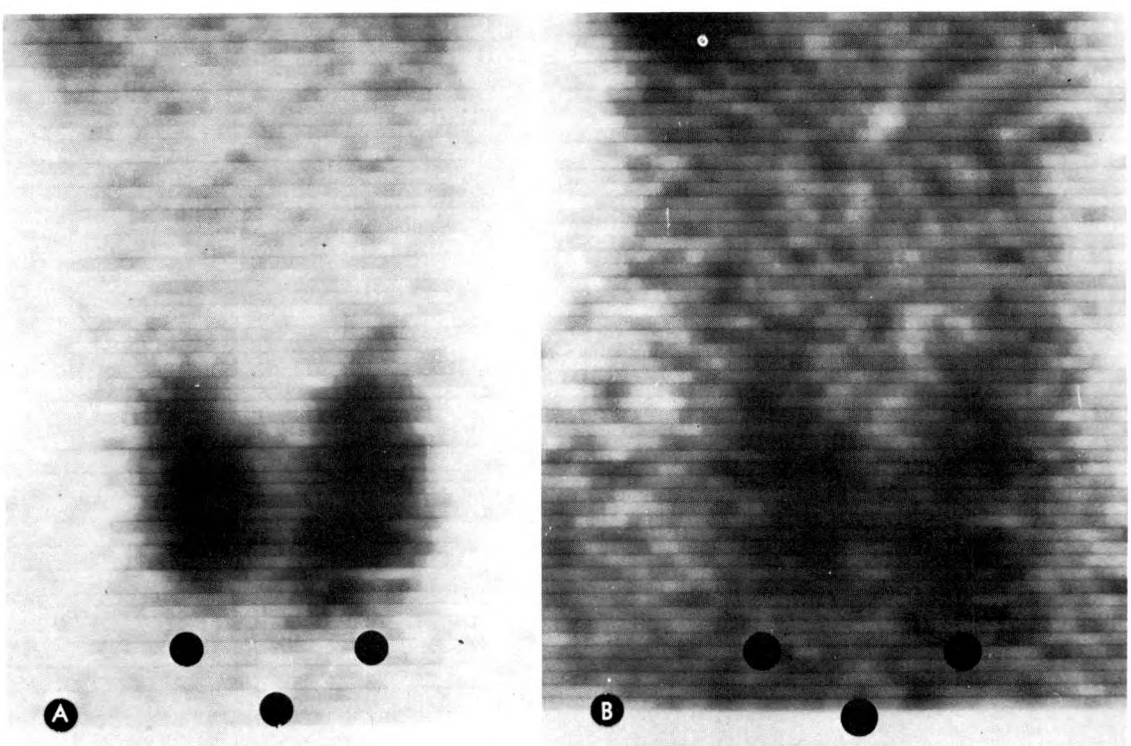


FIG. 6. Euthyroid gland. (*A*) Presuppression 315 c.p.s./mc. (*B*) Postsuppression 90 c.p.s./mc.

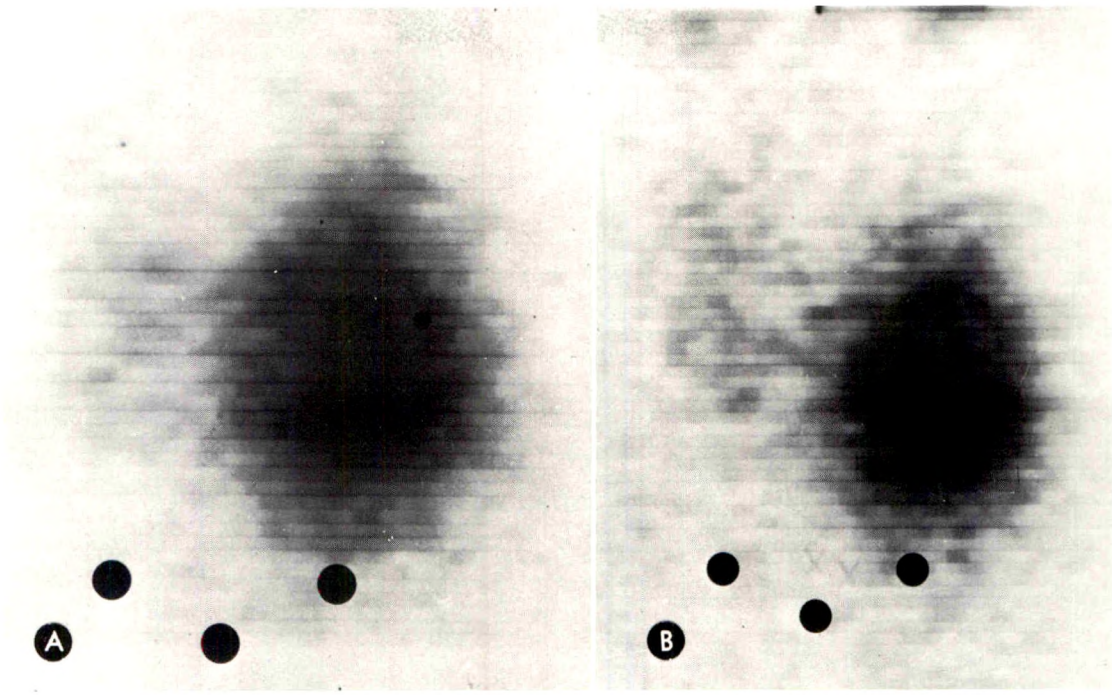


Fig. 7. Thyrotoxic gland. (*A*) Presuppression 400 c.p.s./mc. (*B*) Postsuppression 360 c.p.s./mc. Patient No. 5 (D.L.) in Table 1.

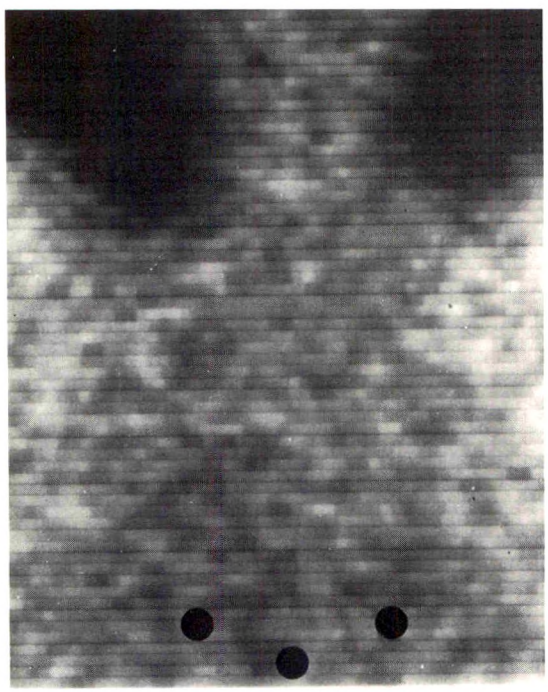


Fig. 8. Scan in thyrotoxicosis factitia: 66 c.p.s./mc. Note nondelineation of thyroid gland with relatively prominent submandibular glands: 105 c.p.s./mc. Thyroxine iodine 10.1 μg. per cent.

sessed and this in itself can be helpful as is demonstrated in Figure 9, in which the patient was thought to have a toxic nodule clinically, but subsequent scans showed the nodule in question to be suppressed with diffuse increased activity in the remainder of the gland. Readings can similarly be taken over hot and cold areas, respectively, and following radioiodine therapy, the response to treatment can be assessed with a fair degree of accuracy. The performance of the thyroid scanning following initial $Tc^{99m}O_4^-$ readings involves little extra work and only takes an additional 10–15 minutes. This is worthwhile as the scan may be helpful in determining the management of a particular patient and, in the case of radioiodine therapy, the optimum dosage to be given.

The technique used in this study differs significantly from all previously described thyroid uptake studies. It has always been the practice to determine the activity of the entire thyroid gland as a percentage of the total dose of radionuclide (I^{131} or $Tc^{99m}O_4^-$)

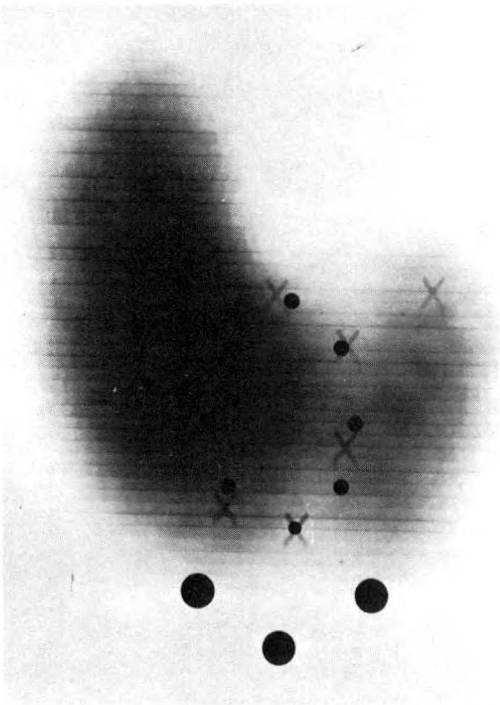


FIG. 9. Thyrotoxic gland. Markings indicate palpable midline nodule. Right lobe: 4,285 c.p.s./mc. Left lobe: 2,142 c.p.s./mc.

administered to the patient. In QTS only the absolute activity in a small well-defined area of thyroid tissue is measured. It has been shown that there is a variation in count rate dependent upon the type of gland, but even so, the results remain valid (Fig. 2). It is, however, important to appreciate that the results are least reliable in elderly patients with large multinodular goiters (Fig. 10). I^{131} uptake studies have similarly been shown to be less dependable in this group of patients.[12] It is of interest that QTS was more reliable (Table II) than I^{131} uptake studies in 3 patients with a hot nodule and 1 with a retrosternal goiter. The reason for this is uncertain, but it may be related to the relatively greater thickness of the toxic nodule as compared to a hyperactive gland with diffuse uptake. The latter situation is superior for counting with a nonfocusing (flat field) collimator, as is used for all I^{131} uptake studies. In the patients with solitary toxic nodules, the suppressed portion of the thyroid gland does not contribute significantly to the count rate, whereas in retrosternal goiter not all of the gland will be in the field of view of the collimator. These factors do not apply in QTS where a focusing collimator is used. We have recently had 2 more patients with solitary toxic nodules, not included in this series, with elevated $Tc^{99m}O_4^-$ levels (1,000 c.p.s./mc and 1,150 c.p.s./mc, respectively), and 2 hour I^{131} uptakes in the normal range (both 18 per cent). In those few cases with equivocal results the triiodothyronine suppression tests have always helped to arrive at the correct diagnosis (Table III).

This study was designed to assess the reliability of QTS in the diagnosis of thyrotoxicosis only. In the few cases of hypothyroidism in which it was used, pertechnetate uptake was usually decreased, except in 1 patient with Hashimoto's disease where it was increased. The value of pertechnetate studies in Hashimoto's disease has already been documented.[8]

CONCLUSION

Quantitative Thyroid Scanning (QTS) is a simple, reliable and informative test of thyroid function and preferable in many ways to I^{131} uptake studies in the diagnosis of thyrotoxicosis.

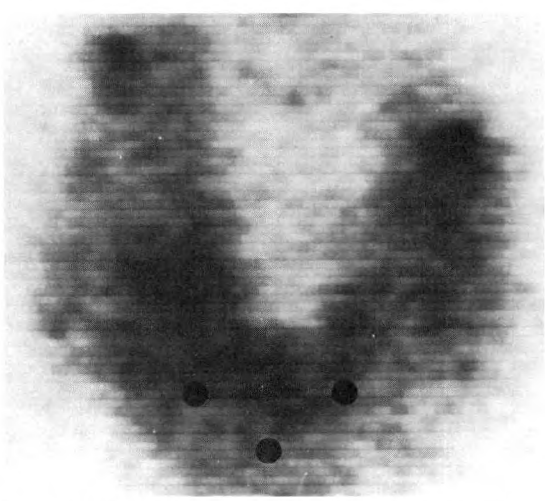


FIG. 10. Toxic multinodular retrosternal goiter: 200 c.p.s./mc. Patient No. 6 (W.T.) in Table II. At postmortem examination 7 months following this scan, the gland weighed 200 gm.

The information provided is complementary to *in vitro* thyroid function tests and is likely to remain so, despite the recent advances in the latter field. Calculation of extrathyroidal neck radioactivity and percentage uptake would not appear to be an essential prerequisite for satisfactory intravenous $Tc^{99m}O_4^-$ thyroid function studies.

Quantitative thyroid scanning is not suitable as a routine screening test, but is a valuable examination in the diagnosis of thyrotoxicosis.

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REFERENCES

1. Ashkar, F. S., and Smith, E. M. Dynamic thyroid study: rapid evaluation of thyroid function and anatomy using ^{99m}Tc as pertechnetate. *J.A.M.A.*, 1971, *217*, 441–446.
2. Atkins, H. L., and Richards, P. Assessment of thyroid function and anatomy with technetium-99m as pertechnetate. *J. Nuclear Med.*, 1968, *9*, 7–15.
3. Atkins, H. L. Technetium^{99m} pertechnetate uptake and scanning in evaluation of thyroid function. *Seminars Nuclear Med.*, 1971, *1*, 345–355.
4. De Garreta, A. C., Glass, H. I., and Goolden, A. W. G. Measurement of uptake of ^{99m}Tc by thyroid. *Brit. J. Radiol.*, 1968, *41*, 896–898.
5. Goolden, A. W. G., Glass, H. I., and Silvester, D. J. Choice of radioactive isotope for investigation of thyroid disorders. *Brit. J. Radiol.*, 1968, *41*, 20–24.
6. Goolden, A. W. G., Glass, H. I., and Williams, E. D. Use of ^{99m}Tc for routine assessment of thyroid function. *Brit. M. J.*, 1971, *4*, 396–399.
7. Harden, R. McG., Hilditch, T. E., Kennedy, I., Mason, K. D., Papadopoulos, S., and Alexander, W. D. Uptake and scanning of salivary glands in man using pertechnetate ^{99m}Tc. *Clin. Sc.*, 1967, *32*, 49–55.
8. Hauser, W., Atkins, H. L., Eckelman, W. C., and Richards, P. Thyroidal pertechnetate uptake in Hashimoto's disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *112*, 720–725.
9. Hoschl, R., and Gimlette, T. M. D. Diagnostic value of 20 minute pertechnetate ^{99m}Tc thyroid uptake. *Nuklear Medizin*, 1971, *10*, 305–315.
10. Hurley, P. J., Maisey, M. N., Nataragan, T. K., and Wagner, H. N., Jr. Computerized system for rapid evaluation of thyroid function. *J. Clin. Endocrinol.*, 1972, *34*, 354–360.
11. Shimmins, J. G., Alexander, W. D., McLarty, D. G., Robertson, J. W. K., and Sloane, D. J. Pertechnetate ^{99m}Tc for measuring thyroid suppressibility. *J. Nuclear Med.*, 1971, *12*, 51–54.
12. Stiel, J. N., Hales, I. B., and Reeve, T. S. Thyrotoxicosis in elderly population. *M. J. Australia*, 1972, *2*, 986–988.
13. Thyroid radionuclide uptake measurements—report of International Atomic Energy Agency Panel. *Int. J. Appl. Radiat. Isot.*, 1972, *3*, 305–313.
14. Tong, E. C. K., and Rubenfeld, S. Scan measurements of normal and enlarged thyroid glands. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 706–708.
15. Van't Hoff, W., Pover, G. G., and Eiser, N. M. Technetium^{99m} in diagnosis of thyrotoxicosis. *Brit. M. J.*, 1972, *4*, 203–206.
16. Williams, E. D., Glass, H. I., Goolden, A. W. G., and Satyavanich. Comparison of two methods of measuring thyroidal uptake of ^{99m}Tc. *J. Nuclear Med.*, 1972, *13*, 159–162.

AN UNUSUAL CONCENTRATION OF RADIO-PERTECHNETATE IN A PATIENT WITH THYROID CANCER*

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GANATRA *et al.*[1] recently reported an unusual concentration of radioiodide in the nonlactating breasts of 1 of their patients investigated for lung metastases after thyroidectomy for carcinoma of the thyroid.

In their review of the literature, they found only 1 previous report of such a concentration of radioiodide in nonlactating breasts of thyroidectomized females.[3] The rarity of this observation merits the reporting of additional cases.

In this article, a similar response, but with $Tc^{99m}O_4^-$, is presented.

REPORT OF A CASE

A single 19 year old female with a histologically proven papillary and follicular adenocarcinoma of the thyroid, for which total thyroidectomy had been carried out 13 years previously, was investigated for a possibility of lung metastases. At the time of scanning, she was complaining of menstrual irregularities of 1 year's duration and had just been submitted to a T.S.H. stimulation test of 5 days' duration.

A thoracic scan was obtained immediately after administration of 4 mc $Tc^{99m}O_4^-$ (Fig. 1*A*) and again 3 hours later (Fig. 1*B*). No lung lesions were demonstrated on the scans; however, on the delayed scan of the chest, the breasts were clearly revealed (Fig. 1*B*).

DISCUSSION

It is not too much of a surprise to encounter this response with $Tc^{99m}O_4^-$, once the phenomenon has been observed with radioiodide, since their respective metabolism in various parts of the body follows many similar pathways.

Meighan and Dworkin[2] reported recently an uptake of $Tc^{99m}O_4^-$ in the mammary gland tissue of a completely thyroid-

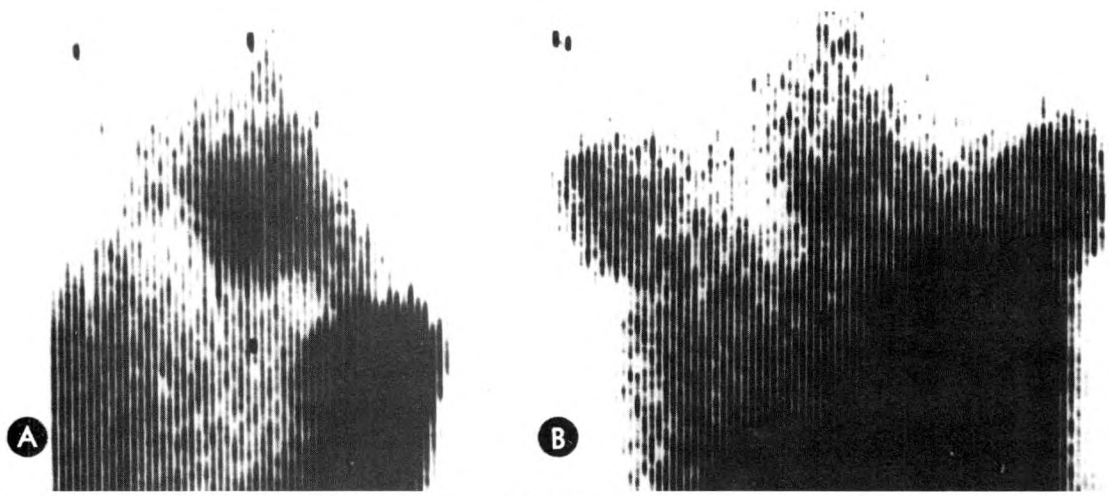


FIG. 1. (*A*) Scan obtained immediately after administration of 4 mc $Tc^{99m}O_4^-$. No uptake is seen in the area of mammary gland tissue. (*B*) Scan obtained 3 hours later. Uptake is now seen in the area of mammary gland tissue.

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ectomized 55 year old female. The chest scan was obtained half an hour after administration of 5 mc $Tc^{99m}O_4^-$. However, no significant uptake was reported in the area of the breasts in the same patient when a chest scan was obtained 24 hours after administration of 1 mc I^{131}. No explanation was offered for this discrepancy.

CONCLUSION

The few observations reported to date in the literature were all made, to our knowledge, in thyroidectomized patients. This may be due to the fact that radioiodide and radiopertechnetate chest surveys are routinely performed almost only in thyroidectomized patients. Along this line of thought, we reviewed some of our pertechnetate chest scans obtained on the Dynapix, a large area rectilinear scanner, for other conditions such as pericardial effusion. In rare cases, trace amounts of the radionuclide could be suspected over the breast areas in nonlactating euthyroid patients.

Nonlactating breasts have the potential to concentrate radioiodide and radiopertechnetate at levels detectable on scans. In this paper, the most striking case encountered in our laboratory is presented.

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REFERENCES

1. Ganatra, R. D., Atmaram, S. H., and Sharma, S. M. Unusual site of radioiodide concentration in patient with thyroid cancer. *J. Nuclear Med.*, 1972, *13*, 777.
2. Meighan, J. W., and Dworkin, H. J. Failure to detect ^{131}I positive thyroid metastases with ^{99m}Tc. *J. Nuclear Med.*, 1970, *11*, 173–174.
3. Sitterson, B. W., and Andrews, G. A. Introduction to thyroid scanning. In: Progress in Medical Radioisotope Scanning. Oak Ridge, ORINS, 1962, pp. 279–295.

DIAGNOSIS OF BARRETT'S ESOPHAGUS WITH RADIOISOTOPES*

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THE replacement of the normal stratified squamous lining of the lower esophagus by columnar epithelium similar to that of the stomach is known as Barrett's esophagus.

Patients who have this disease develop in most cases esophagitis and ulceration, the usual symptoms being retrosternal and epigastric pain, pyrosis, belching, dysphagia, and sometimes hematemesis.

Diagnosis of this condition is attempted by a roentgenologic study of the upper gastrointestinal tract with barium contrast medium, by which stenosis of the distal part of the esophagus and ulceration are usually found, and by esophagoscopy with measurements of the motility and mucosal electrical potentials.

With these techniques, the diagnosis may be strongly suspected, and is usually confirmed by mucosal biopsy and surgery.

In this report, we describe the application of another diagnostic procedure, making use of radioisotopes, based on the selective concentration of Tc^{99m} pertechnetate by the ectopic gastric mucosa found in this disease.

MATERIAL AND METHOD

The radioisotopic technique was used in a group of 15 persons who were considered to have no disease of the upper gastrointestinal tract and whose findings were considered normal. The patients of this group received the radioisotope for brain scanning, which was performed after the application of this method.

A second group of 7 patients suspected of having Barrett's esophagus was studied with the same method. These patients had been previously examined by roentgenologic and endoscopic conventional methods. Subsequently, the diagnosis was confirmed in every case by mucosal biopsy and by surgery. In all cases of suspected Barrett's esophagus, 5 millicuries Tc^{99m} pertechnetate was administered endovenously without any previous preparation. In all subjects considered for control, a standard dose for brain scanning (15 mc) was used.

With the patient standing, in order to avoid reflux of gastric contents from the stomach into the distal esophagus, scintigraphic images were obtained every 5 minutes after the injection, for a total of 40 minutes. The optimal images were obtained between 20–30 minutes. For each image, 300,000 counts of radioactivity were accumulated.

The scintigraphic images were made with a Nuclear Chicago H.P. Anger camera, provided with a 28.75 cm. (11.5 inch) crystal detector, annexed to a 4096 channel data processor. Analogue or digitized information in black and white was recorded on Polaroid film.

RESULTS

In all cases considered as normal, the images showed a uniform concentration of the radioisotope by the gastric mucosa, with a clear limitation of the profile of the organ (Fig. 1). In 1 case the air which is usually present in the gastric fundus created an image of decreased concentration in this region.

In all cases of Barrett's esophagus, apart from the normal concentration in the stomach, concentration of the radioisotope was also observed in the distal esophagus,

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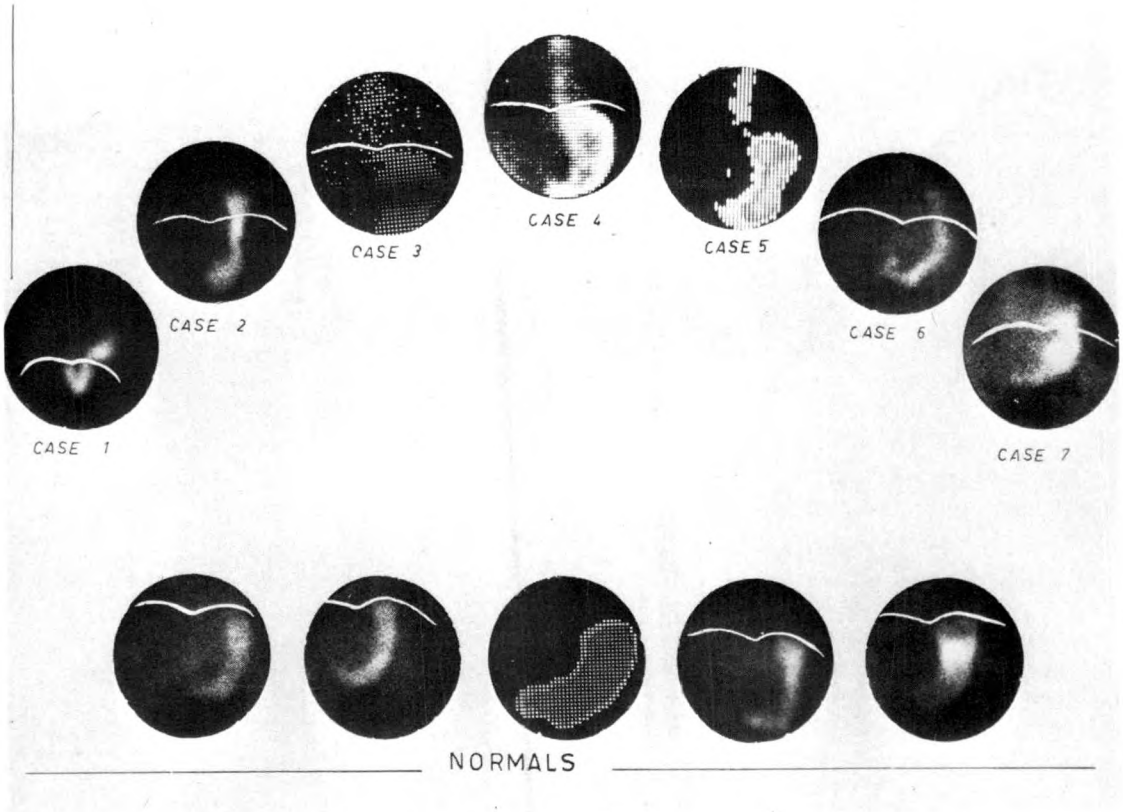


FIG. 1. *Upper row.* Tc^{99m} pertechnetate scintigraphic images of 7 cases of Barrett's esophagus. *Lower row.* Scintigraphic images of 5 normal cases.

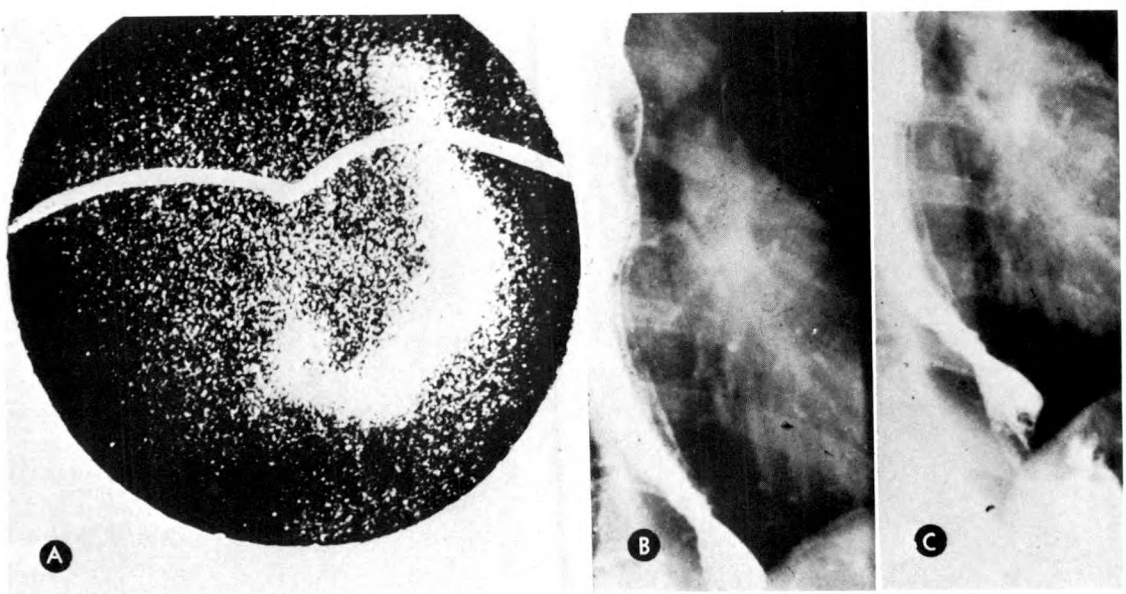


FIG. 2. Barrett's esophagus. Case 6. (*A*) Scintigraphic image. (*B* and *C*) Roentgenographic images.

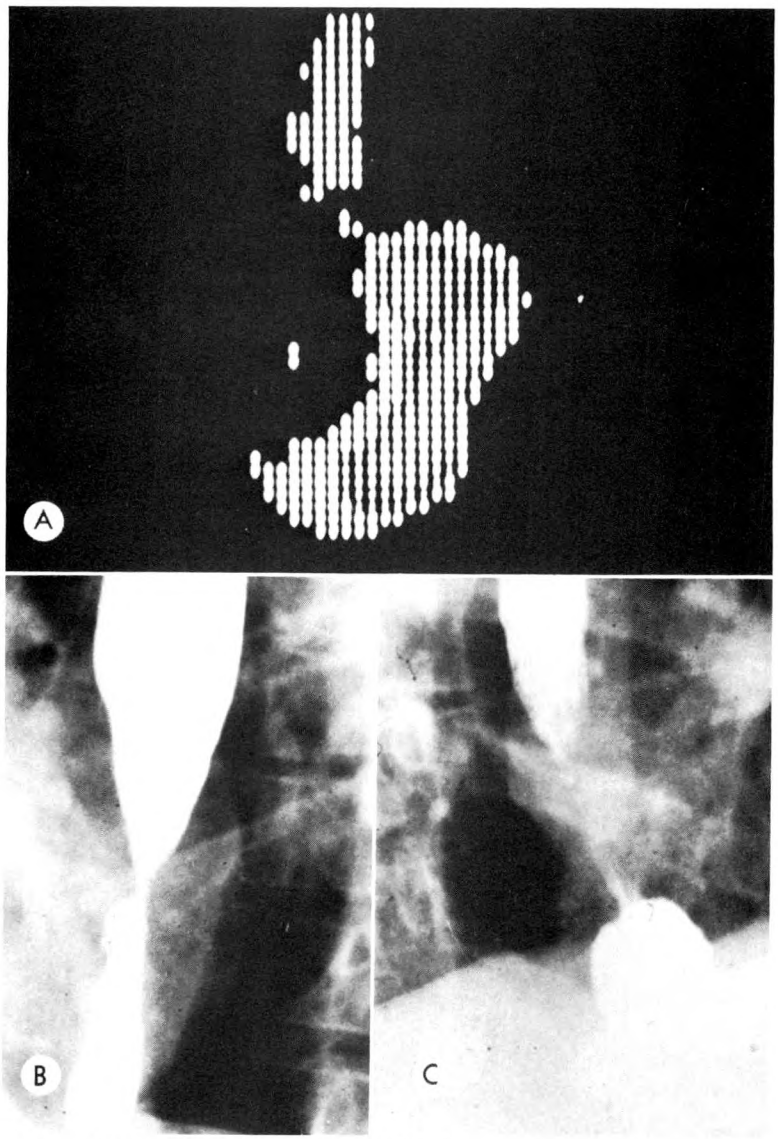


FIG. 3. Barrett's esophagus. Case 5. (*A*) Scintigraphic image. (*B* and *C*) Roentgenographic images.

corresponding to the location of the ectopic gastric mucosa (Fig. 1–3). In every case, the scintigraphic findings had a good correlation with the previous roentgenologic and endoscopic findings and the findings noted at surgery.

DISCUSSION

In 1950, Barrett[1] described a condition, with congenital etiology, in which the squamous stratified epithelium of the lower esophagus was replaced by columnar epithelium similar to that of the stomach.

In 1958, Wolf and his co-workers[6] proposed a second theory, assuming that this condition could be due to a pathologic process during fetal development. In support of this theory, they cited the persistence of islets of columnar epithelium within the squamous epithelium of the esophagus—the area where the ulcers described by Barrett were localized.

In 1960, Goldman and Beckman[4] demonstrated the gradual appearance of co-

lumnar epithelium in the lower esophagus after a prolonged period of irritation produced by reflux of acid gastric juice.

Recently, Burgess *et al.*[3] described a linear ulceration and esophagitis in 14 of 17 patients with Barrett's esophagus. These findings in the human have been corroborated by the results of experimentation in animals, in which columnar epithelium developed in the esophageal mucosa after removal of the squamous epithelium of the lower esophagus and establishment of reflux by cutting the inferior sphincter.

The radioisotopic method is based on the use of Tc^{99m} pertechnetate which shows selective concentration in the gastric mucosa. Tc^{99m} pertechnetate is obtained by eluting a Mo^{99m}-Tc^{99m} generator with normal saline solution. The process is performed aseptically. Tc^{99m} has a half life of 6 hours and emits a monoenergetic gamma photon of 140 kev., which reduces the radiation received by the patient and adapts perfectly to the collimators of the Anger camera.

Pertechnetate ion replaces the anions of the elements of Group VII and, when introduced into the body, is accumulated by the thyroid, the salivary glands and by the gastric mucosa, as well as by other organs, but not by the esophagus. It has been effectively used as a diagnostic aid, mainly for brain scanning.[5] Its concentration by the gastric mucosa is the basic principle for the use of this radioisotope in the detection of ectopic gastric mucosa, as in cases of Barrett's esophagus and Meckel's diverticulum. With Berquist *et al.*,[2] who described their results in 1 case of Barrett's esophagus, we have corroborated the usefulness of this method as a new diagnostic aid in such cases.

The radiation dose absorbed by the patient is minimal, and permits the use of Tc^{99m} in amounts ample enough to produce a large number of counts of radioactivity, which generally improves the diagnostic efficiency of the method.

CONCLUSION

Diagnosis of Barrett's esophagus with Tc^{99m} pertechnetate is a fast, easy, reliable and innocuous method, that may be used as a complementary procedure in the study of patients suspected of having this disease.

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REFERENCES

1. Barrett, N. R. Chronic peptic ulcer of oesophagus and oesophagitis. *Brit. J. Surg.*, 1950, *38*, 175–182.
2. Berquist, T. H., Nolan, N. G., Carlson, H. C., and Stephens, D. H. Diagnosis of Barrett's esophagus by pertechnetate scintigraphy. *Mayo Clin. Staff Proc.*, 1973, *48*, 276–279.
3. Burgess, J. N., Payne, W. S., and Andersen, H. A. Barrett esophagus: columnar-epithelial-lined lower esophagus. *Mayo Clin. Staff Proc.*, 1971, *46*, 728–734.
4. Goldman, M. C., and Beckman, R. C. Barrett syndrome: case report with discussion about concepts of pathogenesis. *Gastroenterology*, 1960, *39*, 104–110.
5. Mikolajków, A., and Chomicki, O. A. Scanning of stomach with ^{99m}Tc. *Digestion*, 1970, *3*, 357–367.
6. Wolf, B. S., Marshak, R. H., and Som, M. L. Peptic esophagitis and peptic ulceration of esophagus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1958, *79*, 741–759.

CLINICAL EVALUATION OF PREOPERATIVE LIVER SCINTIGRAPHY IN GASTRIC CARCINOMA PATIENTS*

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RECENT Japanese advances in techniques for roentgenologic examination of the stomach, in endoscopy, and in biopsy have increased the detection rate of early gastric carcinoma, whose infiltration is still localized to the mucosa or submucosa.[9] However, the majority of gastric carcinomas are still detected after the cancer cells have reached the serosa.[14] Hereinafter, the latter condition is referred to as the "progressive stage of gastric carcinoma."

In this progressive stage, the rate of metastasis to the liver is high. Autopsy findings reveal liver metastases in 30–70 per cent of gastric carcinoma.[2,15,19] The detection rate of liver metastases at surgery is lower—reportedly between 5 and 15 per cent.[12] It is very important to know whether liver metastases are present when therapeutic procedures for progressive gastric carcinoma are instituted.

Liver scintigraphy has been widely accepted as an easy, safe method to detect liver metastases, but its capability to detect metastatic foci is somewhat limited. Present liver scintigraphic techniques cannot detect metastatic foci less than 2 cm. in diameter. After they have exceeded 4 cm., they are practically always detectable, irrespective of the hepatic site.[6,7,20] Therefore, the value of liver scintigraphy is dependent on the size of metastatic foci in the liver when progressive gastric carcinoma is diagnosed.

Since gastrectomy is often performed for gastric carcinoma, even in the progressive stage, gross confirmation of liver metastases is facilitated. Our experience in correlating preoperative liver scintigraphy and surgical findings in progressive gastric carcinoma is reported here.

METHOD AND MATERIAL

Fifteen minutes after the intravenous administration of 250 μc Au^{198} colloid or 3 mc Tc^{99m} colloid, scanning was performed using a scintiscanner (Shimadzu, SCC-150S) with a 5 inch×2 inch NaI crystal, and a focusing collimator having either 37 holes and a 7.5 cm. focus, or 109 holes and an 11 cm. focus. Frontal and right lateral views were obtained in all cases. The scanning speed was 100 cm./min. In all cases photographic records were made; in some, recording was by the data blending method using a Gaussian filter.

A total of 1,379 liver scintigraphies were performed during 2 years, 150 of which were part of preoperative evaluations for detecting hepatic metastases from gastric carcinoma. These 150 were the subject of the present study. Confirmation methods and the results obtained are shown in Table I.

Confirmation was by autopsy in 7 cases, by laparotomy in 128 cases, and by clinical diagnoses in 15 cases. Fifteen of those diagnosed clinically did not have laparotomy because of obvious hepatic metastases.

One hundred and thirty-seven of the 150 cases had progressive gastric carcinoma; and 13, "early" gastric carcinoma limited to the mucosa or submucosa. These 13 were preoperatively diagnosed as progressive gastric carcinoma. All cases diagnosed as "early" gastric carcinoma had no preoperative liver scintigraphy. The Borrmann's classification of the 137 progressive gastric carcinomas is shown in Table II. Borr-

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Table I

METHODS OF CONFIRMING LIVER METASTASES

(150 cases)

Confirmation	Metastases		Total
	Present	Absent	
Autopsy	4	3	7
Laparotomy	25	103	128
Clinical Diagnosis	15	0	15
Total	44 cases	106 cases	150 cases

mann's Type II and III predominated in 85 per cent of these. In all cases, surgery was within 2 weeks of the liver scintigraphy.

RESULTS

DIAGNOSTIC ACCURACY

In 44 cases with liver metastases, 37 had space-occupying lesions scintigraphically, for a detection rate of 84 per cent. Of 106 cases, without metastases, 99 had normal scintigrams, for an accuracy of 93 per cent. The diagnostic accuracy of liver scintigraphy in detecting liver metastases was 90.7 per cent (136/150), as shown in Table III. No liver metastases were observed in the 13 cases with early gastric carcinoma.

Table II

CATEGORIES ACCORDING TO BORRMANN'S CLASSIFICATION OF GASTRIC CANCER

Type	No. of Cases
"Early" gastric cancer	13
I	9
II	57
III	60
IV	10
Unclassifiable	1

FALSE NEGATIVES AND FALSE POSITIVES

The reasons for the false negative and false positive diagnoses are shown in Table IV.

Among the 44 cases with liver metastases. there were 7 (16 per cent) false negatives. Surgery revealed scattered metastatic foci less than 2 cm. in diameter in 4. Even retrospectively, no scintigraphic abnormalities could be identified in these 4 cases.

In the remaining 3 patients, metastatic

Table III

SCINTIGRAPHIC DETECTION OF LIVER METASTASIS

Liver metastasis \ Scintigram	Abnormal Low Activity Area		Total
	Present	Absent	
Present	37	7	44
Absent	7	99	106
Total	44 cases	106 cases	150 cases

True positive $\frac{37}{44} = 84\%$

True negative $\frac{99}{106} = 93\%$

Correct diagnosis $\frac{136}{150} = 90.7\%$

TABLE IV

CAUSES OF FALSE NEGATIVE AND FALSE POSITIVE CASES: A RETROSPECTIVE ANALYSIS

False negative $\frac{7}{44}=15.9\%$	
1. <2 cm. diameter metastatic foci	4 cases
2. Overlooking abnormal low activity areas on scintigrams	3 cases
False positive $\frac{7}{106}=6.6\%$	
1. Error due to gallbladder fossa at inferior hepatic margin	2 cases
2. Error due to unsatisfactory examination	2 cases
3. Displacements and thinning due to extrahepatic tumors suggesting metastasis	3 cases

foci larger than 4 cm. in diameter were demonstrated at laparotomy. Retrospectively, review of these 3 cases suggested possible low activity areas on the scintigrams caused by metastatic foci. In one of these, a metastatic focus in the left hepatic lobe was misinterpreted as an area of low activity because this lobe itself was thin. In the second case a well-defined defect in the lower portion of the right hepatic lobe was regarded merely as a deformity of the liver. In the third, an irregular defect extending from the right hepatic lobe to the portal fissure was overlooked.

Seven (6.6 per cent) of the 106 cases proved to be false positives, because the low activity areas on the scintigrams have been attributed to space-occupying lesions. Retrospectively, 2 of these show isolated defects in the inferior portion of the right lobe. A low activity area due to the gallbladder was misdiagnosed as a metastasis. Small patchy hepatic defects in 2 cases were misdiagnosed as metastases. These misdiagnoses were due to statistical variations in counting rates because of poor radioactive uptakes in damaged livers. In the remaining 3 cases, central defects were observed in the lateral views of the right hepatic lobe (Fig. 1), in the inferior and central portion of the left hepatic lobe (Fig. 2), and in the central portion of the left hepatic lobe (Fig. 3). Even retrospectively, these defects did not merely suggest normal variations. At surgery, however, no liver metastases were found.

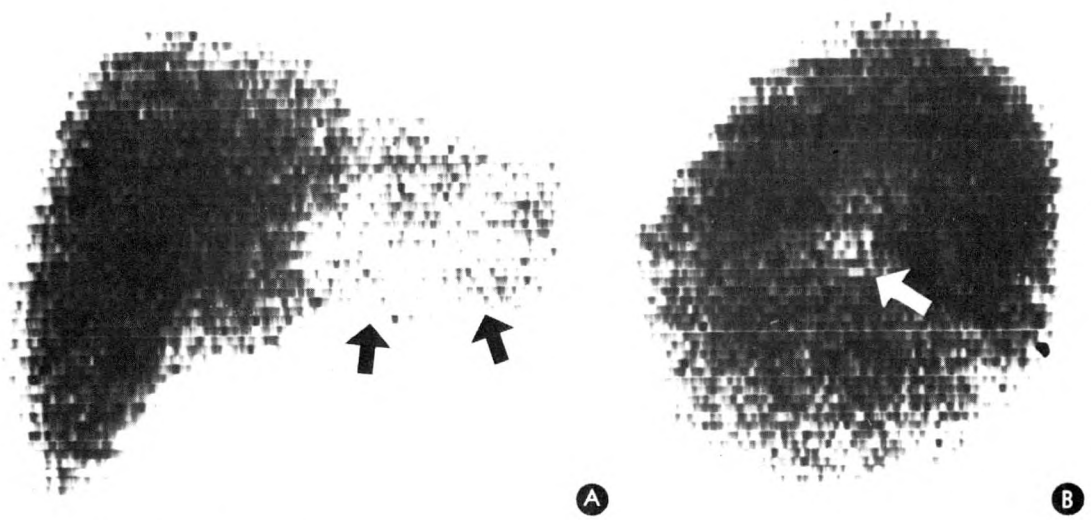


FIG. 1. (*A* and *B*) *False positive case.*

(*A*) In the anterior view, there is an area of decreased activity due to thinning of the left hepatic lobe. (*B*) In the right lateral view, there is a well-defined small defect in the center of the right lobe, suggesting hepatic metastasis. However, no liver metastasis was found at surgery.

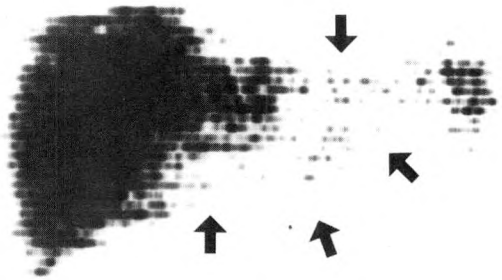


FIG. 2. *False positive case.*
In the anterior view, there is decreased activity in the left hepatic lobe and porta hepatis. This was suspected to be hepatic metastasis. However, at surgery no liver metastasis was observed.

SCINTIGRAPHICALLY DEMONSTRATED LIVER METASTASES IN HEPATOMEGALY

In hepatomegaly due to metastases, the foci are often relatively large, facilitating scintigraphic detection (Fig. 4 and 5). Without hepatomegaly, metastases are usually so small that they are detected only with difficulty. Since palpability is the simplest index of hepatomegaly, liver scintigraphy as a preoperative examination was evaluated in relation to palpation of the liver. The results are shown in Table v.

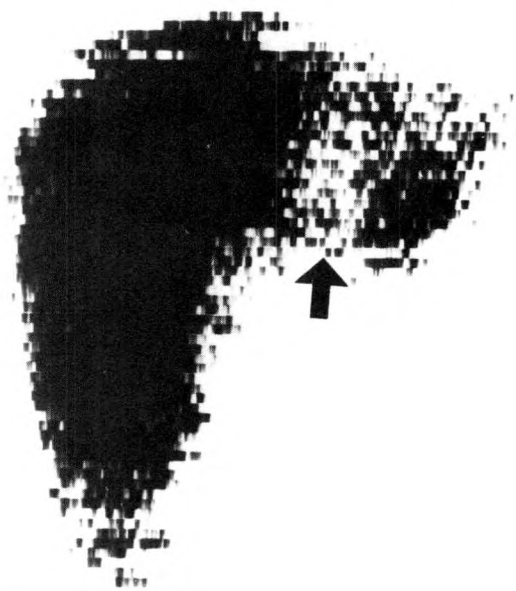


FIG. 3. *False positive case.*
In the anterior view, there is a well-defined defect in the central portion of the left hepatic lobe. Surgery revealed no abnormality of the liver.

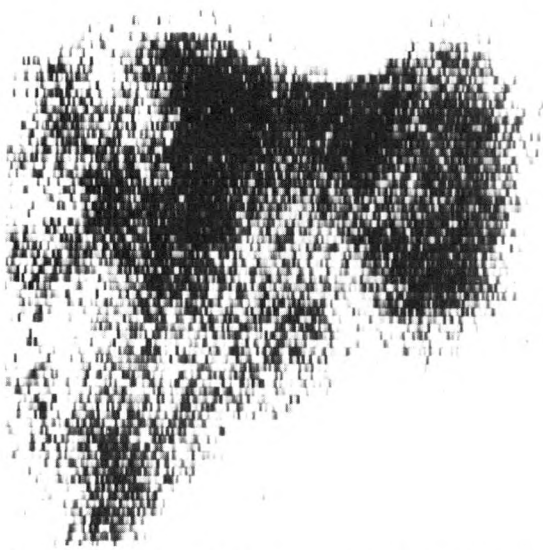


FIG. 4. *Hepatic metastases associated with hepatomegaly.*
The liver is enlarged with multiple areas of decreased activity in both lobes.

The liver was palpable in 50 of the 150 cases. In 28 (56 per cent) of these, metastases were confirmed. In these 28, the foci were scintigraphically detected in 25 (89 per cent). In the 100 with nonpalpable livers, metastases were confirmed in 16 (16 per cent). Among these 16, metastases were scintigraphically detected in 12 (75 per cent). Ninety per cent of the metastases in



FIG. 5. *Hepatic metastases associated with hepatomegaly.*
There are multiple clear-cut defects involving both lobes of the liver.

TABLE V

DETECTABILITY OF LIVER METASTASES SCINTIGRAPHICALLY EVALUATED IN RELATION TO HEPATOMEGALY

Palpability of Liver		Metastases	Metastases Detected Scintigraphically
Palpable	50 cases	28/50 cases (56%)	25/28 cases (89.2%)
Not palpable	100 cases	16/100 cases (16%)	12/16 cases (75.0%)

palpable livers were detected scintigraphically, as compared to a detection rate of 75 per cent among those not palpable. In all 3 cases with palpable livers and undetected metastases, the scintigraphically demonstrated defects were erroneously interpreted. In 4 cases with nonpalpable livers, whose metastases were undiagnosed, all foci were less than 2 cm. and undetectable scintigraphically. In 10 of the 44 with metastases, but nonpalpable livers and normal liver function studies, the preoperative detection of liver metastases by liver scintigraphy was of great value. Figures 6, 7 and 8 show the liver scintigrams of these cases.

DISCUSSION

There are numerous reports that the diagnostic accuracy of liver scintigraphy in detecting space-occupying lesions is about 80 per cent.[1,3,10,13,16,17]

We compared the surgical and preoperative liver scintigraphic findings in 150 cases diagnosed as progressive gastric carcinoma, and found a 90 per cent correlation as to the presence of liver metastases. According to Ozarda and Pickren[15] 11 per cent of liver

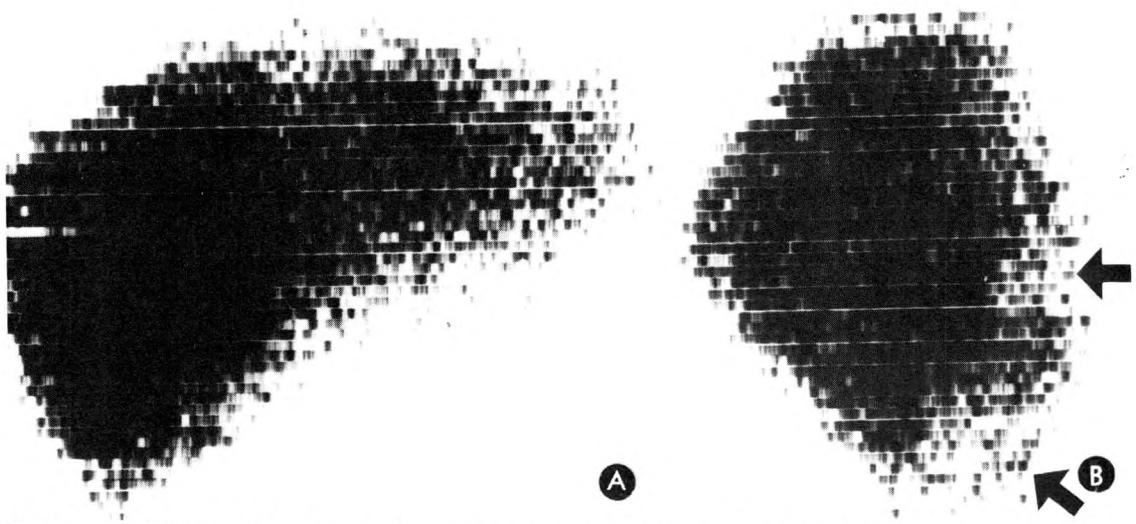


FIG. 6. (*A* and *B*) *Small hepatic metastases without hepatomegaly detected scintigraphically.*

(*A*) In the anterior view, the distribution of activity is uniform and appears normal. (*B*) In the right lateral view, there are 2 focal defects in the anterior portion of the right lobe. At surgery metastatic foci were found in this portion, as suggested by the scan.

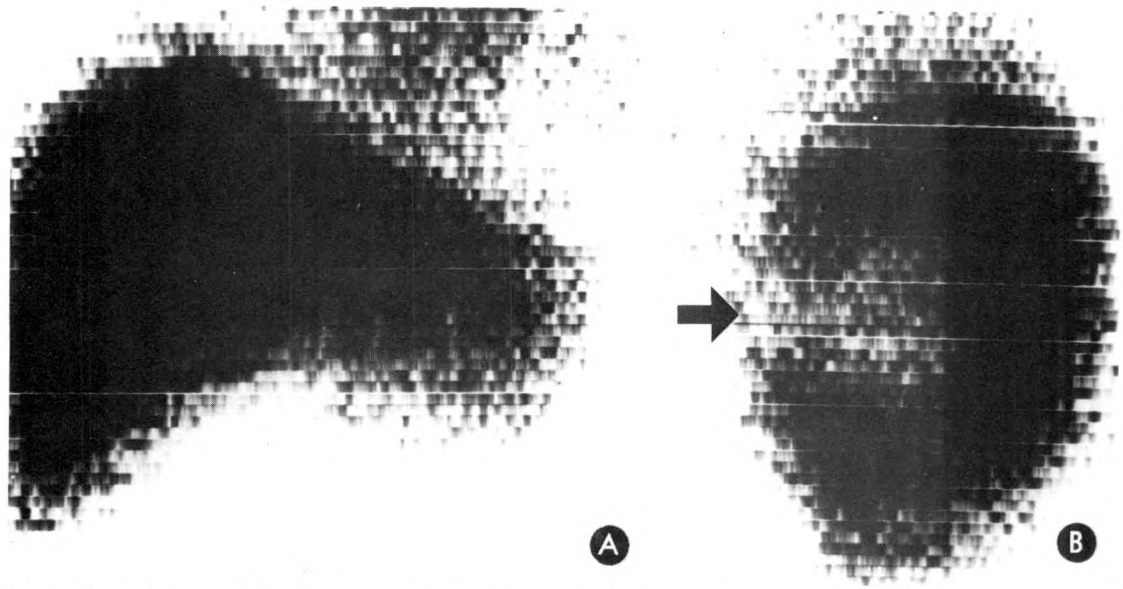


Fig. 7. (*A* and *B*) *Small hepatic metastasis without hepatomegaly detected scintigraphically.*
(*A*) In the anterior view, there is no evidence of metastasis. (*B*) In the right lateral view, there is a focal defect in the posterior portion of the right hepatic lobe. Surgery revealed a hepatic metastasis in the same portion.

metastases do not appear on the liver surface, so that liver metastases may be missed at laparotomy. Metastases less than 1 cm. in size and deep in liver tissue may be undetectable, not only scintigraphically, but also at surgery. Despite this, we found good correlation between scintigraphy and laparotomy when these were within 2 weeks of each other.

There were 7 (16 per cent) false negative cases: 4 with small metastatic foci undetectable scintigraphically; and 3 whose findings were erroneously interpreted. In the latter, more careful observation of the scintigrams might have facilitated detection. Forty (90 per cent) of the 44 cases had liver metastases large enough for scintigraphic detection. It is notable, however,

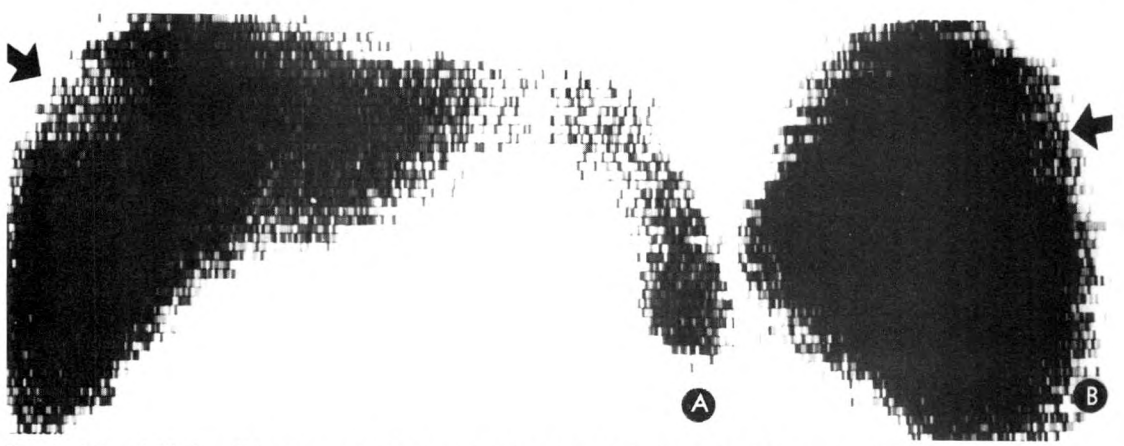


Fig. 8. (*A* and *B*) *Small hepatic metastasis without hepatomegaly detected scintigraphically.*
(*A*) In the anterior and (*B*) right lateral views there is a well-defined small defect in the anterosuperior portion of the right hepatic lobe. A hepatic metastasis 4 cm. in diameter was found in this portion at surgery.

that 10 per cent had scintigraphically undetectable foci. False negative detection rates for such lesions range from 5 to 20 per cent: Nagler *et al.*[13] reported 17 per cent; Smith and Williams[18] 9.1 per cent; Gutierrez *et al.*[8] 5.5 per cent; Ariel *et al.*[1] 5.1 per cent; Rossi and Gould[17] 19 per cent; and Jhingran *et al.*[10] 17 per cent; underscoring the limitations in the scintigraphic detection of liver metastases.

There were 7 (6.6 per cent) false positives in the present series. Low activity areas on liver scintigrams occur for various reasons. Covington[4] attributed false positives to the gallbladder fossa, pressure defects of tumors, the fossa for the inferior vena cava, the porta hepatis, midline vertebral pressure defects, thinning of the left hepatic lobe, and anatomic variations in hepatic size, shape, or position. Ironically, defects on liver scintigraphy due to other than space-occupying lesions have increased with technical improvements—further complicating the detection of small metastatic foci. Training in scintigraphic interpretation permits discrimination between space-occupying lesions and other defects, but in some cases such differentiation is impossible.

Liver metastases generally cause multiple defects on the scintigram. Localized central defects in the left or right hepatic lobe, even though isolated, strongly suggest space-occupying lesions, but when in the portal fissure or at the lower hepatic margin, diagnosis is often difficult. One aid in the differentiation is that metastases to these regions are often palpable. As for the 7 false positives, space-occupying lesions were excluded in 4 retrospectively, but in 3 some question remained. However, considering the location and size of the low activity area on the scintigrams as shown in Figures 1, 2 and 3, liver metastases should have been easily detectable at surgery if they were present. Therefore, these abnormal findings can be assumed to have been due to pressure by extrahepatic tumors. In fact, in all of the 3 cases, there was a large mass in the stomach and an extensive infiltration of the regional lymph nodes.

Extensive liver metastases are often accompanied by hepatomegaly. Ozarda and Pickren[15] reported hepatomegaly at autopsy in 70 per cent of their series with liver metastases, while Poulose *et al.*[16] noted it in 46 per cent of their series. Twenty-eight (64 per cent) of the 44 cases in the present study of liver metastases had palpable livers. In hepatic metastases associated with hepatomegaly, scintigraphic detection is easy because of the sufficient size of the metastatic foci. The scintigraphic detection rate of liver metastases in fact approached 89 per cent. Hepatomegaly in patients with gastric cancer in the progressive stage may be due to other than metastases, and liver scintigraphy is especially valuable in determining whether metastases or other causes are responsible.

However, liver metastases without hepatomegaly occurred in 16 out of 100 cases. Suspicion of metastases was not restricted to cases with palpable livers. Theoretically, preoperative liver scintigraphy would have much more value in detecting early metastases before the liver becomes palpable. Our detection rate for such cases was 75 per cent, in 12 of 16 cases. All 4 cases with scintigraphically undetected metastases in normal liver size had small metastatic foci less than 2 cm. in diameter.

Among 150 cases diagnosed as progressive gastric carcinoma and 4 of 44 with liver metastases, only 4 had metastases considered undetectable, indicating the great value of preoperative liver scintigraphy in progressive gastric carcinoma.

SUMMARY

The surgical and liver scintigraphic findings were compared for evaluation of preoperative diagnostic value in 150 cases of gastric carcinoma with extension to the muscularis and serosa. There was correlation in 90 per cent of the cases, with a false negative rate of 16 per cent, and a false positive rate of 6.6 per cent.

Liver metastases were detected in 89 per cent, when the liver was palpable, com-

pared to 75 per cent in those with nonpalpable livers. Four of the 150 cases had small scintigraphically undetectable metastatic foci. Although search for liver metastases at laparotomy may be inconclusive in confirming the presence of metastases, our scintigraphic detection rate correlated closely with the surgical findings. Preoperative liver scintigraphy is therefore considered to be of great diagnostic value.

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REFERENCES

1. Ariel, I. M., Molander, D., and Galey, D. Hepatic gammascanning: aid in determining treatment policies for cancer involving liver. *Am. J. Surg.*, 1969, *118*, 5–14.
2. Cameron, G. R. Liver as site and source of cancer. *Brit. M. J.*, 1954, *1*, 347–352.
3. Covington, E. E. Accuracy of liver photoscans. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 742–744.
4. Covington, E. E. Pitfalls in liver photoscans. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 745–748.
5. Einhorn, J., and Lundell, G. Detection of space occupying in liver. *Acta radiol.*, 1962, *57*, 366–372.
6. Freeman, L. M., Meng, C. H., Johnson, P. M., Bernstein, R. G., and Bosniak, M. A. False positive liver scans caused by disease processes in adjacent organs and structures. *Brit. J. Radiol.*, 1969, *42*, 651–656.
7. Friedell, H. L., MacIntyre, W. J., and Rejali, A. M. Method for visualization of configuration and structure of liver. Part A. Preliminary clinical investigation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *77*, 455–470.
8. Gutierrez, R. R., Bacon, H. F., and Gennaro, A. R. Value of liver scan in management of cancer of large bowel. *Dis. Colon & Rectum*, 1968, *11*, 61–68.
9. Hayashida, T., and Kidokoro, T. End results of early gastric cancer collected from 22 institutions (Japanese). *Stomach & Intestine*, 1969, *4*, 1077–1085.
10. Jhingran, S. C., Jordan, L., Jahns, M. F., and Haynie, T. P. Liver scintigrams compared with alkaline phosphatase and BSP determinations in detection of metastatic carcinoma. *J. Nuclear Med.*, 1971, *12*, 227–230.
11. Johnson, P. M., and Sweeney, W. A. False-positive hepatic scan. *J. Nuclear Med.*, 1967, *8*, 451–460.
12. Kitaoka, K. Metastasis of Cancer (Japanese.) Edited by H. Ishikawa. Nakayama Company, Tokyo, 1972, pp. 109–146.
13. Nagler, W., Bender, M. A., and Blau, M. Radioisotope photoscanning of liver. *Gastroenterology*, 1963, *44*, 36–43.
14. Nishi, M., Nanasawa, H., Seki, M., Hirose, M., and Nakashima, S. On five-year survival rate of stomach cancer—with special reference to advanced cancer (Japanese). *Stomach & Intestine*, 1969, *4*, 1087–1100.
15. Ozarda, A., and Pickren, J. Topographic distribution of liver metastases: its relation to surgical and isotope diagnosis. *J. Nuclear Med.*, 1962, *3*, 149–152.
16. Poulose, K. P., Reba, R. C., DeLand, F. H., and Wagner, H. N. Role of liver scanning in preoperative evaluation of patients with cancer. *Brit. M. J.*, 1969, *6*, 585–587.
17. Rossi, P., and Gould, H. R. Angiography and scanning in liver disease. *Radiology*, 1970, *96*, 553–562.
18. Smith, L. B., and Williams, R. D. Relative diagnostic accuracy of liver radioactive isotope photoscanning. *A.M.A. Arch. Surg.*, 1968, *96*, 693–697.
19. Stout, A. P. Pathology of carcinoma of stomach. *Arch. Surg.*, 1943, *46*, 807–822.
20. Wagner, H. N., Jr., McAfee, J. G., and Mozley, J. M. Diagnosis of liver disease by radioisotope scanning. *A.M.A. Arch. Int. Med.*, 1961, *107*, 324–328.

THE USE OF MULTIPLE RADIONUCLIDE IMAGING TO DIFFERENTIATE THE FOCAL INTRAHEPATIC LESION*

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CONVENTIONAL imaging of the liver with radiocolloids is acceptably sensitive, but notably nonspecific, in diagnosing focal intrahepatic disease. A negative defect can result from primary or metastatic tumor, abscess, cyst, hemangioma, or advanced cirrhosis.[13] Recent reports suggest that evaluation of a defect detected by the conventional colloid study can be aided by additional imaging with tracers having different metabolic and chemical properties.[1,5,9,11,12,16,18]

An important diagnostic characteristic of an intrahepatic mass lesion is its vascularity relative to that of surrounding normal liver tissue. Blood pool scanning, used in conjunction with the conventional colloid scan, has proven helpful in assessing the vascularity of a lesion as compared with normal hepatic sinusoidal blood flow. The "vascular" intrahepatic lesions include: hepatoma, hamartoma, aneurysm, adenoma, hemangioma and a minority of metastatic lesions. The "avascular" or "hypovascular" lesions include: cyst, abscess, infarction, the cirrhotic pseudomass, and most metastases.

$In^{113m}Cl_3$ at pH 1.7 binds to transferrin *in vivo*, and thus is an excellent agent for blood pool scanning. Its photon energy of 390 kev. is easily discriminated from Tc^{99m}-sulfur colloid by spectrometry, and, therefore, blood pool scanning can immediately follow the colloid study.

Another diagnostic characteristic is the ability of a lesion to incorporate Se^{75}-selenomethionine. The uptake of this agent is related to protein synthesis. It has also been postulated that there is correlation between organ blood flow and Se^{75}-selenomethionine uptake; however, this relationship is not constant.[9]

Dual radionuclide scanning, long utilized in the evaluation of renal lesions,[2,15] is now finding increasing use in the diagnosis of the focal intrahepatic lesion.[1,5,11,12,18]

It is the purpose of this paper to present our experience with 35 patients having hepatic mass lesions studied with 2 or more radiopharmaceuticals and to propose a systematic approach to evaluate intrahepatic lesions.

MATERIAL AND METHOD

All patients had large focal intrahepatic defects detected on the conventional colloid liver scan using 3 mc Tc^{99m}-sulfur colloid. Each patient underwent blood pool scanning with 3–5 mc $In^{113m}Cl_3$ at pH 1.7, usually performed without delay. Six patients also received 300 μc Se^{75}-selenomethionine and were scanned at 2 and 24 hours post injection. The scans were evaluated according to criteria described below. All studies were performed using a Nuclear-Chicago Pho/Gamma HP camera with appropriate collimation. Biopsy specimens and angiographic studies were correlated with the results of the scans.

We are currently utilizing the radionuclide angiogram in the evaluation of patients. After a bolus injection of 10 mc $Tc^{99m}O_4^-$, a 1 minute continual videotape data recording is made for later analysis. As will be discussed later, this method allows relative arterial and portal venous blood flow to be assessed.

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P.Q., Canada, September 25–28, 1973.
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RESULTS

Of the 35 patients studied, there were 18 with hepatic metastases, 9 with hepatoma, 4 with abscess or cyst, 3 with hemangioma, and 1 with a hamartoma. On the basis of the colloid and blood pool scans, the lesions were classified as follows:

Category 1: Avascular or relatively hypovascular.

Category 2: Vascularity approaching or equal to that of normal liver.

Category 3: More vascular than normal liver.

Utilizing the above diagnostic criteria, our cases were classified in the manner demonstrated in Table 1.

Six patients with Category 2 lesions were scanned with Se^{75}-selenomethionine. Four patients with hepatoma showed uptake approaching or equal to that of normal liver, while 2 patients with metastases had little or no uptake of the tracer.

DISCUSSION

Recently, Lubin and Lewitus[12] categorized intrahepatic lesions on the basis of their blood pool as follows:

Category 1: No blood pool activity.

Category 2: Blood pool activity less than normal liver.

Category 3: Blood pool activity equal to normal liver.

Category 4: Blood pool activity greater than normal liver.

This classification presents some definite problems. In the first place, overlying normal liver tissue makes it difficult to decide whether or not a study falls into Categories 1 or 2. Secondly, there is a heterogeneity of blood activity in several large lesions so that some of these lesions show both Category 2 and Category 3 activity. Finally, with the exception of a hemangioma (Category 4), no category proved to be diagnostic of any lesion. Yeh and associates[18] described hepatic perfusion as being either good, poor, or absent. Using this classification, one encounters the same difficulties as described in the classification of Lubin and Lewitus.[12]

TABLE I

CLASSIFICATION OF 35 PATIENTS WITH INTRAHEPATIC LESIONS

Diagnosis	Category		
	1	2	3
Cyst or abscess	4	—	—
Metastases	16	2	—
Hepatoma	1*	8	—
Hamartoma	—	1	—
Hemangioma	—	—	3

* Lesion 95 per cent necrotic and hemorrhagic.

Our simplified classification has greatly reduced difficulties with category placement. More importantly, it provides a more thorough basis for diagnostic accuracy, since each initial category leads to a further diagnostic step. For example, Category 1 lesions are comprised of cysts, abscesses, or metastases (Fig. 1, *A–C*). We have found that when diagnosis is in doubt, B-mode ultrasound provides a means for differentiation. Abscesses and cysts have proven to be relatively transsonic, while metastases have appeared as "solid" lesions.

Category 2 lesions are usually hepatomas (Fig. 2, *A–E*), although we found 2 metastases and 1 hamartoma that exhibited considerable blood pool activity. It is for these patients that we propose Se^{75}-selenomethionine scanning. Except for melanoma, most metastatic tumors have markedly reduced or absent uptake of Se^{75}-selenomethionine. Hepatomas, on the other hand, usually take up the tracer in concentrations approaching or equaling that of normal liver.[3,9]

Category 3 lesions are classically hemangiomas (Fig. 3, *A–C*). To date, there have been no reports of hepatomas showing greater activity on blood pool scanning than normal adjacent liver tissue. Thus, by utilizing 3 radiopharmaceuticals and B-mode ultrasound, one can, with reasonable certainty, differentiate cysts and abscesses from solid tumors, most metastases from hepatomas, and hemangiomas from all other intrahepatic mass lesions.

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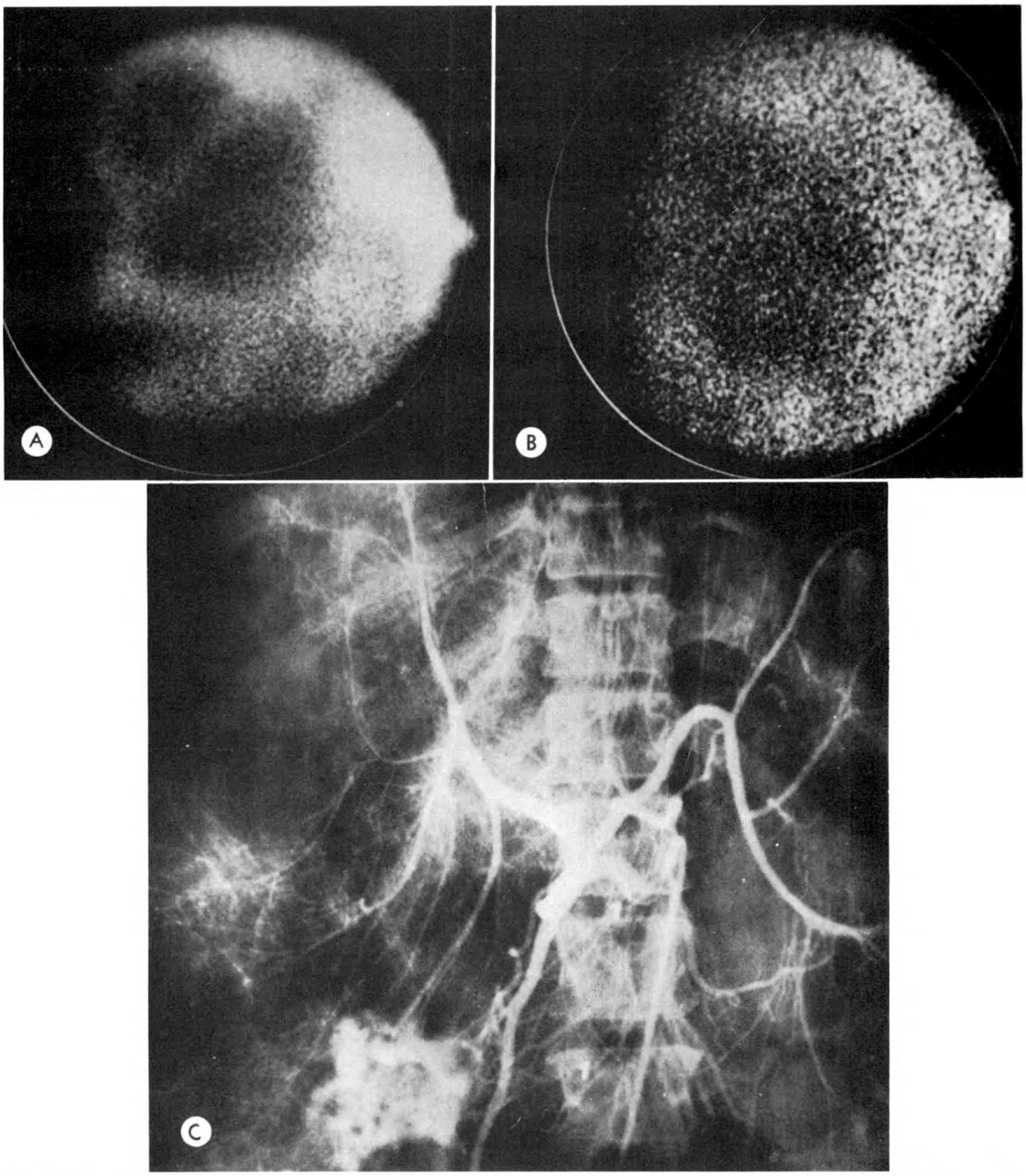


FIG. 1. *Category 1 Lesion.* (*A*) Tc^{99m}-sulfur colloid scan shows large intrahepatic defects in this patient with metastatic disease. (*B*) In^{113m} blood pool scan shows the hypovascular nature of these lesions. (*C*) Celiac angiography confirms large areas of relative hypovascularity.

To better understand the pathophysiology of the lesions studied by scanning, we correlated our findings with angiographic and histologic data. There was excellent correlation between the results of the blood pool scans and the actual vascularity as seen on angiography and tissue section.

For example, a hemangioma, because of its cavernous vascularity, exhibits more blood pool activity than adjacent normal liver. A hepatoma, on the other hand, may be exceedingly vascular, yet there are usually avascular areas of necrosis or hemorrhage within the tumor accounting

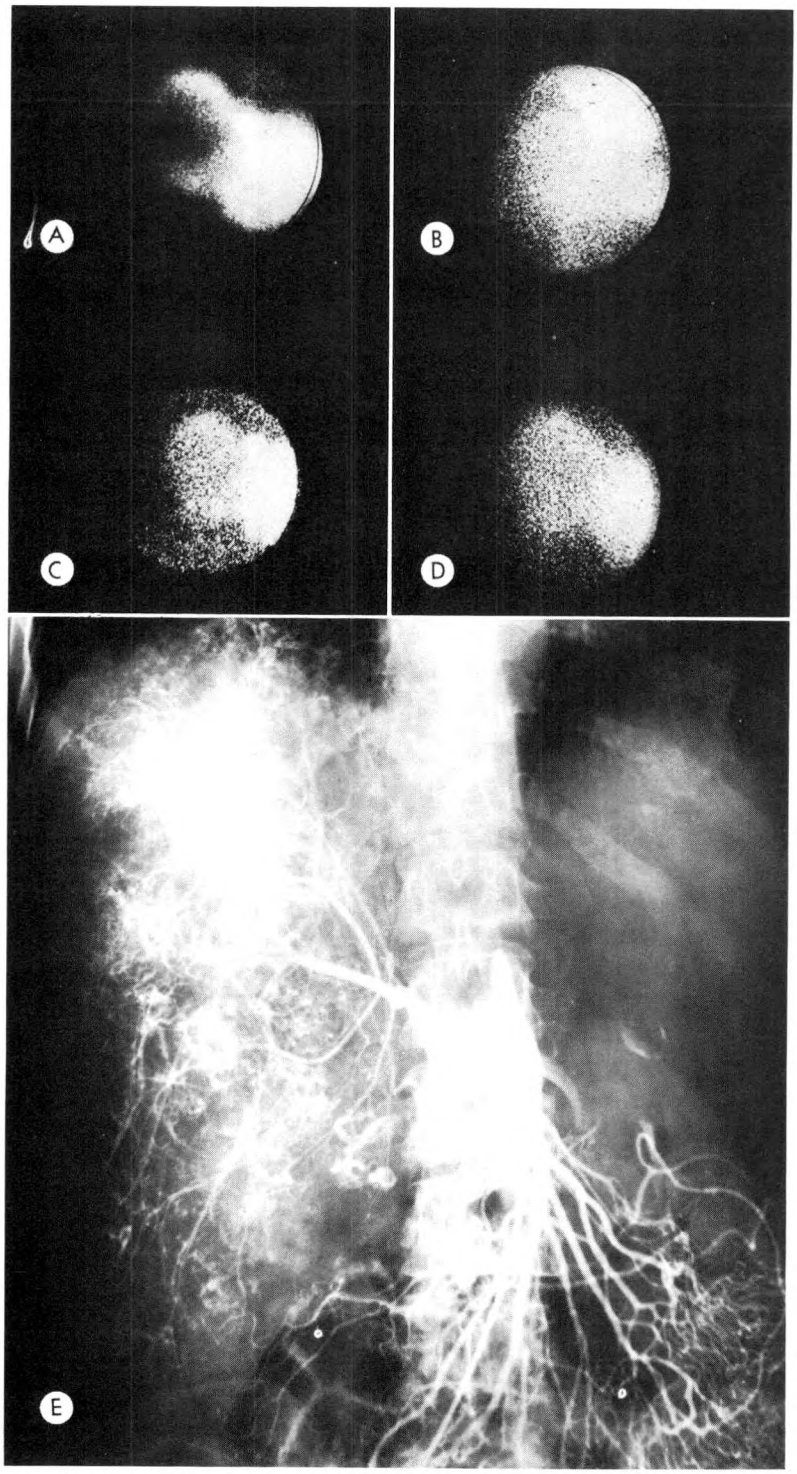


Fig. 2. *Category 2 Lesion.* (*A*) Tc^{99m}-sulfur colloid scan shows a large intrahepatic defect in this patient with hepatoma. (*B*) In^{113m} blood pool scan shows the lesion to have vascularity comparable to normal liver. Se^{75}-selenomethionine scans at (*C*) 2 hours and (*D*) 24 hours demonstrate marked uptake in the lesions. (*E*) Superior mesenteric angiography demonstrates a replaced right hepatic artery supplying a richly vascularized hepatoma.

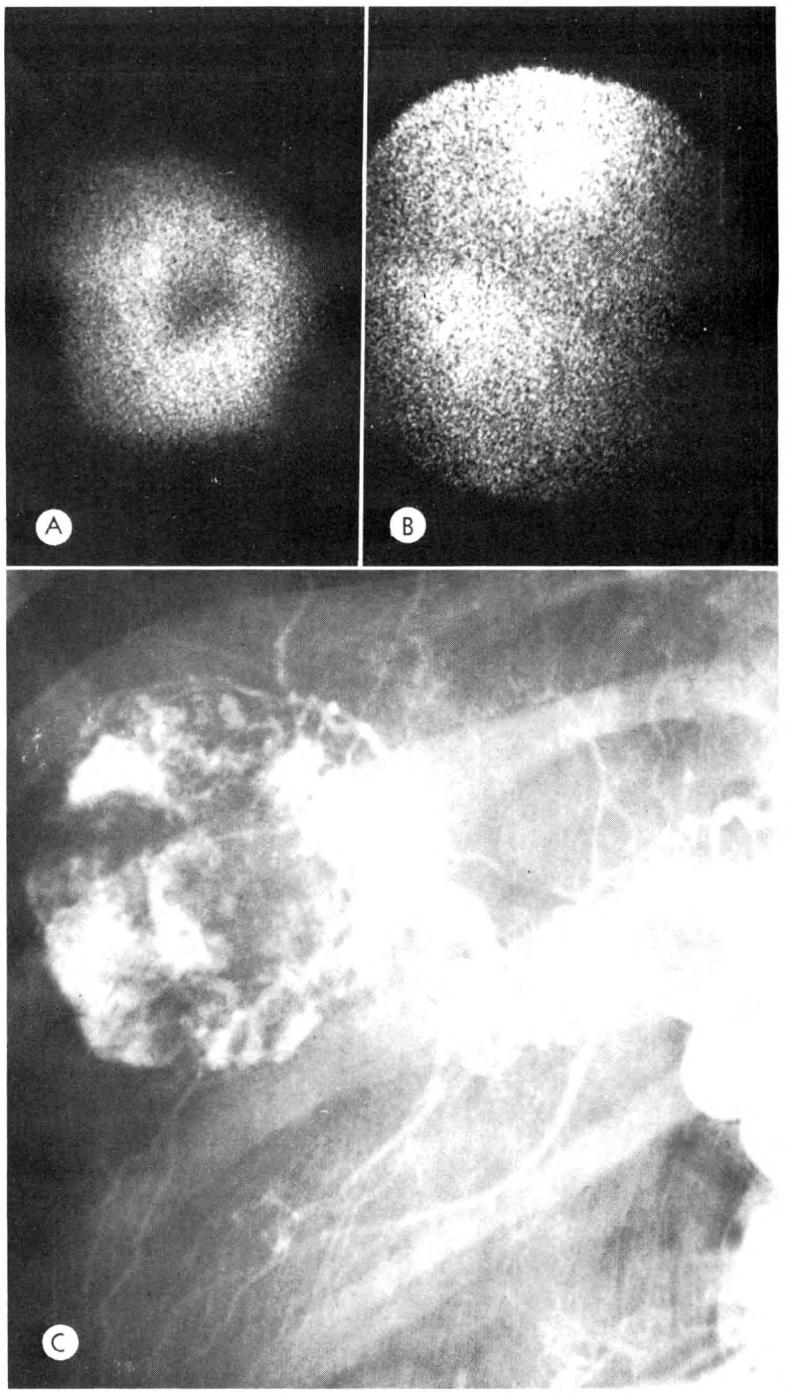


FIG. 3. *Category 3 Lesion.* (*A*) Tc^{99m}-sulfur colloid scan shows a large intrahepatic defect in this patient with a hemangioma. (*B*) In^{113m} blood pool scan shows the lesion to be more vascular than surrounding normal liver. (*C*) Celiac angiography confirms the large cavernous hemangioma.

for the heterogeneity of the blood pool often seen with hepatomas.

In an attempt to increase the specificity of preoperative diagnosis, newer procedures to evaluate the intrahepatic mass lesion will be utilized with greater frequency. Tumor-localizing agents such as Ga^{67} citrate, $In^{111}Cl_3$, and In^{111}-bleomycin have aided in this evaluation.[4,6–8,10,14,17] However, experience with these agents is limited, and preliminary reports are often contradictory.

We believe that dynamic radionuclide angiography will prove valuable in assessing the contribution of arterial and venous blood flow to the liver, as well as the relative vascularity of intrahepatic mass lesions (Fig. 4, *A–F*; and 5, *A* and *B*). Most tumors are supplied by the hepatic artery and show prompt visualization during the arterial phase of the study. Normal liver, which derives up to 80 per cent of its blood supply from the portal vein, is not well visualized until the venous phase. Thus, dynamic radionuclide angiography provides additional information not obtained with the $In^{113m}Cl_3$ blood pool scan.

CONCLUSION AND SUMMARY

Thirty-five patients with various focal intrahepatic lesions were evaluated with multiple radionuclides.

Comparison of the blood pool scans with angiography and histology demonstrated an excellent correlation of these studies.

Diagnostic accuracy was improved by separating the blood pool scans into 3 categories. Each category suggested, at most, one further study to provide specific diagnostic information. Thus, by utilizing 3 radiopharmaceuticals, one can be more specific in preoperative diagnoses.

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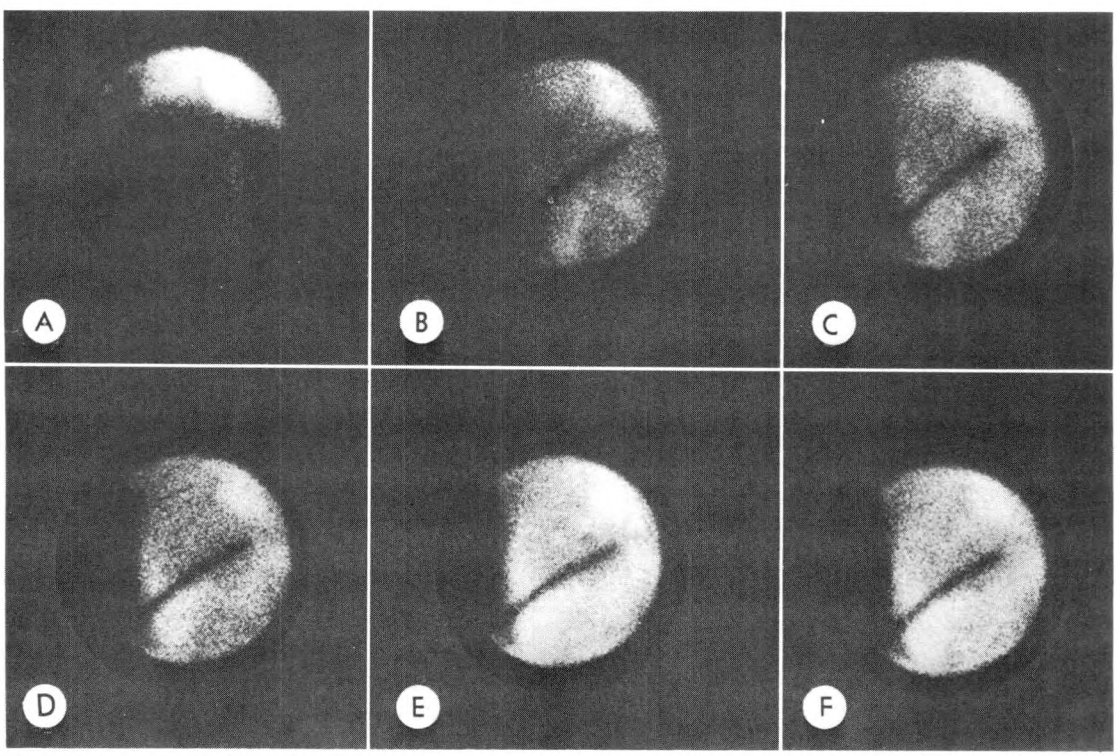


Fig. 4. *Dynamic Radionuclide Angiogram.* Following a 10 mc $Tc^{99m}O_4^-$-intravenous bolus injection, a continuous videotape data recording was made. Serial scintiphotographs obtained at (*A*) 0–11 sec.; (*B*) 11–18 sec.; (*C*) 18–27 sec.; (*D*) 27–36 sec.; (*E*) 36–54 sec.; and (*F*) 54–69 sec. confirm the large avascular areas in this extensively necrotic hepatoma.

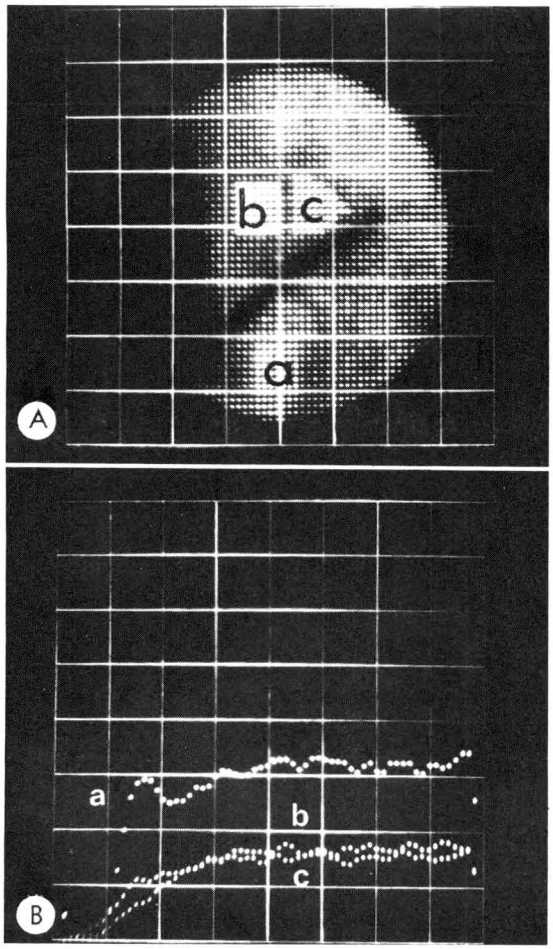


FIG. 5. *Dynamic Radionuclide Angiogram.* (*A*) Equal areas of interest can be selected for study of relative vascularity. Areas *b* and *c* were located within the tumor; area *a* is normal liver tissue. (*B*) A graphic representation of the selected areas demonstrates poor vascularity of the tumor as compared with normal liver. Surgical exploration confirmed extensive necrosis in this hepatoma.

REFERENCES

1. ANDERSON, J. E., and PERLMUTTER, G. S. Diagnosis of hepatoma using multiple radionuclides. *Radiology*, 1972, *102*, 387–389.
2. BLACK, M. B., KING, C. D., and SMITH, D. R. Double isotope scintiphotography for differentiating between renal cysts and renal tumors. *J. Urol.*, 1967, *98*, 728–734.
3. EDDLESTON, A. L. W. F., RAKE, M. O., PAGALTSOS, A. P., OSBORN, S. B., and WILLIAMS, R. ^{75}Se-selenomethionine in scintiscan diagnosis of primary hepatocellular carcinoma. *Gut*, 1971, *12*, 245–249.
4. EMMERSON, D. A., O'MARA, R. E., and LILIEN, D. L. ^{111}Indium-bleomycin as tumor imaging agent. *J. Nuclear Med.*, 1973, *14*, 625 (abstract).
5. FREEMAN, L. M., BERNSTEIN, R. G., and HOYT, D. B. Diagnosis of hepatic hemangioma with combined scanning technique. *Radiology*, 1970, *95*, 127–128.
6. GOODWIN, D. A., LIN, M. D., DIAMANTI, C. I., GOODE, R. L., and MEARES, C. F. ^{111}In-labelled bleomycin for tumor localization by scintiscanning. *J. Nuclear Med.*, 1973, *13*, 401 (abstract).
7. GROVE, R. B., ECKELMAN, W. C., and REBA, R. C. Preparation, distribution, and tumor imaging properties of ^{111}In, ^{57}Co, ^{67}Ga, and ^{59}Fe labeled bleomycin. *J. Nuclear Med.*, 1973, *14*, 627 (abstract).
8. GROVE, R. B., MADEWELL, J. E., RAPP, G. S., PINCKY, S. M., and JOHNSON, M. C. Practical application of ^{67}Ga-citrate to evaluation of liver pathology. *J. Nuclear Med.*, 1973, *14*, 402 (abstract).
9. KAPLIN, E., and DOMINGO, M. ^{75}Se-selenomethionine in hepatic focal lesions. *Sem. Nucl. Med.*, 1972, *2*, 139–149.
10. LOMAS, F., DIBOS, P. E., and WAGNER, H. N. Increased specificity of liver scanning with use of ^{67}gallium citrate. *New England J. Med.*, 1972, *286*, 1323–1329.
11. LUBIN, E., LAOR, J., SHIMEONI, A., LEWITUS, Z., PICK, A. I., and KADISH, V. Two stage scintiscanning in differential diagnosis of vascularized and nonvascularized space-occupying lesions. *Nuc. Medizin*, 1970, *9*, 17–34.
12. LUBIN, E., and LEWITUS, Z. Blood pool scanning in investigating hepatic mass lesions. *Sem. Nucl. Med.*, 1972, *2*, 128–132.
13. MCCREADY, V. R. Scintigraphic studies of space-occupying liver disease. *Sem. Nucl. Med.*, 1972, *2*, 108–127.
14. ROBBINS, P. J., SILBERSTEIN, E. B., and FORTMAN, D. L. Preparation and distribution of ^{111}In-bleomycin in mice as compared to $^{111}InCl_3$ for tumor scanning. *J. Nuclear Med.*, 1973, *14*, 637 (abstract).
15. ROSENTHAL, L., and REID, E. Radionuclide distinction of vascular and nonvascular lesions of kidney. *Canad. M. A. J.*, 1968, *98*, 1165–1170.
16. STOLZEBERG, J. Uptake of ^{75}Se-selenomethionine by hepatoma. *J. Nuclear Med.*, 1972, *13*, 565–566.
17. VERMA, R. C., BENNETT, L. R., TONYA, J. J., MORTON, D. L., and WITT, E. ^{111}Indium-bleomycin: new radiopharmaceutical for tumor scintigraphy. *J. Nuclear Med.*, 1973, *14*, 641 (abstract).
18. YEH, S-H., SHIH, W-J., and LIEUNG, J-C. Intravenous radionuclide hepatography in differential diagnosis of intrahepatic mass lesions. *J. Nuclear Med.*, 1973, *14*, 565–567.

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THE VALUE OF Tc^{99m}-DIPHOSPHONATE (HEDSPA) AS A SKELETAL SCANNING AGENT*

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BONE metastatic lesions present numerous difficulties, due to occasional inability of the conventional roentgenographic methods to establish the diagnosis.

Synthesis and both experimental[1,2] and clinical application[5] of the disodium salt of phosphonate (HEDSPA) labeled with Tc^{99m} seem to open new horizons in the era of bone scanning. The compound used is quite innocuous in bone formation,[4] and more stable compared to inorganic polyphosphates, while its ability to be labeled with Tc^{99m} provides the physician with a useful radiopharmaceutical for clinical application entailing a low radiation dose administration.[1] Calculations on the dose delivered by Tc^{99m}-HEDSPA compare favorably with those of other agents;[1] *i.e.*, Sr^{87m}, Sr^{85m} and F^{18}.

MATERIAL AND METHOD

Experiments were carried out in 15 rabbits, a dose of 400 μc being injected in the marginal ear vein. The experimental animals were divided into 5 groups (3 rabbits each); were sacrificed at 1, 2, 6, 12, and 18 hours; and the radiation ratio of the skeletons to the remaining organs was calculated (the concentration in the urine is not included).

Clinically, the skeletons of 21 patients with known or suspected bone metastatic lesions were imaged with a scintillation camera,* after the intravenous administration of Tc^{99m}-diphosphonate in doses of 12 mc, which contained 0.5 mg. of carrier diphosphonate. Multiple posterior and anterior Polaroid photographs were obtained, when necessary.

* PHO-Gamma III, Nuclear Chicago, U.S.A.

RESULTS

The images obtained in all cases were of a very satisfactory quality. Referring to the best timing for scanning due to the rapid background disappearance of this agent, photoscans can be obtained after 30 seconds. However, the optimum time ranges between 12 and 18 hours, as indicated in our experimental study, which showed a gradually improving target to nontarget ratio (Table I), based on the time elapsed, up to 18 hours. Eighteen of our patients had had roentgenographic evidence of bone metastatic lesions from known primary tumors (Table II). In 3 patients, treated for carcinomas of the breast, kidney and the colon, metastatic lesions of the vertebral

TABLE I

RATIO OF RADIATION OF TARGET (SKELETON) TO NONTARGET (ORGANS AND SOFT TISSUES), ACCORDING TO THE TIME ELAPSED FROM INTRAVENOUS ADMINISTRATION OF THE RADIOPHARMACEUTICAL (EXCRETION IN THE URINE IS NOT INCLUDED)

Time (hr.)	1	2	6	12	18
Ratio	3/1	4.5/1	6/1	19/1	17/1

TABLE II

SITE OF THE PRIMARY LESION

Primary Lesion	No. of Cases
Carcinoma of the gastrointestinal tract	10
Carcinoma of the breast	7
Carcinoma of the lung	2
Carcinoma of the kidney	1
Carcinoma of the prostate	1
Total	21

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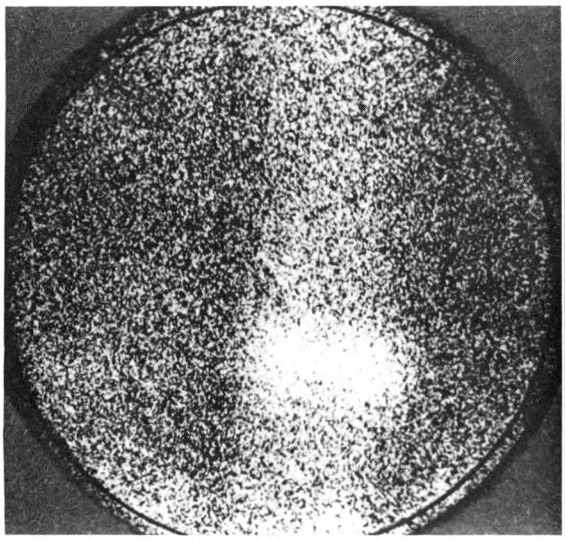


Fig. 1. Concentration of the radioisotopic substance in the 4th lumbar vertebra, indicating a metastatic lesion.

column, left femur and left scapula, respectively, were found although no definite clinical or roentgenographic findings were present. The radioisotopic findings were confirmed by a drill biopsy or at autopsy.

Figure 1 shows a metastatic lesion at the level of the 4th lumbar vertebra in a patient with breast carcinoma, operated upon 1 year previously, whose roentgenogram was normal (Fig. 2).

Figure 3 shows a metastatic lesion of the left femur, in a patient with carcinoma of the colon, with no evidence of bone lesions on the roentgenographic examination (Fig. 4).

In Figure 5 accumulation of the radioisotopic substance in the left scapula, in a patient with carcinoma of his right kidney, proved to be metastatic from the primary lesion. The roentgenographic examination had shown no bone destruction (Fig. 6).

DISCUSSION

Due to the not infrequent unsatisfactory images obtained by means of various bone scanning agents, *i.e.*, Sr^{87m}, Sr^{85m}, and F^{18}, and the relatively high radiation dose administered to the patient when given[1] "bone scanning" compounds have been tried, in-

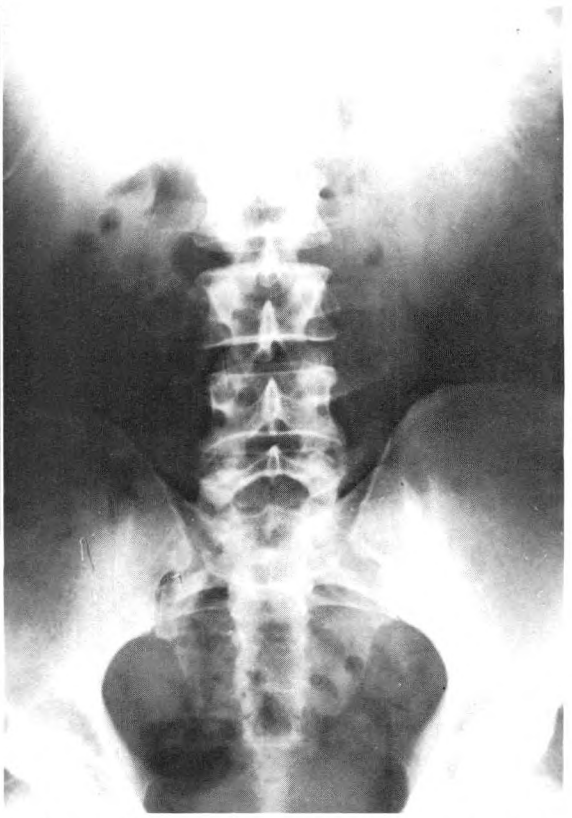


Fig. 2. Plain roentgenogram of the same patient shows no abnormalities.

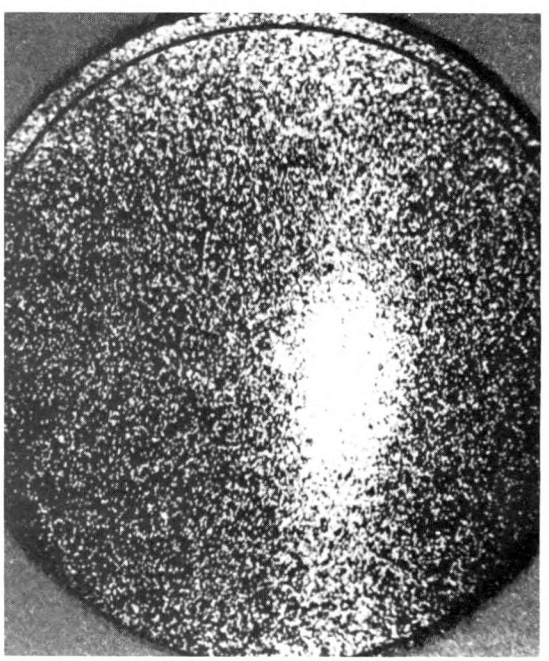


Fig. 3. Metastatic lesion of the left femur, as indicated by the gamma-camera scanning.

organic polyphosphates have been used and very satisfactory bone scannings were obtained.[6] However, because of their chemical instability[3] a new organic and more stable compound, namely diphosphonate, was introduced.[1]

Lethality studies, blood clearance and tissue dose calculations have been carried out and the labeled compound proved to be safe and relatively innocuous.

As a result of the rapid blood clearance of this radiopharmaceutical, which is mainly eliminated by the kidney, the ratio of radiation concentrated in the target organ, *i.e.*, the skeleton, to nontarget organs is quite high, even 1 hour after the

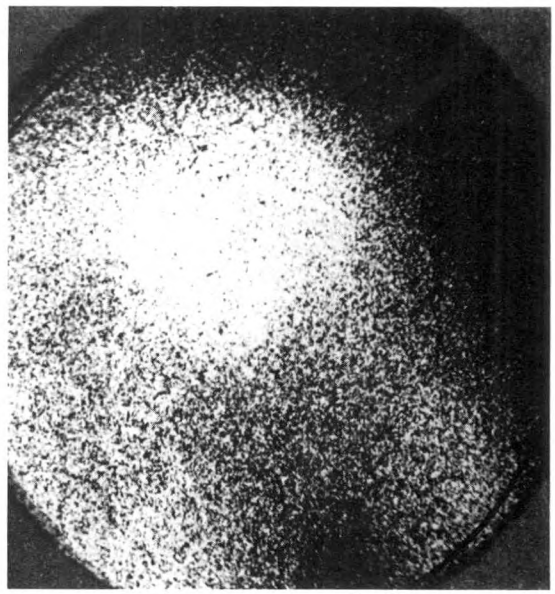


Fig. 5. Accumulation of the radioisotopic substance in the left scapula (deposit from pre-existing carcinoma of the kidney).



Fig. 4. No evidence of a bone lesion in the roentgenographic examination.

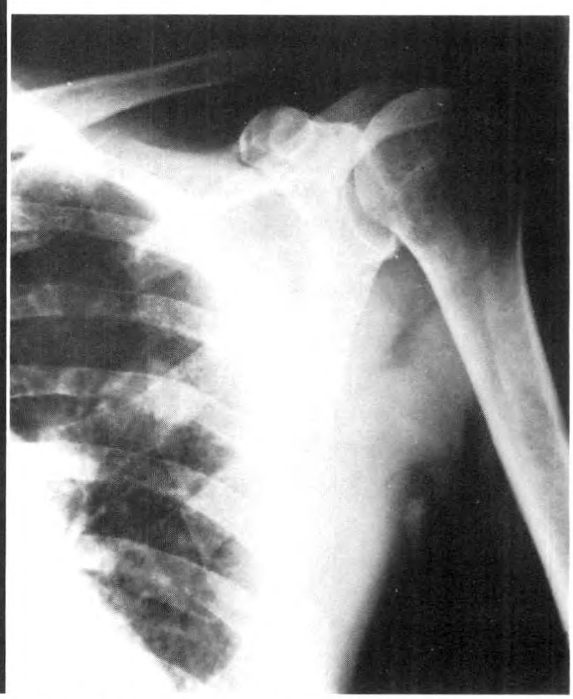


Fig. 6. Roentgenographic examination of the left scapula shows no bone destruction.

intravenous administration of the compound. The optimum time, however, is after 12 hours (Table 1), when the ratio ranges between 19:1 and 17:1.

Patterns of normal and abnormal examples of bone scanning after using the new agent have been reported in the literature.[5] Wide experience, however, with the clinical use of Tc^{99m}-diphosphonate is not available because it is quite new.[1]

We present the results following the application of this method to emphasize:

(a) The safety in using this organic diphosphonate, which is very stable and when labeled with Tc^{99m} delivers an acceptable low dose of radiation.

(b) The accuracy in the results obtained, as the images are superior compared to those of other bone scanning agents. This results in earlier discovery of metastatic bone lesions and an early institution of the appropriate treatment.

(c) The necessity of submitting every patient with potential neoplastic bone lesions to skeletal scanning by means of Tc^{99m}-diphosphonate.

(d) The absence of known disadvantages of this compound, in the dose suggested.

(e) The optimum time of obtaining scannings which ranges between 12 and 18 hours following its administration. Taking into account the aforesaid advantages of Tc^{99m}-diphosphonate, we do recommend its use as a screening test in patients suspected of having neoplastic bone lesions.

SUMMARY

A new "bone seeking" agent, namely the organic compound diphosphonate labeled with Tc^{99m} was tried in 21 patients for skeletal scanning. In 3 cases, although the clinical and roentgenographic evidence of bone lesions was absent, bone metastases were found, and these were confirmed histologically.

The organic diphosphonate is more stable compared to the inorganic polyphosphates and the images obtained are excellent.

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REFERENCES

1. Castronovo, F., and Callahan, R. New bone scanning agent ^{99m}Tc labeled 1-hydroxy-ethylidene-1, 1-disodium phosphonate. *J. Nuclear Med.*, 1972, *13*, 823–827.
2. Castronovo, P. F., Callahan, R., Potsaid, M. S., Pentegrasse, H. P., and Neel, H. S. New Tc^{99m} skeletal imaging radiopharmaceutical: 1-hydroxy-ethylidene-1, 1-disodium phosphonate Tc^{99m} complex. IAEA Symposium of new developments in radiopharmaceuticals and labeled compounds, Copenhagen, 1973, *171*, S. M., 1–23.
3. Francis, M. D., Russel, R. G., and Fleish, H. Diphosphonates inhibit formation of calcium phosphates crystals in vitro and pathological calcification in vivo. *Science*, 1969, *165*, 1264–1266.
4. King, W. R., Francis, M. D., and Michael, W. R. Effect of disodium ethane-1, hydroxy-1, 1-diphosphonate on bone formation. *Clin. Orthopaed.*, 1973, *72*, 251–270.
5. Pentegrasse, H., Potsaid, M., and Castronovo, F. Clinical use of ^{99m}Tc diphosphonate. *Radiology*, 1973, *107*, 557–562.
6. Subramanian, G., McAfee, J. G., Bell, E. G., Blair, R., O'Mara, R., and Ralston, P. ^{99m}Tc labeled polyphosphate as skeletal imaging. *Radiology*, 1972, *102*, 701–704.

PITFALLS IN Tc^{99m} POLYPHOSPHATE SKELETAL IMAGING*

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THE introduction of technetium 99m polyphosphate (Tc-PP) in 1971 provided a significant advance in skeletal imaging agents in terms of availability, cost, patient radiation dose and scan quality.[4] At the same time, the clinical demand for bone scans has increased rapidly and whole body studies have become an accepted part of many diagnostic and staging work-ups. The information provided is unique and particularly for patients with carcinomas of the breast, lung and prostate; the critical decisions between curative and palliative therapy often hinge on the scintigraphic demonstration of bone metastatic disease.

O'Mara has compiled numerous benign causes for positive bone scans with F^{18} fluoride.[1] From the mechanisms involved, most of these may logically be expected to result in positive Tc-PP scans and must be recognized for proper diagnosis. Charkes *et al.*[2] have described the elements of normal Tc-PP rectilinear bone scans and have cited the need for new diagnostic skills in their interpretation.

The purpose of this report is to describe certain additional problems in scan interpretation and quality control encountered in Tc-PP skeletal imaging.

MATERIAL AND METHOD

Six hundred consecutive anterior and posterior total body (skull to below knees) bone scans were included in the study. The scans were obtained 60–240 minutes post intravenous injection of 15 millicuries Tc-PP using an Ohio-Nuclear Dual Five Scanner with 5 inch focal depth collimators. Patients were hydrated following injection and instructed to void just prior to scanning.

Scans were initially interpreted for the presence of any bone lesion and assessed for quality. They were then reviewed with clinical and roentgenographic correlations to determine presence of false positive or misleading skeletal patterns, presence of normal variations, and presence of abnormal patterns of extraskeletal radionuclide distribution. In addition, a subset of 150 consecutive cases was analyzed statistically for per cent positives and for quality as a function of patients' age.

RESULTS AND DISCUSSION

Forty-five per cent of the 150 case subset were positive. Table 1 lists the distribution by pathologic diagnosis. This percentage of positives is higher than for most nuclear medicine procedures and probably reflects a greater patient selectivity still exercised by referring physicians.

A. SCAN QUALITY

Scan quality was best in younger patients. Eighty-two per cent of the studies on patients less than 40 years of age were judged good and only 18 per cent fair or poor. In patients over 40, 63 per cent were good and 37 per cent fair or poor. The most frequent cause of poor quality studies was increased extraskeletal background activity (Fig. 1). Slower clearance and uptake kinetics for Tc-PP in the metabolically less active older patients would help explain the age discrepancy and might be usefully studied. Free Tc^{99m}-pertechnetate (TcO_4^-) also contributes to background and this source of activity should be suspected if

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TABLE I

DISTRIBUTION BY DIAGNOSIS IN 150 CONSECUTIVE CASES

Diagnosis	Total Number	Number Positive	Per Cent Positive
Breast carcinoma	22	12	55
Hodgkin's disease	19	2	10
Prostatic carcinoma	19	9	47
Lung carcinoma	13	7	54
Non Hodgkin's lymphomas	9	4	44
Colon and other gastrointestinal carcinomas	8	4	50
Melanosarcoma	8	6	75
Testicular tumors	7	0	0
Renal cell carcinoma	5	1	20
Head and neck tumors	4	3	75
Primary bone tumors	3	3	100
Multiple myeloma	2	0	0
Eosinophilic granuloma	2	2	100
Miscellaneous malignancies	17	10	58
Miscellaneous benign conditions	10	4	40
Total	150	65	45

excessive localization is noted in the thyroid gland.

Several factors adversely affect scan quality locally. Excessive activity at the injection site due to soft tissue extravasation may obscure bone lesions. Care must be used to avoid any symptomatic or roentgenographically positive area for the venipuncture site. Jewelry and other superficial objects such as prostheses or pacemakers can also obscure underlying areas. The 140 kev. principal gamma photons of Tc^{99m} are significantly less penetrating than the 511 kev. annihilation quanta of F^{18} or the 513 kev. photons of Sr^{85}. The problem is therefore accentuated on Tc-PP studies compared to those with the other agents. If an object cannot be removed (subcutaneous pacemaker), the shape and location should be noted at the time of patient interview for subsequent scan correlation.

Another local problem is Tc-PP activity in the bladder which may obscure much of the bony pelvis. Patients should void immediately prior to scanning. This allows adequate pelvic visualization in patients with normal bladder function, although patients with prostatic carcinoma often have partial bladder obstruction making one of the areas of prime scan interest difficult to evaluate. Also, after voiding, there is invariably some staining of the skin and possibly clothing by Tc-PP. Recognition of this is necessary to avoid reporting spurious lesions due to extraneous superficial contamination.



FIG. 1. Poor quality scan due to excessive extraskeletal background activity. The skeletal structures are obscured and poorly defined. The intense thyroid accumulation indicates the presence of free TcO_4^-. Separation of the intestinal and abdominal wall activities (arrows) indicates the presence of moderate ascites.

B. EFFECTS OF UNBOUND TC^{99m} PERTECHNETATE

As noted above, excessive unbound TcO_4^- detrimentally affects general scan quality. Free TcO_4^- normally localizes in the thyroid and salivary glands, stomach, intestines and until cleared, the blood pool (Fig. 1). Thyroid activity must not be confused with bone activity and evaluation of the cervical vertebrae must be accomplished by lateral or posterior views when the thyroid obscures the anterior view.

An occasional diagnosis results from the presence of free TcO_4^-. In Figure 1, bowel and body wall soft tissue activity allowed demonstration of ascites. Ascitic fluid does not exchange readily and the effect is similar to the total body opacification effect sought in pediatric contrast urography. We have also noted "cold" areas due to renal cysts and gastric and colonic air on the same basis.

C. NORMAL VARIATIONS IN SKELETAL DISTRIBUTION

Scans obtained with Tc-PP ensure greater average information density in a practical scanning period than is the case with either F^{18} or Sr^{85}.[2] This significantly accentuates physiologic variations in nuclide distribution. These variations must be recognized as normal to avoid false positive interpretations.

The skull provides an important example. Wide variations in calvarial thickness occur normally (even in patients without hyperostosis) and are easily appreciated roentgenographically in areas of the skull viewed tangentially. On both gamma camera scintiphotos and rectilinear scans, the parts of the skull viewed tangentially provide the highest count rates and lesions may be simulated along the periphery at points of local calvarial thickening. For this reason, suspected skull lesions must be demonstrated *en face* before being accepted as true abnormalities. Also, due to the curvatures of the skull, the actual nature or configuration of a lesion may not be appreciated on tangential views (Fig. 2; 3; and 5).

Normally, there are three distinct loci of Tc-PP uptake at the shoulder corresponding to ossification centers of bone development: the proximal humerus, the acromioclavicular joint, and the coracoid process of the scapula. In 19 (13 per cent) of the 150 case subset, one of the shoulders demonstrated more activity than the other in all 3 loci (Fig. 4). Only cases in which this existed on both the anterior and posterior views were counted in order to eliminate positioning effects. In all but 2 cases, the activity was greater on the right than the left. In several there was also diffuse increase in ipsilateral rib activity. The right-left percentages correlate well with the "handedness" of the population and these unilateral increases may be secondary to musculoskeletal use factors. Less frequently, similar right–left discrep-

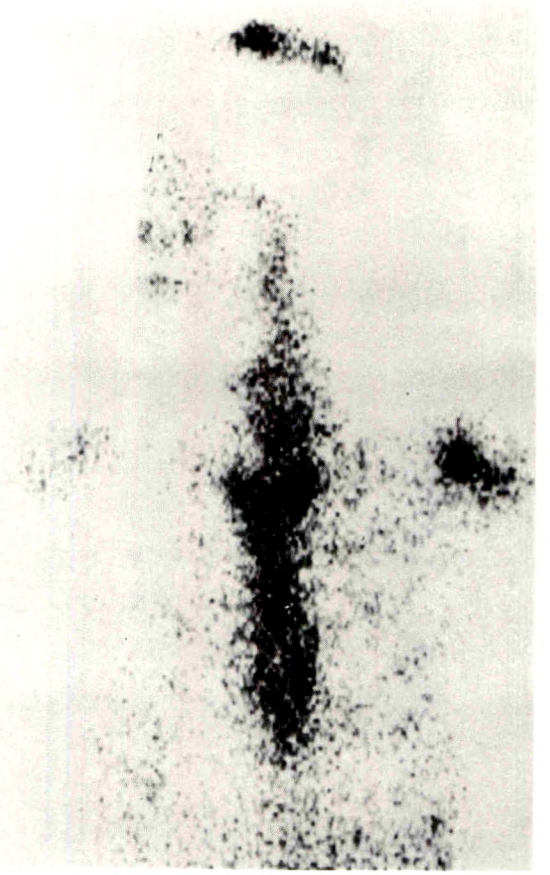


FIG. 2. Probable metastatic lesion near the vertex of the skull in a patient with pancreatic carcinoma. On the rectilinear scan the area of abnormality is viewed tangentially.

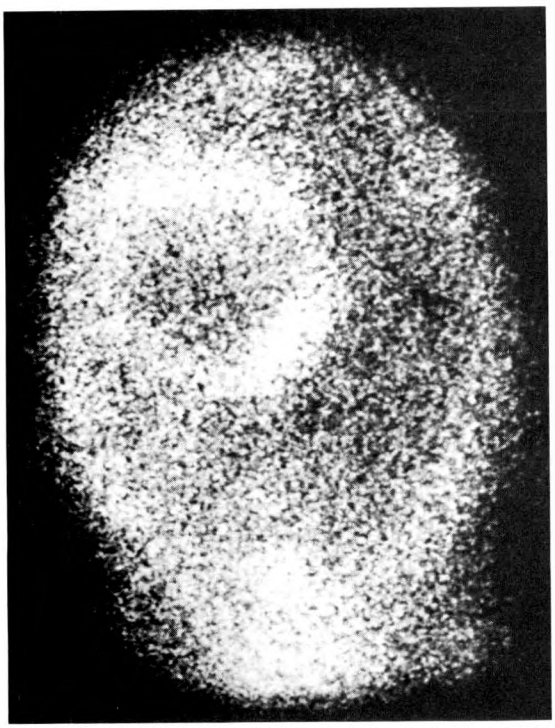


Fig. 3. Gamma camera vertex view of the patient in Figure 2. The suspicious area is demonstrated *en face* and confirmed. Even if the original tangential view had been accepted as sufficient proof of abnormality, the unusual rim configuration of increased uptake would have been missed. Subsequent roentgenograms revealed a lytic metastasis.

ancies occur in the hips and knees.

The sternum, including the manubrium, develops from multiple ossification centers. Any of these centers may be visualized on Tc-PP bone scans analogous to epiphyseal or apophyseal visualization. They are round and discrete simulating metastatic deposits (Fig. 5). Close clinical correlation is required, especially in view of the difficulties in roentgenographic study of the sternum. The sternomanubrial and sternoclavicular joints may also be seen normally.[2]

The dorsal and lumbar vertebrae lie in different frontal planes due to the kyphotic and lordotic curves of the spine. On rectilinear scans the vertebral bodies demonstrate different apparent activities as the spinal column undulates in and out of the focal planes of the collimators. Usually the resulting pattern is easily recognized with smooth gradations of activity from one vertebral level to the next (Fig. 6). If the apex of one of the curves occurs just at the focal plane, a lesion may be simulated. By flexing the patient's hips during lumbosacral scanning, the lumbar lordosis is partially straightened, minimizing the problem at this level. Questionable areas should be imaged with the gamma camera to avoid the tomographic effect of the rectilinear scanner.

D. HYPEREMIC EFFECTS

Pathologic conditions which induce local



Fig. 4. The right shoulder demonstrates subtle but definitely increased Tc^{99m} polyphosphate (Tc-PP) accumulation in all 3 normal loci. The same effect was noted on the posterior scan, eliminating position artifact as the cause. The scan also demonstrates symmetric Tc-PP accumulation in normal breast tissue.

or regional hyperemia may cause increased accumulation of Tc-PP in otherwise normal bone by increasing bone blood flow. Hyperemia of any etiology may also cause an increase in bone metabolic activity. This creates a problem in interpreting pathologic conditions where hyperemia is present. In over 90 per cent of primary malignant bone tumors of the upper and lower limbs, diffusely increased Tc-PP localization was noted in the entire involved extremity. The increase occurs both proximal and distal to the lesions (Fig. 7). It occurs on both sides of joints and in 1 case of Ewing's sarcoma of the ilium, involved the entire ipsilateral lower extremity. Marked increase in soft tissue activity is usually associated with the bone increases. The diagnostic dilemma is in deciding how far the lesions extend and how much of the scan abnormality is the result of hyperemia. This is particularly true for round cell tumors (Ewing's sarcoma, reticulum cell sarcoma) which can actually involve an entire long bone.

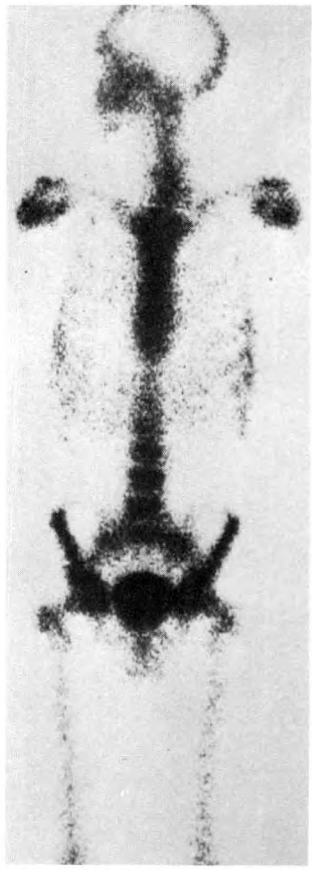


Fig. 6. Normal anterior view of a whole body rectilinear scan. The lumbar vertebral bodies demonstrate progressively increasing activity cephalocaudad as the lumbar lordotic curve brings them into the focal plane of the collimator.



Fig. 5. Persistent sternal ossification center (arrow). These may occur in any sternal segment and simulate metastasis. Incidentally, note also the variations in skull activity along its tangentially viewed margin. Gamma camera views were negative.

The same hyperemia induced phenomenon of diffusely increased Tc-PP uptake also occurs secondary to arthritis, fracture, osteomyelitis, thrombophlebitis and an occasional case of bone metastatic disease. In 1 of our cases a metastasis in the ischium was obscured by diffuse soft tissue accumulation secondary to the hyperemia of thrombophlebitis. The opposite effect occurs in patients with severe occlusive vascular disease. One patient with unilateral claudication showed virtually no Tc-PP uptake on the affected side in either bone or soft tissue.

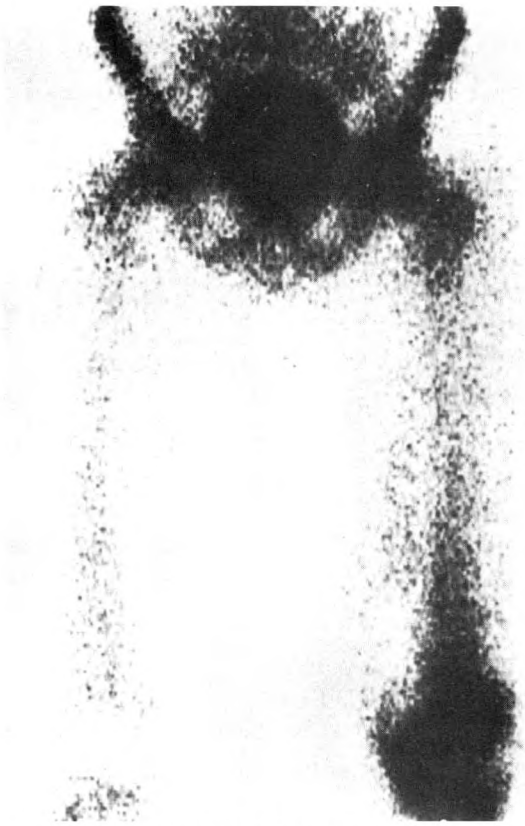


Fig. 7. Example of the hyperemic effect secondary to an osteosarcoma of the distal femur. Pathologically the tumor was confined to the distal 1/3 of the shaft. The scan demonstrates increased Tc-PP accumulation throughout the entire extremity including the whole femoral shaft, proximal tibia and diffusely in the soft tissues. The scan findings do not accurately portray the extent of the tumor.

E. NORMAL AND ABNORMAL EXTRASKELETAL PATTERNS

Extraskeletal uptake of Tc-PP is normal in several structures. Faint outlines of the heart blood pool and liver are normally seen. The kidneys and bladder are well visualized and should be inspected in all cases. Absence of a kidney, renal masses and urinary tract obstruction have all been noted incidentally. The abnormalities are often unsuspected clinically and should be reported. Another incidentally noted and initially perplexing area of extraskeletal Tc-PP accumulation was a distinct curvilinear line medial to the femur (Fig. 8). This was demonstrated in 45 per cent of patients over 40 years of age. The finding is most marked in patients with Mönckeberg's medial sclerosis but also occurs without arterial calcification visible roentgenographically. It probably represents accumulation of Tc-PP in the calcified media of the femoral artery.

Tc-PP frequently localizes in normal breast tissue and accumulation is usually bilateral when this occurs (Fig. 3; and 9). Unilateral visualization should be considered abnormal until proven otherwise. Breast carcinomas may show uptake and in 1 case in this series, uptake in a pathologic rib fracture simulated breast activity (Fig. 10). Also, the remaining normal

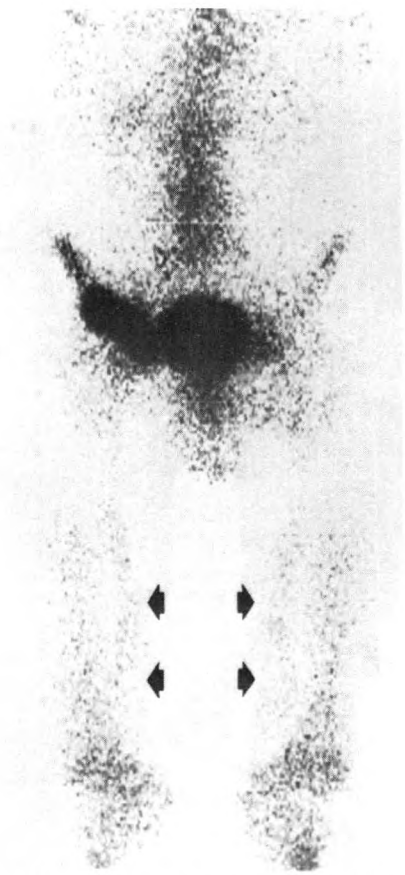


Fig. 8. A 65 year old male with extensive iliofemoral vascular calcification demonstrated roentgenographically. The scan reveals a distinct curvilinear line of Tc-PP accumulation medial to the femurs bilaterally (arrows). This probably represents Tc-PP uptake in the diseased femoral vessels. (The patient has a right acetabular metastasis.)

breast may accumulate Tc-PP in patients who have had a mastectomy.

Several malignancies demonstrated extraskeletal Tc-PP accumulation. Six of 8 patients with malignant melanoma being scanned for possible bone lesions demonstrated Tc-PP uptake in soft tissue masses (Fig. 11). Sporadic cases of Hodgkin's disease, bronchogenic carcinoma and several types of soft tissue sarcoma also demonstrated soft tissue tumor uptake. Visualization of osteosarcoma metastatic to the lung has been reported with other agents.[3] There were 2 such cases in this series with Tc-PP and in 1 of them the scan revealed numerous pulmonary lesions not seen on whole lung tomograms.

Healing surgical wounds frequently showed diffusely increased activity (Fig. 12). Clinical correlation was usually easy, but in the chest incisions often paralleled ribs. Wound and rib activities then superimpose and distinction may not be possible.

A definitive pattern has not been established for soft tissue uptake in irradiated areas. Both increased and decreased activity have been noted in treated areas. The problem is often compounded by soft tissue losses and surgical wounds in treatment fields.

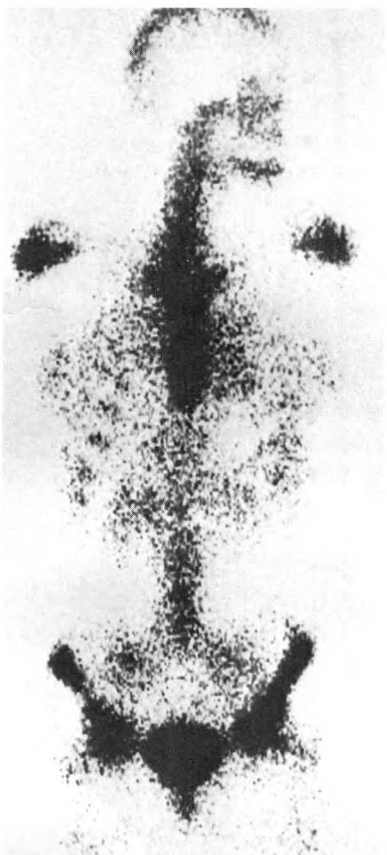


Fig. 9. Bilaterally symmetric Tc-PP accumulation in normal breast tissue. The mechanism for this remains to be defined. Unilateral uptake may indicate breast tumor.



Fig. 10. A 13 year old female with Ewing's sarcoma of the left femur. Accumulation of Tc-PP in left chest area (arrow) was initially thought to be in the breast. The unilaterality raised suspicion and roentgenograms revealed a silent pathologic rib fracture. The patient had received cobalt 60 teletherapy to the left hemipelvis and femur 18 months previously. Note the decreased bone development (short femur, hypoplastic left ilium).

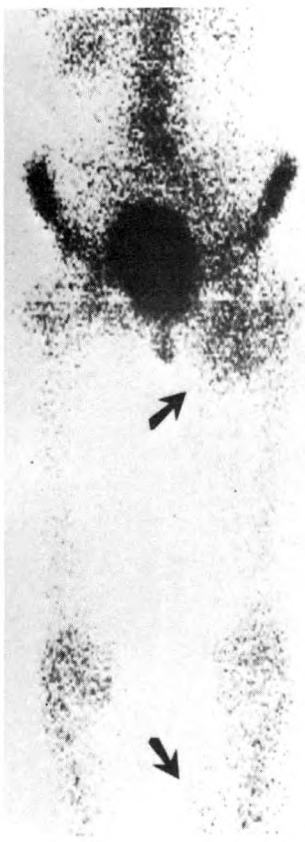


Fig. 11. Malignant melanoma. The primary site was in the soft tissues of the left calf with metastasis to the left inguinal lymph nodes. Both the primary tumor (lower arrow) and the metastasis (upper arrow) demonstrate increased accumulation of Tc-PP.

SUMMARY

The quality of Tc-PP scans is best in younger patients. In all age groups quality is adversely affected by free TcO_4^-. This should be suspected if excessive background is present or marked accumulation is noted in the thyroid gland.

Direct physician supervision of all studies is recommended. Symptomatic and roentgenographically positive areas may then be determined for special interpretative attention. Objects which attenuate activity should be removed or noted. Potential sources of superficial contamination (product spill, urine, saliva) must be known and considered when extraneous activity does not correlate with skeletal structures or clinical findings.

Normal variations in skeletal uptake of Tc-PP include asymmetries between periarticular areas on the right and left, local increases due to physiologic calvarial thickening and uptake in persistently visualized sternal ossification centers. Apparent variations in the vertebral activity may be due to focal plane effects of rectilinear scanners.

Hyperemia can cause diffusely increased Tc-PP localization in an entire extremity. This effect may completely obscure a lesion or obscure the extent of a lesion.

Tc-PP excretion through the kidneys occasionally provides useful morphologic or functional information concerning the

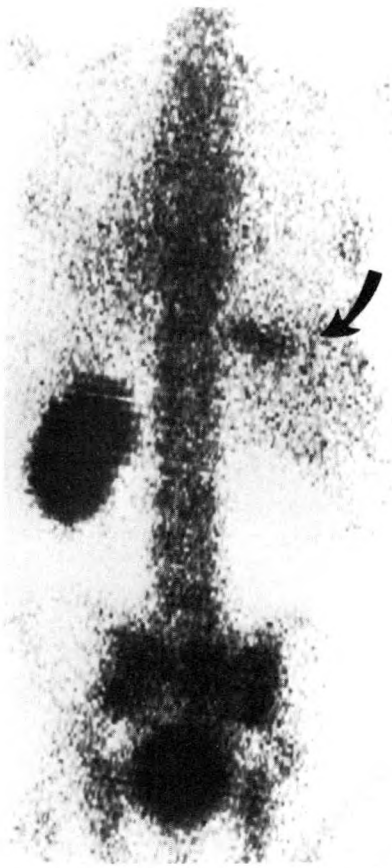


Fig. 12. Increased activity in area of surgical wound (arrow) following right nephrectomy through a flank incision. It is often impossible to separate wound activity and skeletal activity, particularly in the thorax.

urinary tract which should be inspected on all scans.

Abnormal soft tissue uptake occurs in a variety of conditions including healing wounds and some malignant tumors. A large percentage of patients over 40 years of age demonstrate localization of Tc-PP in the walls of the femoral vessels.

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REFERENCES

1. Blau, M., Ganatra, R., and Bender, M. ^{18}F-fluoride for bone imaging. *Seminars Nuclear Med.*, 1972, *2*, 31–37.
2. Charkes, N. D., Valentine, G., and Cravitz, B. Interpretation of normal Tc^{99m} polyphosphate rectilinear bone scan. *Radiology*, 1973, *107*, 563–570.
3. Schall, G. L., Zeiger, L., Primack, A., and De Lellis, R. Uptake of ^{85}Sr by osteosarcoma metastatic to lung. *J. Nuclear Med.*, 1971, *12*, 131–133.
4. Subramanian, G., and McAfee, J. G. New complex of Tc^{99m} for skeletal imaging. *Radiology*, 1971, *99*, 192–196.

INFLUENCE OF FRACTIONATION ON ACUTE AND LATE REACTIONS IN VOCAL CORD CARCINOMA*

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THE curability of vocal cord carcinoma by irradiation is well established.[11,17] Local control and survival rates are reasonably objective end points for analysis. The preservation of voice is a major justification for the use of radiation in preference to surgery. Inconvenience of treatment, severity of acute reactions and late tissue changes, and quality of voice are more subjective and less important than cure, but nevertheless are real considerations for the patient. One schedule frequently used for the treatment of localized vocal cord carcinoma is 6,000 rads/30 treatments/6 weeks, using Co^{60} or other megavoltage radiation. A brisk acute reaction develops, usually a confluent mucositis, with a moderate incidence of permanent aryepiglottic edema. This edema is frequently minimal, and is accepted as a sequela of therapy, to be distinguished from the severe edema which is considered a complication.

The present study was started 13 years ago, to determine whether the acute reaction and *all* visible late change could be decreased or eliminated, without reduction in cure rate, by decreasing daily dose but increasing total dose and time. The inconvenience to the patient of the increased number of treatments would be at least partially balanced by the decreased discomfort; and the high cure rate would permit most patients to derive an over-all benefit from even a minimal improvement in subsequent functional status. Any conclusions might have more general applicability, since the anatomy of the aryepiglottic folds permits visible change from even minor irritation, but similar changes could occur in less visible tissues throughout the body. Three years after this study was started, a preliminary report by Buschke and Vaeth on a similar study offered encouragement to continue.[5,19]

MATERIAL AND METHOD

All patients with T_1, T_2, or T_3, N_0 squamous cell carcinoma of the true vocal cords referred between January 1, 1960 and January 1, 1971 were treated with 1 of 4 daily dose schedules, 5 days per week, these schedules having been sequential and not randomized. The lesions were restaged for this analysis using the criteria of the July, 1972, revision of the American Joint Committee. Directly opposed 25 mev. betatron photon fields were used. Most patients were treated with gelatin-glycerin bolus to achieve a calculated uniform dose distribution within the laryngeal contents (the "tumor dose"). This distribution has subsequently been checked using a DEC RAD-8 computer with agreement. T_1 lesions were treated using 5×5 to 7×7 cm. fields, T_2 and T_3 lesions using larger fields up to 12×8 cm. The data are presented in "R," 1 "R" being 0.89 rad. Since most recurrences and complications are evident within 2 years, the data include all patients who started treatment prior to January 1, 1971. All patients were checked for 5 years or to the present time. Each group of patients was treated using a constant daily dose. Within each group, the total number of treatments varied within narrow limits, several extra treatments having been given to the more slowly regressing lesions.

RESULTS

The first group, 7 patients, was given 200

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R tumor dose daily to a total dose of 6,000 R, to determine whether the acute mucosal reactions from 25 mev. photon radiation were comparable to those from Co^{60} gamma radiation. A confluent mucositis developed within 3 to 4 weeks in all 7. The second group, 3 patients, received 180 R daily to 6,300–6,600 R total. The reactions were limited to a patchy mucositis. The third group, 5 patients, received 160 R daily for 43 treatments (6,880 R in 8½ weeks). No reactions developed except for a minimal erythema with a mild "sore throat."

An approximate time-dose-response relationship for these specific conditions having been established, the fourth group, 27 patients, was treated at the rate of 170 R daily. A standard dose of 7,140 R, or about 6,400 rads, in 42 treatments was adopted, with several patients receiving up to 3 additional treatments. Acute reactions developed but remained minimal.

The results are given in Tables I–III. The number of patients is too small to provide significant control or survival statistics, except that these are comparable to other reported results. The virtual elimination of acute reactions and of visible late change by decreasing the daily dose is quite definite, however. In the 200 R/treatment group, all developed moderate aryepiglottic edema which persisted for years or was permanent. Half of the 170 R group never developed visible edema; most of the remainder had minimal transient edema; and only about 10 per cent had more definite edema, none persisting beyond 2½ years. None of the 160 R group developed any visible edema at any time.

DISCUSSION

A number of mathematical expressions of the time-dose relationship have been proposed both for normal tissues and for malignancies. Typically, because of recovery, a higher dose must be administered when time is prolonged, although the complexities of the problem are accentuated by Rubin's demonstration of a "reverse Strandquist effect."[3,4,6,7,8,10,15,20] Formulae which express a time-dose relationship for

TABLE I

T_1N_0 VOCAL CORD LESIONS

Daily Dose (R)	Total Dose (R) / Number of Treatments	Number of Patients	Results
200	6,000 / 30	3	No recurrence 3 edema for 1 to 3½ years
180	6,300 / 35	1	No recurrence No edema
170	7,140–7,310 / 42–43	15	2 recurrence {d. @ 8 mo.; d. @ 1 yr. 1 mo.} 5 slight edema, cleared by 6 mo. 8 no edema
160	6,880 / 43	4	No recurrence No edema
	Total	23	2 recurrence (died of disease)

TABLE II

T_2N_0 AND T_3N_0 VOCAL CORD LESIONS

Daily Dose (R)	Total Dose (R) / Number of Treatments	Number of Patients		Results
200	6,000 / 30	4	1—T_2	No recurrence
			3—T_3	4 edema for 1½ to 5 yr.
180	6,480–6,660 / 36–37	2	1—T_2	1 recurrence (T_3 d. @ 1 yr. 3 mo.)
			1—T_3	1 slight edema, cleared by 1½ yr.
170	7,140–7,650 / 42–45	12	6—T_2	3 recurrence (T_2 l. @ 5 yr.—surgery); (T_2 d. @ 1 yr. 9 mo.); (T_3 d. @ 10 mo.) 3 edema for 2 to 2½ yr.
			6—T_3	3 slight edema, cleared by 6 mo. 3 no edema
160	6,880 / 43	1	1—T_2	No recurrence No edema
	Total	19		4 recurrences (1 living after laryngectomy)

TABLE III

T_1N_0, T_2N_0, AND T_3N_0 VOCAL CORD LESIONS

Daily Dose (R)	Total Dose (R) / Number of Treatments	$D/t^{0.22}$ (R)	NSD (rad rets)	Results	Number of Patients
200	6,000 / 30	2,372	1,573	No recurrence 7 edema	7
180	6,480 / 36	2,461	1,594	1 recurrence 1 slight edema 1 no edema	3
170	7,140 / 42	2,621	1,664	5 recurrence 3 edema 8 slight edema 11 no edema	27
160	6,880 / 43	2,516	1,591	No recurrence No edema	5
	Total	2,500±5%	1,600±3%	6 recurrence (1 living—surgery)	42

tumor control tend to be reasonably reliable, since growth and recovery are relatively autonomous, and tumor size or control as end points can be reasonably well defined. Several authors have obtained a regression coefficient of about 0.22 (but with a range from 0.12 to 0.30) for squamous cell carcinoma in various organs, treated 5 days per week. Since the main aim of treatment is tumor control, this relationship permits approximate projection of equivalent doses for different total time schedules. Regression coefficients for malignancies other than squamous cell carcinoma seem less reliable.

The most frequently used formula at the moment, however, is that of Ellis (NSD $=$ Total Dose $\times N^{-0.24} \times T^{-0.11}$), which is intended to define normal tissue tolerance as the limiting factor for most radiation therapy.[7–9] Both the principle, and the initial equation which is based on skin reactions, were offered as starting, not as final, suggestions. Ellis proposed that the equation may have more general applicability, with the tolerance of the connective tissues usually being the limiting factor for recovery. He has said[9] that "continual testing and sifting of data are desirable to verify the concept and to adjust the exponents of the formula." Despite these words of caution, many authors have presented conclusions based on the *a priori* assumption that the formula is already of proven validity. In one recent publication[2] a single optimal dose in rets is proposed for the treatment of laryngeal carcinoma, based upon an analysis of 6 published dosage schedules from different institutions.

Doubts about the Ellis formula have been expressed by Liversage[13,14] and Probert,[18] among others.[11] Very few reports on the tolerance of the larynx are available to explicitly test the validity of the formula. Intercomparisons between institutions are of dubious value unless pre-planned, with agreement on dosimetry, details of technique and definition of dose. Comparative series with only one variable (for example, the demonstration of an increase in necrosis with increased dose, while keeping the number of treatments and total time constant), cannot explicitly confirm the validity of the Ellis formula, but can only indicate that it is one of several relationships which may be consistent with the data. Definite changes or necrosis of the normal tissues must be demonstrated, since an infinite number of equations can fit data showing no change.[16] Data from authors reporting on comparative dosage schedules within a given institution are shown in Table IV.[1,12,20] The Ellis formula with the present exponents is not consistent with these data, but would be closer if total time were a more important factor, indicating a greater rate of recovery.

Tolerance is a less well-defined end point than is tumor control, and is more difficult to formulate. Healing of the acute reaction is a possible end point, but this does not distinguish between the cicatrized tissues which frequently result from short intensive therapy compared with the more normal tissues following more prolonged treatment even though to higher total doses. Several authors have proposed schedules based on larger daily increments with acceptable acute reactions, only to subsequently report severe late complications. Minor differences in late tissue reactions are readily visible in the larynx. Mild edema or fibrosis elsewhere in the body is less obvious, but may produce symptoms or complications of greater importance than hoarseness. Other authors have also observed that a tumor dose of 800 rads weekly to the whole pelvis is much better tolerated than is 1,000 rads, with decrease in both acute and late reactions, without apparent decrease in tumor control. We have treated several patients who have had recurrences about the head and neck following extensive irradiation and surgery, at the rate of 90 rads daily to total doses of 8,500–9,500 rads, without appreciable local reactions or further tissue break-down, with remissions of several years to date. This technique is not practical as a routine, nor even necessarily optimal for control of previously untreated

TABLE IV

NSD COMPARISONS

Author	Dose/Treatments/Time	NSD rets	Results
Horiot *et al.*[12] Co^{60}	5,500 rads/20 rxs/4 wk.	1,872	Unacceptable edema and necrosis
	6,000 rads/27 rxs/5½ wk.	1,828 ↓ 2%	Acceptable: T_1—12% recurrence; T_2—25% recurrence
Loeffler 25 mev.	6,000 R/30 rxs/6 wk.	1,573	Edema: T_1—9% recurrence 2/23
	7,140 R/42 rxs/8½ wk.	1,664 ↑ 6%	Rare edema: T_1T_2—25% recurrence 5/20
Abramson *et al.*[1] Co^{60}	6,000 rads/18 rxs/6 wk.	2,000 △12%	Similar reactions: 10% recurrence 6/63 Stage I and IIA
	6,000 rads/30 rxs/6 wk.	1,760	8% recurrence 4/50
Botstein Co^{60}	5,500 rads/18 rxs/6 wk.	1,835 △2%	?Similar reactions: Recurrences
	6,000 rads/24 rxs/8 wk.	1,804	Acceptable control

malignancy, but is of value for this specific problem.

The use of shorter treatment schedules, either in total time or in number of treatments, seems justified for palliative therapy or for the treatment of patients who have little likelihood of cure, to relieve the burden on busy therapy facilities, and to decrease the inconvenience and expense to the patient. However, for patients who have highly curable lesions, where the total treatment time is likely to be small compared with future longevity, convenience for the therapist and economic factors should be of lesser importance. In practice, while increasing the number of treatments for the more curable patients does prolong the daily schedule somewhat, this requires more technical than professional time. The patients can safely be examined less frequently, and they have fewer problems to discuss. Most of the patients reported here continued near-normal activities, and many "dropped in" for their treatments on the way to or from work.

In January, 1973, after reviewing the data presented here, the standard doses were increased in an attempt to further reduce the local recurrence rates. Current schedules are 6,750 rads (7,500 R) in 9 weeks for T_1 lesions, and 7,500 rads (8,300 R) in 10 weeks, or more, for T_2 and T_3 lesions, with a constant daily dose of 150 rads (169 R).

SUMMARY

Four schedules for the treatment of localized vocal cord carcinoma were compared, to establish a relationship between daily and total dose, and acute and late normal tissue reactions. All patients were treated 5 days per week.

All of those who received 200 R daily (6,000 R in 30 treatments) developed a confluent mucositis and minimal to moderate subsequent aryepiglottic edema.

Patients who received 180 R daily (6,480 R in 36 treatments) developed a patchy mucositis.

Those who received 170 R daily (7,140 R or 6,400 rads in 42 treatments) developed only a brisk erythema, with decreasing incidence and severity of edema.

At 160 R daily (6,880 R in 43 treatments) only a mild erythema developed, with no visible edema at any time.

Local recurrence rates of about 10 per cent for T_1 lesions, and of about 25 per cent for T_2 and T_3 lesions, indicate that the decrease in reactions with longer fractionation has been achieved without loss of tumor control. Even minimal improvement in functional status following treatment for this highly curable malignancy well justifies the inconvenience of the increased number of treatments.

The present formula of Ellis for normal tissue tolerance is not consistent with these findings, since the NSDs are essentially identical for all 4 schedules, while tolerance is notably improved by increasing fractionation.

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REFERENCES

1. Abramson, N., Cavanaugh, P. J., and Connell, H. C. Radiation therapy in early carcinoma of vocal cords: change in fractionation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 548–552.
2. Aristizabal, S. A., and Caldwell, W. L. Radiation tolerance of normal tissues of larynx. *Radiology*, 1972, *103*, 419–422.
3. Baclesse, F. Clinical experience with ultrafractionated roentgen therapy. In: Progress in Radiation Therapy. Edited by F. Buschke. Grune & Stratton, Inc., New York, 1958, pp. 128–143.
4. Baclesse, F. Hyperfractionation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *91*, 32–36.
5. Buschke, F., and Vaeth, J. M. Radiation therapy of carcinoma of vocal cord without mucosal reaction. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 29–34.
6. Buschke, F. Conclusion of symposium on protraction and fractionation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *91*, 61.
7. Ellis, F. Fractionation in radiotherapy. In: Modern Trends in Radiotherapy. Volume 1. Edited by T. J. Deeley, and C. A. P. Wood. Butterworth & Co., Ltd., London, 1967, pp. 34–51.
8. Ellis, F. Dose, time and fractionation: clinical hypothesis. *Clin. Radiol.*, 1969, *20*, 1–7.
9. Ellis, F. Nominal standard dose and ret. *Brit. J. Radiol.*, 1971, *44*, 101–108.
10. Fowler, J. F. Estimation of total dose for different numbers of fractions in radiotherapy. *Brit. J. Radiol.*, 1965, *38*, 365–368.
11. Goffinet, D. R., Eltringham, J. R., Glatstein, E., and Bagshaw, M. A. Carcinoma of larynx: results of radiation therapy in 213 patients. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 553–564.
12. Horiot, J. C., Fletcher, G. H., Ballantyne, A. J., and Lindberg, R. D. Analysis of failures in early vocal-cord cancer. *Radiology*, 1972, *103*, 663–665.
13. Liversage, W. E. Critical look at ret. *Brit. J. Radiol.*, 1971, *44*, 91–100.
14. Liversage, W. E. Nominal standard dose and ret. *Brit. J. Radiol.*, 1971, *44*, 477–478.
15. Marcial, V. A. Time-dose-fractionation relationships in radiation therapy. In: National Cancer Institute Monograph. No. 24: Radiobiology and Radiotherapy, Feb., 1967, pp. 187–203.
16. Marks, J. E., Lowry, L. D., Lerch, I., and Griem, M. L. Glottic cancer: analysis of recurrence as related to dose, time, and fractionation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 540–547.
17. Morrison, R. Review article: radiation therapy in diseases of larynx. *Brit. J. Radiol.*, 1971, *44*, 489–504.
18. Probert, J. C. Doubts about nominal standard dose. *Brit. J. Radiol.*, 1971, *44*, 648.
19. Vaeth, J. M., and Buschke, F. Radiation therapy of carcinoma of vocal cord without mucosal reaction. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *97*, 931–932.
20. Vaeth, J. M. Editor. Relationship of time and dose in radiation therapy of cancer. In: Proceedings of the Third Annual San Francisco Cancer Symposium. S. Karger AG, Publisher, 1968.

THE RESULTS OF SPLIT-COURSE RADIATION THERAPY IN CANCER OF THE LUNG*

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THE primary role of radiotherapy in the patient with advanced carcinoma of the lung is to offer palliation of distressing symptoms. This palliation may be expressed in terms of prolongation of survival, if relief of symptoms is also obtained. Occasionally, aggressive palliative therapy may result in a cure or a long term survival.

Since most carcinomas of the lung have limited radiosensitivity, for effective palliation the dosage has to be of a moderately high level. Various time-dose relationships with standard daily fractionation and split-course radiotherapy have been employed.[1,3–5,7] The aim of palliation for the patient with lung cancer is to achieve effective relief of symptoms with minimal morbidity from the treatment, and limitation of the time and cost involved for an individual who is probably incurable. At the same time it is felt valid not to jeopardize his chances for cure if a cure is possible. If survival is the objective, then it is important that relief of symptoms are also obtained and that a significantly greater survival is achieved than with no therapy at all.

Radiotherapists utilizing split-course treatment for lung carcinomas have reported variable results. A high order of palliation has been achieved with improved survival[1] and on the other hand the palliation and survival have been at least as good as with conventional irradiation.[4,5,7] Certain advantages, however, are inherent in the split-course approach: a planned rest period allows reactions to improve and a positive attitude on the patient's behalf to further treatment; persistent severe reactions or a poor response after the rest period may determine a less aggressive policy or a radical approach may be adopted if the tumor response appears to warrant it.

The present report deals with a series of patients treated by the split-course technique and compares results with a similar group who were treated by a standard daily fractionation regimen. The results have proved satisfactory and it has been possible to draw certain conclusions as to the reason for the variable results reported in the literature. Variations of the split-course technique will be discussed and their rationale elaborated upon. Finally, the complications of treatment and methods to avert these are described.

MATERIAL AND METHOD

Between September 1968 and December 1971, 293 cases of carcinoma of the lung were referred to the Division of Radiation Oncology, University of Virginia Hospital. A minimum follow-up of 1 year is available for all patients. Of the 293 patients, 235 were considered incurable and were treated for palliation to the lung, a few of these patients having extrapulmonary metastases. Of the remaining 58 patients, 10 received palliation only to specific extrapulmonary metastases, 33 cases received no treatments at all, and 15 were treated for cure with small field, high dose radiotherapy (Table 1). A positive histologic or cytologic diagnosis of carcinoma of the lung was available in all but 5 patients.

The 235 patients receiving palliation to the lung form the study group. These were separated into those receiving standard daily fractionation and split-course radio-

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therapy. A comparison by age, sex, surgery and histology is made in Tables II and III.

From September 1968, the standard daily fractionation schedule was employed and consisted of 3,500–4,000 rads in 3 to 4 weeks; in December 1969 the split-course was introduced and during the remainder of the 3 year period, the split-course treatment was the main method used. Of those treated employing standard daily fractionation, 90 per cent received their treatment on a Cobalt 60 Unit and the remainder on an 8 MV. linear accelerator. For those receiving split-course therapy, 35 per cent were treated on a Cobalt 60 Unit and 65 per cent on the linear accelerator. Two opposed fields were used, with both fields being treated daily without correction for lung transmission. The split-course fractionation schedule was dependent upon the field size required to encompass the lesion. Initially, only those patients whose treatment volume was encompassed by a field size of less than 225 cm.^{2} (15×15 cm.) were treated and these patients received 2,000 rads in 5 days followed by a 2 week rest period and a second course of 2,000 rads in 8 treatment days. The reason for the apparent empirical decision regarding the volume treated was based on the fact that most of the patients treated by the standard daily fractionation regimen received therapy to fields of a similar size. The average field size for the latter group was 182 cm.^{2} (13.5×13.5 cm.). With the apparent success of the split-course treatment, more advanced cases and some patients who had previously been rejected for therapy were introduced to the split-course regimen. These patients received 2,000 rads in 8 to 10 treatments followed by a 2 week rest interval and a second course of 2,000 rads in 5 to 10 treatments, depending upon the ability to reduce the field size for the second half of the split. The majority of these patients had the entire hemithorax treated on the first course; thus, the average field size was much greater than 225 cm.^{2}. For convenience, the short split-course treatment group has been designated Group A and the long split-course group as Group B (Table IV).

One other variation was introduced into the split-course group depending on response to radiotherapy and the age and general condition of the patient. If these were favorable, following a further 2 weeks' rest, an additional 1,000 rads was

Table I

ALL PATIENTS WITH CANCER OF THE LUNG REGISTERED, DIVISION OF RADIATION ONCOLOGY, UNIVERSITY OF VIRGINIA HOSPITAL

(September 1968 through December 1971)

Treated for cure	15
Treated for palliation to lung	235
Treated for palliation to distant metastases	10
Not treated	33
Total	293

Table II

CHARACTERISTICS OF PATIENTS

	Standard Daily Fractionation	Split-Course
Number	93	142
Average age (yr.)	62	59
Sex M:F	3.8:1	4:1
Thoracotomy	38 (41%)	35 (25%)
Resection	3 (3%)	6 (4%)

Table III

HISTOLOGY

(per cent)

	Standard Daily Fractionation	Split-Course
Squamous cell carcinoma	58	47
Adenocarcinoma	12	12
Undifferentiated		
large cell	25	28
oat cell	2 > 27	11 > 39
No diagnosis	2	2

TABLE IV

TREATMENT CHARACTERISTICS

	Standard Daily Fractionation	Split-Course	
		Group A	Group B
Field size	<182 cm.²	<225 cm.²	>225 cm.²
Cobalt 60	90%	35%	
8 MV.	10%	65%	

delivered employing a small field technique to the primary tumor.

RESULTS

SURVIVAL

Two hundred and thirty-five patients were treated for palliation of lung carcinoma and form the present study group. The treatment method and survival of these 235 patients is analyzed in Table v. At 2 years a larger number of split-course patients were surviving, although no marked improvement in survival is noted at 1 year. Table VI shows a breakdown of the split-course group by field size. Patients in Group A (smaller field size) showed a far better survival at both 1 and 2 years compared with those in Group B (receiving the longer split-course) and also those receiving standard daily fractionation.

The effect of thoracotomy on survival has been examined in Table VII and VIII. In Table VII the survival of all patients unresectable at thoracotomy is analyzed by treatment method and a very dramatic improvement in survival is noted for those treated by a split-course. In Table VIII the survival of patients considered too advanced for thoracotomy is analyzed according to the treatment method and shows that the short split-course group (Group A) has a decided advantage over both the other groups.

PALLIATION

The success of symptomatic palliation is presented in Table IX. Palliation was assessed as successful if a definite improvement was noted by the patient. Slight improvement and equivocal statements were considered failures. The major presenting symptoms are considered individ-

TABLE V

TREATMENT METHOD AND SURVIVAL OF 235 PATIENTS RECEIVING PALLIATION TO LUNG

Treatment Group	Surviving 1 Year	Surviving 2 Years	Per Cent Surviving 1 Year	Per Cent Surviving 2 Years
Standard daily fractionation	22/85	6/85	27	7
Split-course	38/128	12/64	30	19
Incomplete treatment—Conventional	0/8	0	0	0
—Split-course	0/14	0	0	0

TABLE VI

SURVIVAL OF 142 PATIENTS TREATED BY SPLIT-COURSE

Treatment Group	Field Size (cm.) and Fractionation	Surviving 1 Year	Surviving 2 Years	Per Cent Surviving 1 Year	Per Cent Surviving 2 Years
Group A	<15×15, 5 treatments/rest 2 weeks/8 treatments	20/48	10/40	42	25
Group B	>15×15, 10 treatments/rest 2 weeks/10 treatments	18/80	2/24	23	8
Incomplete	——	0/14	0	0	0

TABLE VII

SURVIVAL OF 67 PATIENTS UNDERGOING THORACOTOMY AND COMPLETING TREATMENT

Treatment Group	Field Size (cm.) and Fractionation	Surviving 1 Year	Surviving 2 Years	Per Cent Surviving 1 Year	Per Cent Surviving 2 Years
Group A	<15×15 short course	6/13	3/11	46	27
Group B	>15×15 long course	9/19	2/4	47	50
Standard daily fractionation		10/35	4/35	28	11

TABLE VIII

SURVIVAL OF 146 PATIENTS NOT CONSIDERED FOR THORACOTOMY AND COMPLETING TREATMENT

Treatment Group	Field Size (cm.) and Fractionation	Surviving 1 Year	Surviving 2 Years	Per Cent Surviving 1 Year	Per Cent Surviving 2 Years
Group A	<15×15 short course	14/35	7/29	40	20
Group B	>15×15 long course	9/61	0/20	15	0
Standard daily fractionation		12/50	2/50	24	4

ually, although some patients had more than one when originally seen. Cough, the most frequent complaint was relieved in 65 per cent of cases. Hemoptysis was effectively controlled in 86 per cent and shortness of breath and pain improved in over half the patients. All 4 patients with superior venal caval syndrome had complete relief of obstruction. Weight loss, usually signifying a very ominous prognosis was reversed 50 per cent of the time.

COMPLICATIONS

The evaluation of immediate reactions and pulmonary complications following radiation therapy is rather subjective and complicated by the fact that residual tumor and radiation fibrosis are frequently present together. In general, however, it may be stated that the patients tolerated the split-course treatment better and the radiologic changes were less severe. A more serious and assessable complication is that of radiation myelitis. Two patients developed radiation myelitis in the group of 48 patients receiving the short split-course therapy (Group A). This occurred at 9 and 30 months following treatment. With regard to cardiac problems, 2 patients developed congestive cardiac failure. This appeared to be due to extensive fibrosis in the lungs of patients with gross tumors and not the result of direct irradiation of the heart. One case developing a pericardial effusion following treatment was shown to have malignant cells in the aspirated fluid.

TABLE IX

SYMPTOMATIC PALLIATION

(per cent)

Superior vena caval obstruction (6)*	100
Hemoptysis (4)	86
Cough (1)	65
Shortness of breath (2)	59
Pain (3)	58
Weight loss (5)	50
All symptoms	66

* Numbers in parentheses indicate the order of frequency.

DISCUSSION

The present study is neither prospective nor randomized. Rather, reports in the literature suggesting better results with split-course treatment have been accepted and the method of treatment changed. In

retrospect, a comparison has been made between those patients treated by a standard daily fractionation regimen and those receiving a short split-course treatment (Group A). The 2 groups represent different time periods, but no essential changes in referral population, diagnostic investigation or surgical policy have been determined. No policy concerning chemotherapy existed and recurrences were treated in a sporadic and indifferent fashion. Apart from the dose-time relationships, the major differences between the 2 groups are the number of thoracotomies performed, histology, size of tumor (field size) and therapy unit employed. A larger number of thoracotomies were performed and the average field size smaller for the standard daily fractionation group, indicating a more favorable selection of patients. There were more undifferentiated tumors in the split-course group and these are considered to carry a worse prognosis because of their greater tendency to metastasize at an early stage. In these respects, therefore, it may be said that the standard daily fractionation group was the prognostically better group. In spite of this, the 1 and 2 year survival is greater for the split-course group and is independent of whether or not thoracotomy was performed.

It is reasonable to postulate that the difference in survival observed is due to the dose-time relationships. There are 2 factors in the split-course treatment which may be operative: (a) the large treatment fractions during the first course; and (b) the 2 weeks' rest period. With similar variations Abramson and Cavanaugh[1] also reported improved survivals. However, other authors introducing a rest period into an otherwise conventionally fractionated treatment have not observed better results in lung cancer although reactions to treatment were less severe.[4,5] These findings support the view of Sambrook[8] and Levitt[6] that fewer larger fractions are more effective against anoxic cells and, in conjunction with the split-course approach, more effective advantage can be taken of the differential recovery between normal and neoplastic tissues.

Limited gains in survival are only valid in lung cancer if symptomatic relief can also be obtained. The 66 per cent definite improvement in the present series and 78 per cent reported by Abramson and Cavanaugh[2] would seem adequate to justify prolongation of life in patients with advanced lung cancer.

The occurrence of radiation myelitis has been reported by Abramson and Cavanaugh,[2] and Levitt[6] in their series receiving large initial fractions of radiation. Abramson and Cavanaugh[2] suggested certain technical reasons that may be contributory, but all could be excluded in the presently reported cases. The remaining reason would appear to be that the large fractions of the initial course are more damaging to the spinal cord than would be expected from nominal standard dose (N.S.D.) estimations. To overcome this problem, we minimize further irradiation of the cord during the second course by adopting the treatment plan shown in Figure 1.

Group B consisted of patients where the entire hemithorax was considered at risk and was treated employing a split-course. The first course consisted of conventional fractions but advantage was taken during the second course, wherever possible, to reduce the field size and deliver larger fractions. This may account for the difference in survival noted whether thoracotomy was performed or not. Those patients subjected to thoracotomy had smaller lesions than those considered inoperable and would more frequently have their field size reduced during the second course. Although the survival in those with the gross inoperable lesions was poor, not infrequently effusions would be controlled and symptomatic improvement achieved.

The attempt to select favorable patients for additional irradiation taking into account tumor response was not successful, only 1 of 4 patients living more than 1 year.

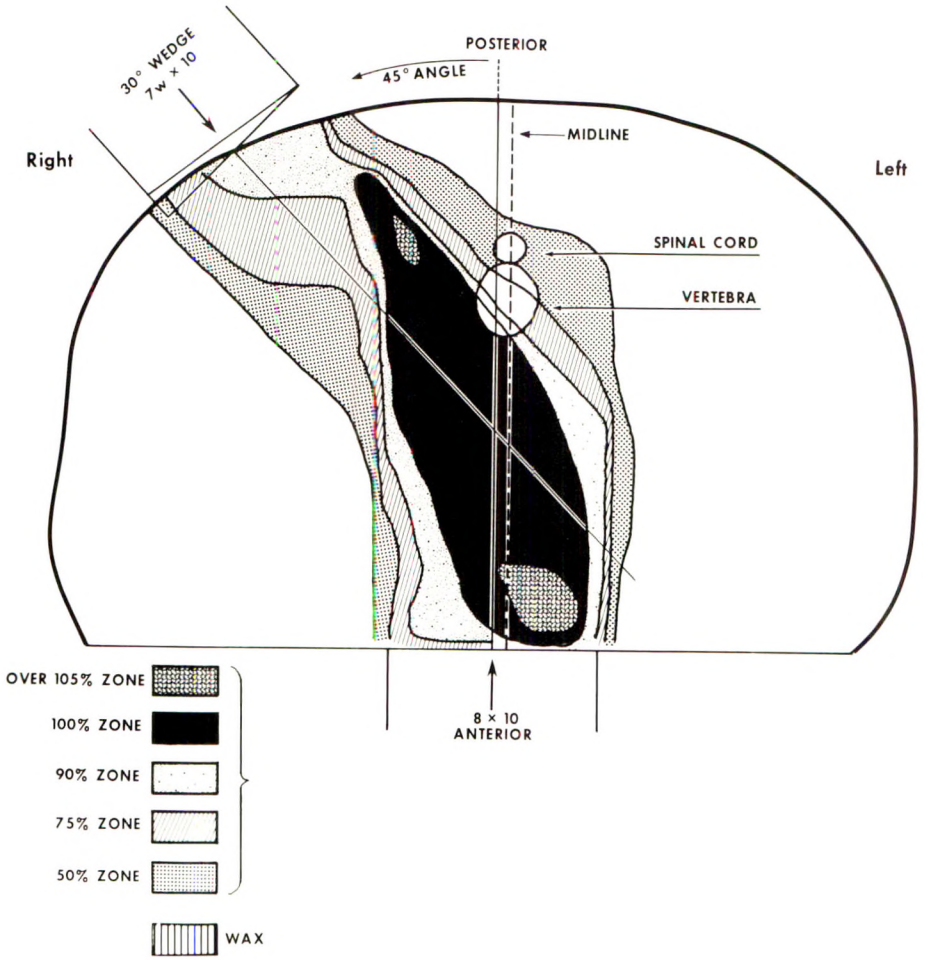


FIG. 1. Treatment plan adopted to minimize radiation to the spinal cord during second part of split-course treatment.

CONCLUSION

Split-course radiotherapy has apparently improved survival with no loss in palliation or increased morbidity except for the question of radiation myelitis. It is anticipated that modifications in treatment planning can eliminate the latter complication.

Success would seem to be related to the utilization of large initial fractions as well as the rest period and a controlled clinical trial is urgently needed. To that end, Levitt[6] is undertaking a randomized study employing these concepts and more recently the Radiation Therapy Oncology Group (R.T.O.G.) has launched a national cooperative study.

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REFERENCES

1. ABRAMSON, N., and CAVANAUGH, P. J. Short-course radiation therapy in carcinoma of lung. *Radiology*, 1970, *96*, 627–630.
2. ABRAMSON, N., and CAVANAUGH, P. J. Short-course radiation therapy in carcinoma of lung: second look. To be published.
3. CALDWELL, W. L., and BAGSHAW, M. L. Indica-

tions for and results of irradiation of carcinoma of lung. *Cancer*, 1968, *22*, 999–1004.
4. GUTHRIE, R. T., PTACEK, J. J., and HASS, A. C. Comparative analysis of two regimens of split-course radiation in carcinoma of lung. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *117*, 605–608.
5. HOLSTI, L. R. Clinical experience with split-course radiotherapy; randomized clinical trial. *Radiology*, 1969, *92*, 591–596.
6. LEVITT, S. H. Split-dose approach in radiation therapy. *Rad. Clin. North America*, 1969, *7*, 293–299.
7. LEVITT, S. H., and BOGARDUS, G. R., JR. Advantages and disadvantages of intensive split-dose radiation therapy. In: Frontiers of Radiation Therapy and Oncology. S. Karger AG., Basel, 1968, pp. 163–172.
8. SAMBROOK, D. K. Split-course radiation therapy in malignant tumors. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 37–45.

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DUAL CAMERA STUDIES OF PULMONARY FUNCTION WITH COMPUTER PROCESSING OF DATA*

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PIONEER work with xenon 133 as a radionuclide for evaluation of lung function was carried out by Knipping and associates.[3] Modifications and improvements in procedures for making these measurements were made by several investigators.[2,12,13,16,17] Through their efforts the concepts of the ventilation (V) and perfusion (Q) indices and ventilation-perfusion ratio (V/Q) were originated. The V/Q ratio has important physiologic significance as it relates regional ventilation to blood flow which must be in reasonable balance for adequate gas exchange to take place. Regions of the lung which are adequately ventilated but poorly perfused, as with pulmonary emboli, will have an inadequate gas exchange (elevated V/Q ratio) just as would other areas which might be poorly ventilated but normally perfused (depressed V/Q ratio). This latter situation is usually not found because of a reflex mechanism which decreases blood flow in regions that are poorly ventilated.[15]

In 1966 we introduced the use of a scintillation camera to replace scintillation probes in the detection of xenon 133 distribution in pulmonary function studies.[5] Pulmonary disorders studied by our technique have included pulmonary embolism, obstructive lung disease (cystic fibrosis, bronchiectasis, emphysema, asthma), and primary or metastatic malignancy.[4,7,8,10,11] These studies have also been used in the pre- and postoperative evaluation of patients with either congenital or acquired heart disease which may have caused alterations in either pulmonary perfusion or ventilation or both.[6] Examples of data obtained from 2 patients will be presented here to illustrate our recent addition of a second scintillation camera to permit simultaneous bidirectional viewing of the distribution of radioactive xenon in the lungs.

MATERIAL AND METHOD

Xenon 133 is obtained as a gas in 1 curie ampules on a weekly basis from the General Electric Corporation. The technique for obtaining a sterile saline solution of radioactive xenon (up to 20 mc per ml.) as well as a consideration of radiation dosimetry of this gas has been presented elsewhere.[9] Except for the addition of the second camera and the employment of an on line computer, the technique is similar to that previously reported.[4,6,8] In brief, this involves the intravenous administration of a bolus of 20 mc of xenon 133 in saline for evaluation of pulmonary perfusion. The xenon gas passes from the capillaries into the alveoli and is exhaled into a spirometer from which the patient rebreathes until equilibrium is established. At this time the distribution of xenon is determined and those data are used to obtain corrections for lung volume and geometry of counting. This is followed by washout of the xenon during which time minute volume is measured for further assessment of ventilation. Scintiphotograms are obtained from both cameras throughout the study. Data from both cameras are also digitized and passed on line to a dedicated computer.

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Computer programs have been written to permit readout of regional ventilation and perfusion indices, ventilation-perfusion ratios and washout times.

The 2 scintillation cameras (Radicamera 50 and Radicamera 60, Nuclear Data) are placed against the anterior and posterior chest wall of a patient in the upright position as is shown in Figure 1. Other positions such as supine or prone have also been used. For ease in positioning, one camera has been placed on a specially designed cart.[1] The cameras are connected on line to a computer (Nuclear Data Med. II) *via* dual camera interface.

A 100 MHZ dual ADC is used to digitize the analog camera signals for computer storage. Programs, written in Nutran, may be entered into the system by CRT or teletype terminal, or by paper tape. Data may be displayed by CRT or as a hard copy from the teletype.

The dual camera interface is designed to accept appropriate "X" and "Y" positional signals from the 2 cameras and to route them through the analog-to-digital converters to specific buffer locations in the core memory. Buffer storage of 4096 words (64×64) is available for data entering from each camera. At preselected time intervals, usually 2–3 seconds, the contents of the 2 buffers are transferred to disks. In the rare instance when scintillation events occur in both cameras simultaneously, a priority system within the interface determines which event is processed.[18]

The Med II computer, although relatively small, has sufficient flexibility to permit regional analyses of pulmonary function to be made from 12 lung regions (6 from each lung). Utilization of this computer for dynamic studies of the cardiovascular system has been considered elsewhere.[14]



Fig. 1. The subject is positioned between 2 scintillation cameras for a xenon lung study. The camera on the left is on a mobile cart to permit it to be moved into position.

RESULTS

Data from 2 patient studies will be presented in the following paragraphs to illustrate the technique. The first patient (110) has essentially normal lung function, whereas the second (115) has chronic obstructive pulmonary disease (COPD). Although the computer program permits the selection of 12 areas of each anterior and posterior lung field, 6 areas are used here to simplify the presentation of quantitative data in Tables I and II.

A computer drawn plot of activity in the left midlung field (posteriorly) *versus* time during the study of one of our patients (110) is shown in Figure 2. The (Q) represents that portion of the intravenously injected bolus of xenon 133 which was initially distributed in this region of lung and is indicative of blood flow. The (V_2) represents activity in this lung region at full inspiration after rebreathing of xenon to equilibrium. This was followed by washout of xenon and then by a single deep breath of the radioactive gas (V_1) indicative of regional ventilation. Similar graphs are obtainable for all posterior and anterior regions of the lung as might be selected. A representation of these sequential data of activity *versus* time from the lower lung fields, as seen from the posterior projection of this patient, are shown in Figure 3. The

TABLE I

COMPUTER GENERATED DATA FROM A NORMAL SUBJECT (110). Q IS THE PERFUSION INDEX, V THE VENTILATION WIDTH AND V/Q THE VENTILATION-PERFUSION RATIO. THE $T_{1/2}$ IS THE CLEARANCE HALF TIME IN SECONDS

	POSTERIOR REGIONS OF THE LUNG					
	L. Lower	L. mid	L. upper	R. lower	R. mid	R. upper
Q	1.15	.98	.82	1.13	1.08	.77
V	1.06	.98	1.13	1.04	.94	.89
V/Q	.92	1.00	1.34	.92	.87	1.16
$T_{1/2}$ sec.	25	18	18	27	19	18
	ANTERIOR REGIONS OF THE LUNG					
	L. lower	L. mid	L. upper	R. lower	R. mid	R. upper
Q	1.06	1.10	.73	1.02	1.06	.88
V	1.08	.97	.94	1.08	.96	.90
V/Q	1.02	.88	1.29	1.06	.91	1.02
$T_{1/2}$ sec.	23	17	18	22	18	18

TABLE II

COMPUTER GENERATED DATA FROM A SUBJECT WITH COPD (115). Q IS THE PERFUSION INDEX, V THE VENTILATION INDEX AND V/Q THE VENTILATION-PERFUSION RATIO. THE $T_{1/2}$ IS THE CLEARANCE HALF-TIME IN SECONDS

	ANTERIOR REGIONS OF THE LUNG					
	L. lower	L. mid	L. upper	R. lower	R. mid	R. upper
Q	2.2	1.12	.70	1.11	.74	.77
V	1.52	.88	1.00	1.11	.908	.915
V/Q	.72	.72	1.42	1.00	1.20	1.19
$T_{1/2}$ sec.	67	55	60	67	63	74
	POSTERIOR REGIONS OF THE LUNG					
	L. lower	L. mid	L. upper	R. lower	R. mid	R. upper
Q	1.14	.83	.56	1.24	1.11	.54
V	1.34	.93	.63	1.05	.98	.97
V/Q	1.18	1.12	1.13	.85	.89	1.03
$T_{1/2}$ sec.	76	64	72	72	71	94

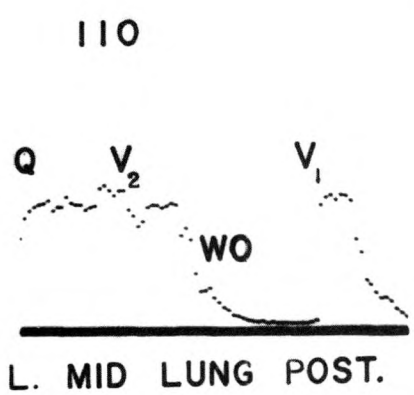


FIG. 2. Computer generated histogram from the normal subject (110) showing counts *versus* time in the left midlung as seen in the posterior projection. (Q), (V_2), and (V_1) indicate the data points which were used to evaluate perfusions, equilibrium, and ventilation. Analysis of washout was started at (WO).

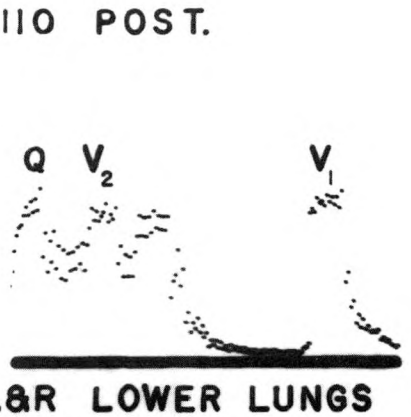


FIG. 3. Computer generated histogram showing counts *versus* time from the left and right lower lung fields as seen in the posterior projection.

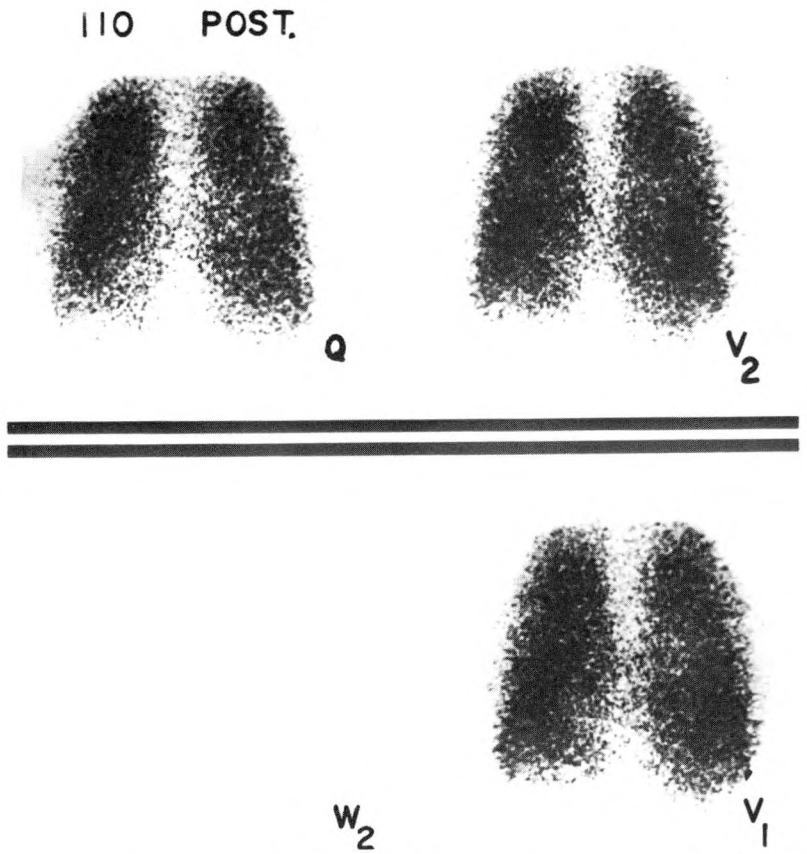


FIG. 4. Camera scintiphotograms from the normal subject (110) showing the distribution of xenon during perfusion (Q), equilibrium (V_2), single breath ventilation (V_1) and later washout (W_2).

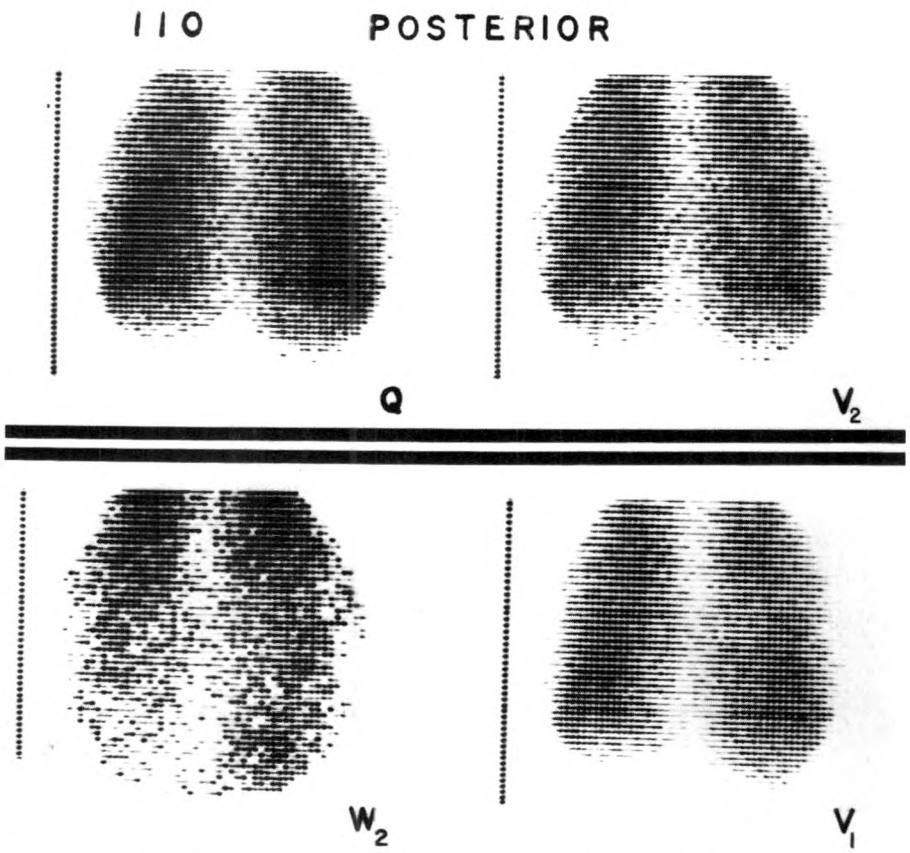


Fig. 5. Digitized computer displayed scintiphotograms from the normal subject (110) showing the distribution of radioactivity during various parts of the study.

2 curves are almost totally superimposed because of nearly identical function of the hemilungs as may also be appreciated from selected scintiphotograms obtained during the course of the study (Fig. 4). The perfusion (Q) shows the gradient of blood flow to be essentially normal, decreasing from base to apex. Washout is rapid and nearly uniform throughout both lungs except for a slight delay in clearance from both lung bases indicative of a minimal component of obstruction to ventilation.

Figure 5 shows selected frames of data displayed on the CRT of the Med II. The digitized format (64×64) is evident. The apparent discrepancy in activity between the picture obtained during washout (W_2) as seen in Figures 4 and 5 is artifactual and relates to poor counting statistics from low counts which was compensated for by a greater intensity setting used in the computerized display. Except for this difference, the similarities in activity distribution for comparable displays in the 2 figures are quite evident.

Computer displays which show the selection of the right and left mid lung regions (anterior view) together with the plots of radioactivity in these regions *versus* time are shown in Figure 6. The inclusion of a small portion of the cardiac silhouette in each of these regions is evident. The plots are nearly identical with those shown in Figures 2 and 3 for other lung regions.

Table 1 shows regional (V) and (Q) indices, V/Q ratios and washout times for this patient. The $T_{1/2}$ values for anterior and posterior regions of the lower lung varied from 23 to 25 seconds on the left and 22 to 27 seconds on the right, confirming a slight

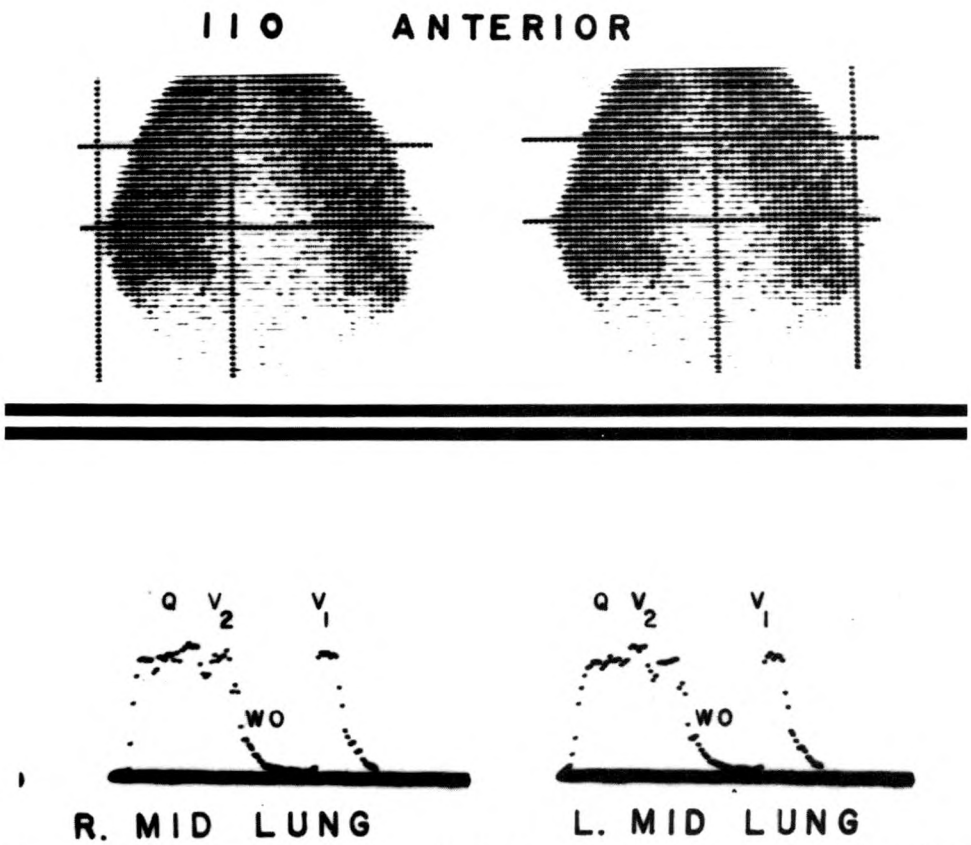


Fig. 6. Computer displayed scintiphotograms with X, Y coordinate markers outlining the regions selected for the mid right and left lungs as seen from the anterior projection. Corresponding time *versus* activity histograms for the regions are also shown.

depression of ventilation in these regions. The remainder of the washout times are within normal limits, as are values obtained for indices and V/Q ratios for both lung fields. The decrease in both (V) and (Q) indices from lung base to apex may be seen in both sets of data although this trend is more evident from the posterior set because they tend to be more consistent than the anterior set. The greater variability in the anterior set appears related to portions of the cardiac "image" being included (Fig. 6).

Table 11 summarizes the regional quantitative data obtained from another patient (115). The variability in (V) and (Q) indices and V/Q ratios is considerably greater than was observed in the study of patient (110) whose data are shown in Table 1. Likewise the clearance half times are prolonged throughout all lung regions (approximately 3 times normal) indicative of the severity and the diffuseness of COPD in this patient.

Figures 7 through 10 show digitized scintiphotograms obtained at various times during the xenon study of this patient. Figure 7 shows computer generated displays of distribution of blood flow as seen from the posterior view. Two somewhat different intensity control settings have been used to display the distribution of blood flow with the display on the right showing an isocontour pattern. Similar representations seen as equilibrium (V_2) and 1 minute into washout (W_2) are shown in Figures 8 and 9, respectively. Figure 10 illustrates with coordinate markers, the selection of 3 regions of the posterior left

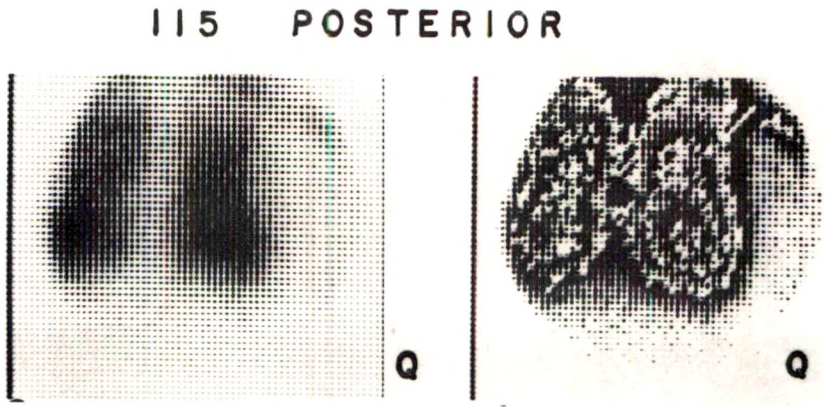


Fig. 7. Computer display of perfusion (Q) distribution of xenon in the subject with COPD (115) seen from the posterior projection. The figure on the right is a contour display showing isocount levels.

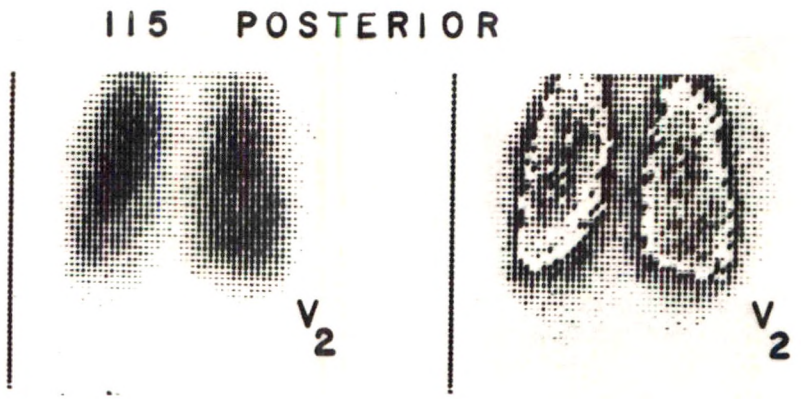


Fig. 8. Computer display showing the distribution of xenon in the lungs as seen from the posterior projection at equilibrium (V_2).

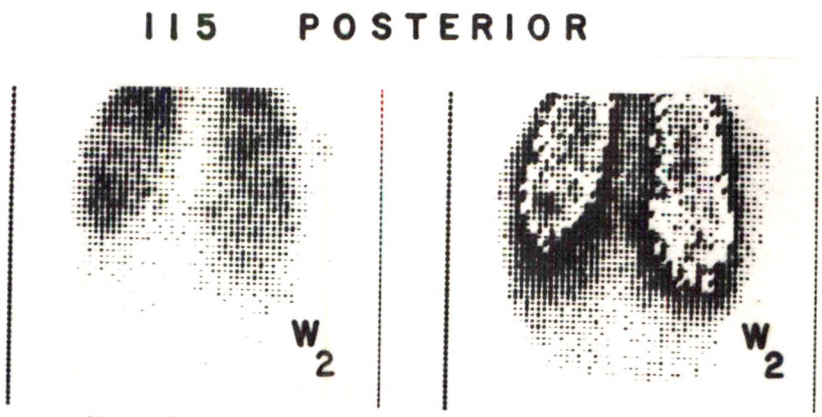


Fig. 9. Computer display of the distribution of xenon during a portion of the washout phase of the study.

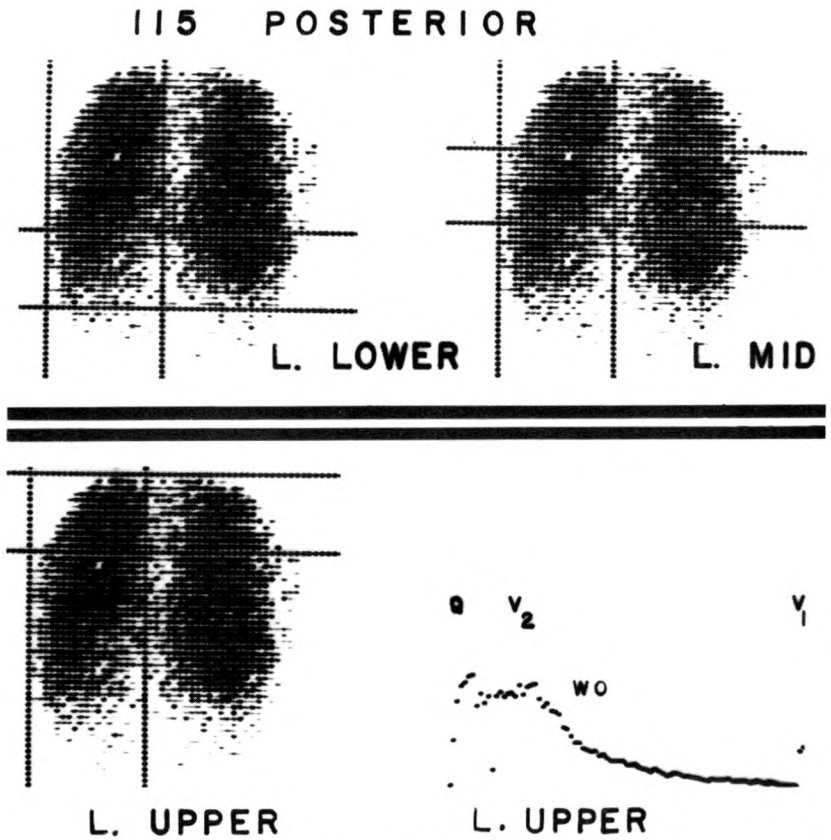


FIG. 10. Computer display with regions outlined by X, Y coordinate markers. A count *versus* time histogram from the left upper region is also indicated.

hemilung. The plot on the lower right corner of this figure quantifies the radioactivity in the upper lung region as a function of time during the study. Figure 11 shows a display (anterior view) obtained during equilibrium (V_2) and late into washout (W_4). The retention of radioactivity in the lungs at 3 minutes into washout

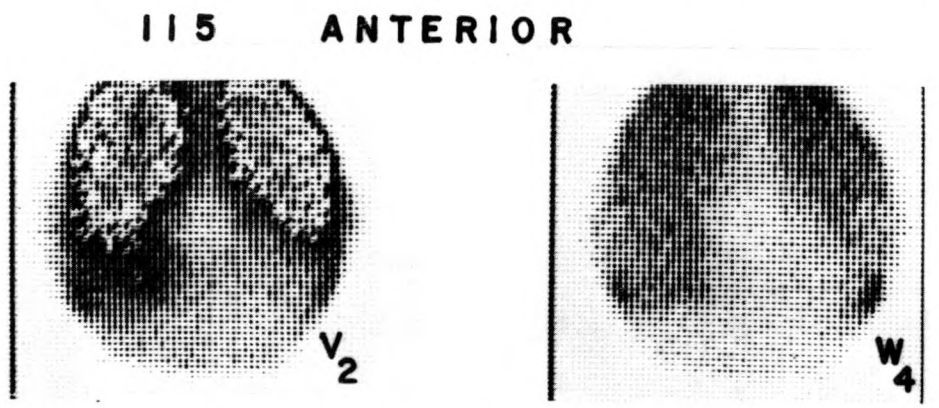


FIG. 11. An isocount contour display of the distribution of activity at equilibrium (V_2) as seen from the anterior projection. The figure on the right is from late washout and shows delayed clearance of xenon from the lungs.

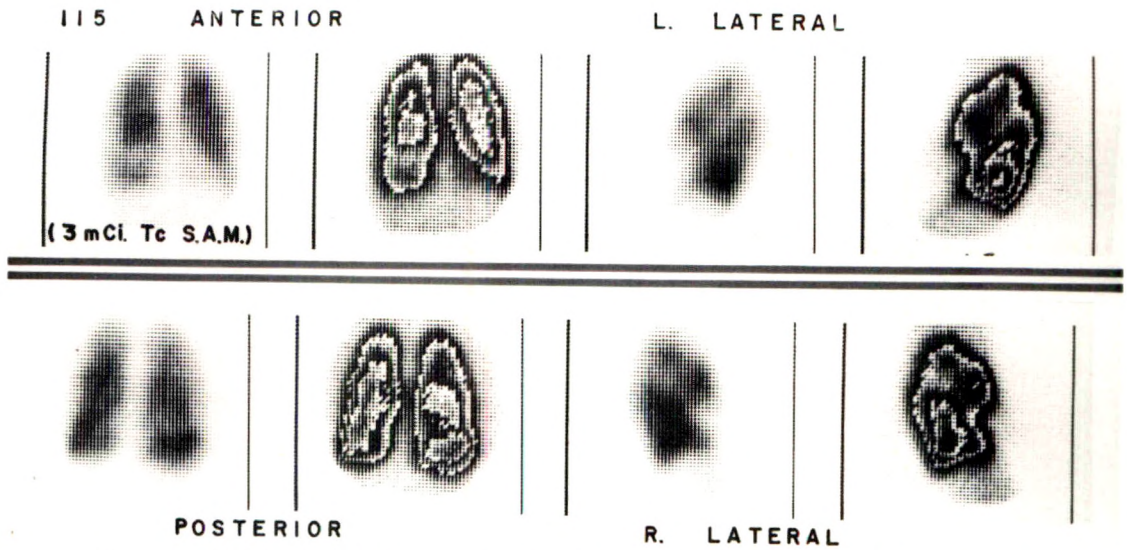


FIG. 12. Computer displayed scintiphotograms showing the distribution of Tc^{99m} SAM in the patient with COPD (115). Isocount contour displays for each projection are included.

confirms COPD as the appropriate diagnosis.

Digitized scintiphotograms obtained from this patient after the administration of 3 mc of Tc^{99m} serum albumin microspheres (SAM) are shown in Figure 12. Two intensity control settings are presented for each view (anterior, left lateral, posterior, and right lateral). Irregularities in perfusion evident in both lungs caused by COPD are not dissimilar from those seen with multiple emboli, indicating the importance of ventilation data when interpreting lung scintiphotograms.

DISCUSSION

The dual camera technique has the potential for providing a significantly better assessment of pulmonary function (blood flow and ventilation) than is provided by a single camera because of the inclusion of a third dimension—depth. This bidirectional approach is of particular importance when low energy gamma emitters, such as xenon 133, are being used because the majority of the gamma rays reaching a detector originate from tissues immediately adjacent to the detector and, as a result, abnormal concentrations of radioactivity (either high or low) that are located in the distal regions of the structure being imaged may go undetected. Such is the case in the assessment of pulmonary emboli which, although most frequently found posteriorly in the lung bases, may be located at other sites which may then require other than a single posterior view of the lungs for optimal detection.

Our standard technique for assessing pulmonary emboli has been to obtain several scintiphotographic views of the lungs following intravenous administration of Tc^{99m} albumin particles (either albumin aggregates or microspheres). Because this approach provides no significant information on ventilation which together with perfusion is regionally altered in patients with obstructive lung disease, we have looked at the regional washout of xenon 133 gas from these patients as well. This latter technique permits a differentiation to be made between abnormalities of perfusion associated with emboli from those seen in cases of obstructive lung disease. With the availability of the dual-camera approach, we are finding that the use of xenon 133 alone is sufficient to provide adequate data to make a definitive differential diagnosis

between pulmonary emboli and obstructive lung disease.

SUMMARY

Xenon 133 and 2 scintillation cameras arranged to view both anterior and posterior lung fields are being used routinely for evaluation of pulmonary function. Scintiphotograms are obtained from both cameras throughout the course of both perfusion and ventilation portions of the study. In addition, data from both cameras are digitized and stored on line in a dedicated computer for further analysis.

Computer processing of data permits quantitative information to be obtained on perfusion, ventilation and clearance of the radioactive gas from selected regions of the anterior and posterior lung fields. Ventilation-perfusion ratios and washout times are likewise calculated by computer.

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REFERENCES

1. Ashburn, W. L. Scanning approaches in evaluation of heart disease. In: Clinical Uses of Radionuclides: Critical Comparison with Other Techniques. ORAU Symposium No. 13, Nov., 1971, pp. 579–593.
2. Ball, W. C., Jr., Stewart, P. B., Newsham, L. G. S., and Bates, D. V. Regional pulmonary function studied with xenon 133. *J. Clin. Invest.*, 1962, *41*, 519–531.
3. Knipping, H. W., Bolt, W., Valentin, H., Venrath, H., and Endler, P. Eine neue Methode zur Prüfung der Herz und Lungenfunktion; die regionale Funktionanalyse in der Lungen und Herzklinik mit Hilfe des radioaktiven Edelgases Xenon-133. *Deutsche med. Wchnschr.*, 1955, *80*, 1146–1147.
4. L'Heureux, P., Loken, M. K., Ponto, R., and Ebert, R. Regional evaluation of pulmonary function in chronic obstructive pulmonary disease. *Radiology*, 1971, *100*, 107–112.
5. Loken, M. K. Radioisotopic techniques for evaluation of pulmonary disease. *J. Nuclear Med.*, 1966, *7*, 354–355.
6. Loken, M. K. Evaluation of pulmonary function and blood flow using xenon-133 and scintillation (Anger) camera. In: Angiography /Scintigraphy. Symposium of the European Association of Radiology, Mainz, Germany, Oct., 1970, pp. 187–193.
7. Loken, M. K. Camera studies of lung ventilation and perfusion. *Seminars Nuclear Med.*, 1971, *1*, 229–245.
8. Loken, M. K., Kronenberg, R. S., Williams, L. E., and Ponto, R. A. Radioisotopic studies evaluating lung function. In: Clinical Uses of Radionuclides: Critical Comparison with Other Techniques. ORAU Symposium No. 13, Nov., 1971, pp. 506–521.
9. Loken, M. K., and Kush, G. S. Handling, uses and radiation dosimetry of 133-Xe. In: Medical Radionuclides: Radiation Dose and Effects. ORAU Symposium, Oak Ridge, Tenn., Dec. 8–11, 1969. USAEC Div. of Tech. Info. Ext., Oak Ridge, Tenn., 1970, pp. 253–270.
10. Loken, M. K., Kush, G. S., and Andersen, C. F. Use of computer for processing pulmonary function data obtained with scintillation (Anger) camera. In: Computer in Radiology. Symposium proceedings, Brussels, Belgium, Sept. 17–20, 1969. S. Karger AG, Basel, 1970, pp. 560–567.
11. Loken, M. K., Medina, J. R., Lillehei, J. P., L'Heureux, P., Kush, G. S., and Ebert, R. V. Regional pulmonary function evaluation using xenon-133: scintillation camera and computer. *Radiology*, 1969, *93*, 1261–1266.
12. Mannell, T. J., Prime, F. J., and Smith, D. W. Practical method of using radioactive xenon for investigating regional lung function. *Scandinav. J. Resp. Dis.*, 1966, Suppl. 162, pp. 41–57.
13. Matthews, C. M. E., Dollery, C. T., Clark, J. C., and West, J. B. Radioactive gases. In: Radioactive Pharmaceuticals. AEC Symposium Series No. 6 (Conf-65111), Oak Ridge, Tenn., 1965. USAEC Div. of Tech. Info. Ext., Oak Ridge, Tenn., 1966, pp. 567–592.
14. Ponto, R. A., Goldberg, M. E., and Loken, M. K. Investigation of cardiac dynamics using scintillation camera and dedicated computer. In: Proceedings of Second Symposium on Sharing of Computer Programs and Technology in Nuclear Medicine. USAEC Conference—720480. ORNL. Oak Ridge, Tenn., April 21–22, 1972, pp. 185–198.
15. Wagner, H. N., Jr., Tow, D. E., Lopez-Majano, V., Chernick, V., and Twining, R. Factors influencing regional pulmonary blood in man. *Scandinav. J. Resp. Dis.*, 1966, Suppl. 62, pp. 59–72.

16. West, J. B. Ventilation, Blood Flow and Gas Exchange. Blackwell, Oxford, 1965.
17. West, J. B., Fowler, K. T., Hugh-Jones, P., and O'Donnell, T. V. Measurement of ventilation-perfusion ratio inequality in lung by analysis of single expirate. *Clin. Sc.*, 1957, *16*, 529–547.
18. Williams, L. E., Ponto, R. A., Goldberg, M. E., Payne, J. T., and Loken, M. K. Dual camera-dual isotope data acquisition system for nuclear medicine studies. In: Proceedings of the Third Symposium on Sharing of Computer Programs and Technology in Nuclear Medicine. Miami, Fla., June 15–16, 1973, Conference 730627. USAEC Tech. Info. Cen., Oak Ridge, Tenn., 1973, pp. 123–132.

EXTENDED MANTLE RADIOTHERAPY IN HODGKIN'S DISEASE AND MALIGNANT LYMPHOMA*

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IN the radiation treatment of Hodgkin's disease (H.D.) and malignant lymphoma (M.L.), most institutions have used "classic mantle" and inverted-Y fields. We have evolved a field called the "extended mantle," which is defined as a "classic mantle" that includes the upper portion of an inverted-Y field. The mediastinal portion of the classic mantle is extended into the abdomen to the level of the intervertebral disk between L2 and L3, so that the upper abdominal lymph nodes and the entire spinal cord are included within a single field. We used the extended mantle in the treatment of H.D. and M.L. according to the stage of the disease: as a single radiation field or in combination with a "lower inverted-Y" field for total lymphoid irradiation. In this combination, the extended mantle eliminates splicing of 2 fields at the level of the diaphragm. The rationale for avoiding such splicing is the prevention of: (1) overlap of the 2 fields which can result in radiation injury to the spinal cord; and (2) excessive separation of the 2 fields which can result in local recurrence of disease. A low incidence of both complications, *i.e.*, radiation-induced transverse myelitis and local recurrence of disease at the splice, has been reported by major treatment centers;[5,9] they probably occur more commonly than reported, however.

The purpose of this report is to show, on the basis of a retrospective study of 95 cases, that patients tolerate radiotherapy to an extended mantle field, and that treatment results are satisfactory with its use. Our experience with such a field is analyzed in terms of technique, relapse, survival, morbidity, hematologic tolerance, and mortality.

MATERIAL AND METHOD

The case material for this report was obtained by retrospective analysis of hospital charts and radiotherapy records of 95 patients who had been treated with an extended mantle field since December, 1968. There were 68 patients with Hodgkin's disease and 27 with malignant lymphoma. Sixty-one of these patients completed total lymphoid irradiation after treatment with the extended mantle field. Eight patients had received radiotherapy at other institutions before being treated by us.

The staging classification introduced at the Ann Arbor Symposium,[1] the histopathologic classification for Hodgkin's disease recommended by Lukes *et al.*,[10] and the histopathologic classification for malignant lymphoma recommended by Rappaport[13] were used. All patients were staged by exploratory laparotomy, which included splenectomy and biopsies of the liver, bone marrow, and the following lymph nodes: porta hepatis, celiac, splenic, hilar, paraaortic, and iliac. Time intervals used to report end results were calculated

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from the date of staging laparotomy to the date of recurrence, or to the date of the most recent follow-up. No patient was lost to follow-up. The status of each case was recorded for variable intervals ranging from 1 to 4 years. Failures were classified as true or local recurrence (TR, LR) when disease occurred in an irradiated field following an attempt at curative radiotherapy; as regional recurrence (RR) when disease occurred at the junction between irradiated and nonirradiated tissues or outside the previously irradiated field, but confined to the same side of the diaphragm; as transdiaphragmatic recurrence (TDR) when disease appeared on the side of the diaphragm opposite to that originally treated; and as extralymphatic recurrence (EL) when disease appeared in extranodal or extrasplenic sites.[19]

TECHNIQUE

The "extended mantle" field is formed by the joining of the mediastinal portion of a "classic mantle" ("Maltese cross") field with the upper portion of an inverted Y. The outline of an extended mantle field on the skin of a patient under treatment is shown in Figure 1; the Cerrobend[12] shield for the field is illustrated in Figure 2; and a port film of an extended mantle field is shown in Figure 3. Patients were treated with 2 mev. roentgen rays from a Van de Graaff generator at a skin-source distance of 175 cm. The average field length was approximately 54 cm. The original technique as previously reported[11] has not changed basically, but several modifications have resulted from our more extensive experience. They can be categorized as changes in: (1) geometry of field; (2) shielding; (3) time, dose, and fractionation; and (4) simulation.

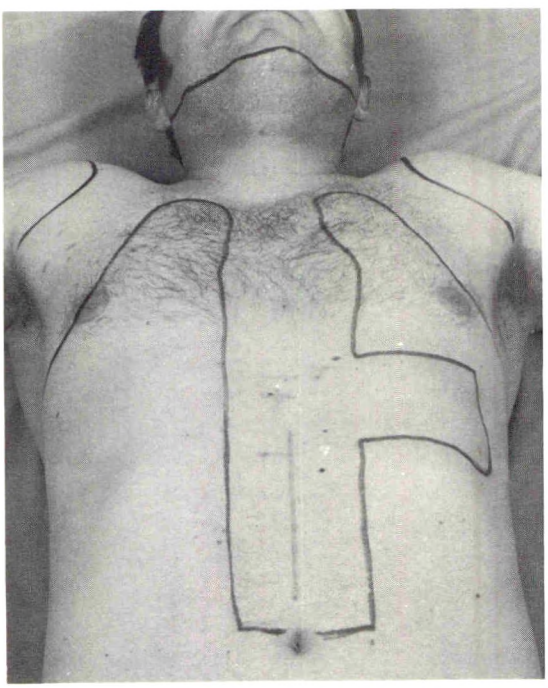


Fig. 1. Outline of extended mantle field on a patient.

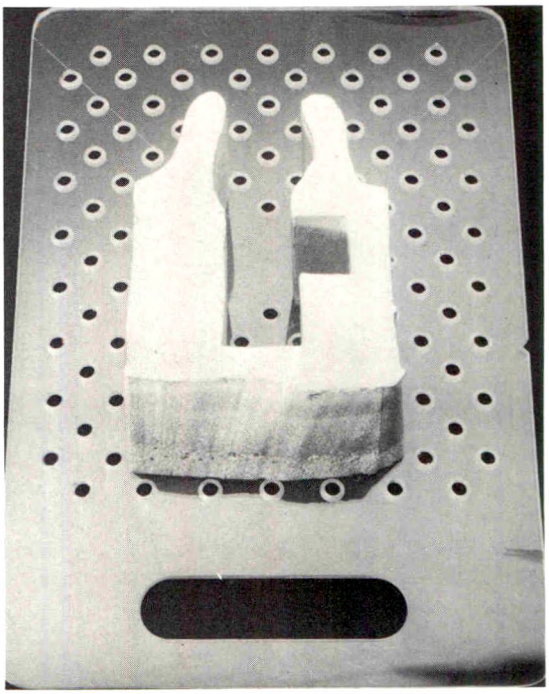


Fig. 2. Shield for extended mantle field.

1. *Geometry of field.* In the presence of massive cervical lymphadenopathy extending to the mastoid process, the upper margin of the portal must be placed high in order to encompass the entire involved area (Fig. 4). Such placement creates some dosimetric problems, since body thickness increases as one proceeds cephalad from

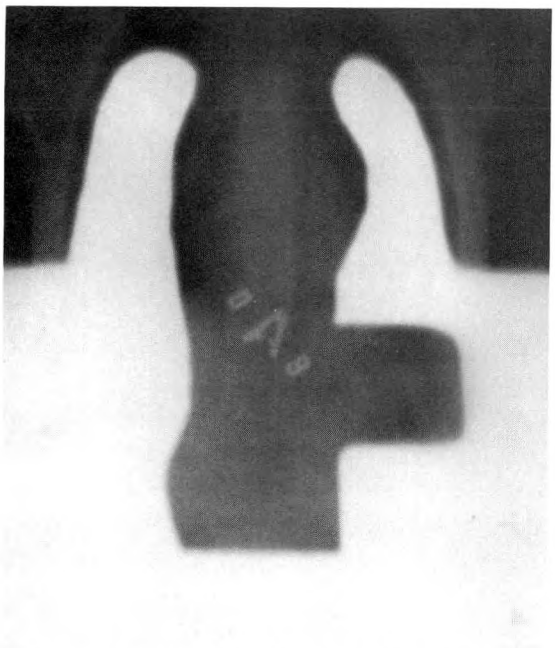


FIG. 3. Verification film of extended mantle field.

neck to mandible and skull. Every effort is made to extend the head maximally during treatment so that the mandible is elevated and the sagittal diameter in this region is reduced. In tall adult males, use of a high upper margin makes it difficult to place the lower margin of the portal at the level of L_2-L_3 because of restrictions in field length. In such cases it may be necessary to use opposed right and left lateral fields that include the head and the upper neck, and to create a cervical splice with the extended mantle field which is AP-PA. The splice is moved 2 cm. upward or downward midway in the treatment to prevent overlap due to possible setup error. This technique has been used in 1 case of histiocytic lymphoma and in 1 case of H.D.; both cases presented with marked cervical lymphadenopathy and prominent oropharyngeal masses extending upward into the nasopharynx. The base of the skull and the retroauricular and suboccipital lymph nodes were included in the portal. The presence of massive disease in the neck and oropharynx with upward extension has been of particular concern because of the difficulty of treating a regional recurrence in this area without exceeding spinal cord tolerance.

When a patient has a large upper abdominal tumor, the abdominal portion of the field is enlarged to encompass known disease. Metallic clips placed around the tumor mass[4] are essential for the correct outline of the enlarged portal. In such cases, one kidney is treated in excess of tolerance. Radiation nephritis with secondary hypertension did not develop in 4 of our patients who have been followed for a minimum of 8 months. A third patient treated in this manner died from extralymphatic metastases before sufficient time had elapsed for the possible development of hypertension.

Involvement of porta hepatis lymph nodes has been found in a significant number of cases.[3] These lymph nodes are always marked by the surgeon with metallic clips that can be visualized on port films. In the majority of cases, the porta hepatis lymph nodes lie near the midline, so that an abdominal extension on the right side as shown in Figure 3 is not necessary.

In patients with Stage III H.D. or M.L., irradiation of the extended mantle field is followed after 1 month of rest by radiation

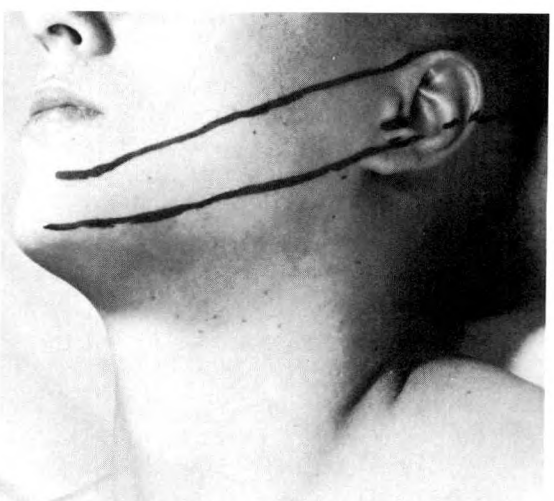


FIG. 4. Upper margins for extended mantle field. Lower line used routinely; upper line used for prominent cervical lymphadenopathy. Major portions of parotid glands are included.

delivered to a "lower inverted-Y" field (which, in fact, represents an "inverted V").

2. *Shielding*. Since the dose for an extended mantle field is calculated in relation to the mid-mediastinum, the cervical spinal cord is necessarily exposed to an increased dose. Using a densitometer to measure exit dose on films of extended mantles, Haus *et al.*[6] have shown that the dose in the neck may exceed that in the mediastinum by a considerable amount. Therefore, a 50 per cent cervical attenuation block is used posteriorly in all cases. Compensators eliminate the discrepancy between doses delivered to the mediastinum and the neck, but they were not used routinely in this series.

In the majority of cases, a 50 per cent attenuation shield was used anteriorly to protect the larynx. In patients with massive cervical lymphadenopathy, however, we removed this shield because persistent submental disease had been noted in 2 patients treated earlier, in whom additional treatment with electrons was necessary.

Shoulder joints are routinely shielded, particularly in cases that present with recurrent disease in the supraclavicular fossae or axillae after radiation therapy given in another institution.

3. *Time, dose, and fractionation*. Because of the large size of the extended mantle field, there has been a tendency to treat some patients with daily doses of 150 instead of 200 rads. Seventy-six patients received 200 rads per day and 1,000 rads per week, whereas the remaining 19 were treated with 150 rads per day. Patients with H.D. and M.L., lymphocytic type, have received a total dose of 4,000 rads. Patients with M.L., mixed lymphocytic-histiocytic (composite) and histiocytic type have received 4,500 to 5,000 rads total dose, with occasional boost doses of 1,000 rads in 1 week to large residual lesions. Opposed fields have been used in all cases, and each field has been treated daily to diminish subcutaneous fibrosis and pericarditis.

4. *Simulation*. For the initial cases in this series, the mantle and abdominal portions of the extended mantle field were set up separately and then joined together. Errors in the location of the splenic pedicle extension with respect to the intraabdominal clips placed along the splenic artery were common with this technique. Based on this experience, the whole field is now simulated using the exact skin-source distance and centralizing the source with respect to the entire field. The cassette holder is simply moved superiorly and inferiorly, and films exposed in each location are joined together at common anatomic landmarks. The field is drawn on the joined 14×17 inch films, and from this drawing a shield (Fig. 2) for the entire perimeter of the field is constructed by means of a hot wire apparatus.[12] With the "cross method,"[16] the shield is then positioned over the patient, and the outline of the shield on the skin is confirmed in relation to internal anatomy by means of radiographs. When the external landmarks coincide with the internal anatomy, the skin marks are tattooed. Sequential films placed under the patient during the entire treatment to record the exit dose have shown that the field can be duplicated with accuracy in most patients over the entire course of treatment.

RESULTS

HODGKIN'S DISEASE

Type of radiation therapy versus stage (Table 1). All 68 patients with H.D. completed extended mantle irradiation; among the 28 cases with Stage I and II disease, 25 had extended mantle irradiation alone and 3 had total lymphoid irradiation. Among the 23 cases with Stage IIIA disease, 2 had extended mantle irradiation alone and 21 had total lymphoid irradiation, and among the 17 with Stage IIIB, 2 had extended mantle irradiation alone and 15 had total lymphoid irradiation.

Adjuvant chemotherapy. None of the 28 cases of Stage I and II H.D. received chemotherapy. Five of the 23 cases with

TABLE I

TYPE OF THERAPY BY STAGE IN 68 CASES OF HODGKIN'S DISEASE

Stage	Extended Mantle Alone	Total Lymphoid Irradiation	Total Lymphoid Irradiation Followed by "COPP"
I and II	25	3	0
IIIA and III_SA	2	16	5
IIIB and III_SB	2	4	11

COPP = cyclophosphamide, vincristine, procarbazine, prednisone.

Stage IIIA disease and 11 of 17 cases with Stage IIIB disease received quadruple cyclic chemotherapy (cyclophosphamide, vincristine, procarbazine, and prednisone = "COPP") 3 months after the completion of total lymphoid irradiation (Table I).

Current status of patients. Of the 68 patients with Hodgkin's disease who completed extended mantle irradiation, 49 are alive without disease, 9 are alive with disease, and 10 are dead with disease (Table II). One patient in the last category died of pulmonary embolism near the termination of therapy. As indicated in Table II, there were 6 patients who were suboptimally treated with radiation elsewhere and who were in relapse at the time of their referral to us. These patients underwent surgical staging and were re-irradiated with curative intent. Three of these patients are alive without disease, 1 is alive with disease, and 2 are dead with disease.

Table III shows the relationships of stage and histopathology to the status of the patient. There were 50 patients with nodular sclerosis, 9 with mixed cellularity, 5 with lymphocytic predominance, 2 with lymphocytic depletion, and 2 who were unclassifiable. Twenty-three patients were Stage IA or IIA, 5 were Stage IB or IIB, 23 were Stage IIIA or III_SA, and 17 were Stage IIIB or III_SB. Of 10 patients dead with disease, 8 were Stage IIIA or IIIB.

Analysis of failures. There were 23 failures among the 68 patients with H.D. who completed extended mantle irradiation (Table IV). Five failures (Cases 2, 3, 8, 14, and 15) were true recurrences not associated with extralymphatic disease; 4 (Cases 4, 5, 9, and 16) were true recurrences associated with extralymphatic disease; 1 (Case 10) was a regional (marginal) recurrence associated with extralymphatic disease; 2 (Cases 12 and 21) were regional recurrences or extensions of disease to unirradiated pelvic lymph nodes; 2 (Cases 11

TABLE II

HODGKIN'S DISEASE

FOLLOW-UP OF EXTENDED MANTLE IRRADIATION ALONE (29 CASES) AND EXTENDED MANTLE IRRADIATION PLUS LOWER INVERTED-Y IRRADIATION (39 CASES)

ALL STAGES

Status	Interval (years)				
	0–1	1–2	2–3	3–4	Total Patients
Alive without disease	6 (1)*	13 (1)	17 (1)	13	49†
Alive with disease	2	2	3	2 (1)	9
Dead with disease	3	3	4 (2)	0	10‡
Subtotal	11	18	24	15	68

* Numbers in parentheses indicate number of patients previously treated with irradiation who were qualified to receive extended mantle irradiation with curative intent.

† This total includes 3 patients who developed a true recurrence (2 in the axilla, 1 in the submental region) and 1 patient who developed extension of disease to untreated pelvic lymph nodes; all were given additional radiotherapy and have been free of disease from 6 to 30 months.

‡ Includes 1 patient who died of pulmonary embolism near termination of therapy.

TABLE III

HODGKIN'S DISEASE

STATUS OF PATIENT IN RELATION TO STAGE AND HISTOPATHOLOGY (68 CASES)

		Pathologic Stage														
		IA and IIA			IB and IIB			IIIA and IIIsA			IIIB and IIIsB			Total		
Histopathology	Status	AsD	AcD	DcD	AsD	AcD	DcD	AsD	AcD	DcD	AsD	AcD	DcD	AsD	AcD	DcD
Nodular sclerosis (50 patients)	AsD	17			1			10			6			34		
	AcD		1			1			2			3			7	
	DcD			1[1]			1			2			5			9
Mixed cellularity (9 patients)	AsD	2			2			2			1			7		
	AcD								1			1			2	
	DcD															
Lymphocytic predominance (5 patients)	AsD	1						4						5		
	AcD															
	DcD															
Lymphocytic depletion (2 patients)	AsD	1												1		
	AcD															
	DcD									1						1
Unclassifiable (2 patients)	AsD							1[2]			1[3]			2		
	AcD															
	DcD															
Subtotal	AsD	21			3			17			8			49		
	AcD		1			1			3			4			9	
	DcD			1			1			3			5			10
Total		23			5			23			17			68		

AsD = Alive without disease.
AcD = Alive with disease.
DcD = Dead with disease.

[1] Died of pulmonary embolism near termination of therapy (no autopsy permitted).
[2] Hodgkin's disease, Lennert's type.
[3] Nodular sclerosis and mixed cellularity.

and 17) were regional recurrences or extensions of disease to upper extremities, associated with extralymphatic disease; 8 (Cases 6, 7, 13, 18, 19, 20, 22, and 23) had extralymphatic disease unassociated with true or regional recurrences; and 1 (Case 1) died near the termination of therapy. Details for each failure are shown in the footnote to Table IV.

Radiation salvage. Four patients alive without disease (Table II), who were radiation failures after extended mantle irradiation, were successfully retreated with curative doses of radiation with local control of the recurrent disease for periods from 6 to 30 months. Two of these failures had true recurrences in the axilla; 1 had a true recurrence in the submental triangle; and 1 had a transdiaphragmatic recurrence with disease in unirradiated pelvic lymph nodes.

Two patients with true recurrences associated with extralymphatic disease were retreated with curative intent and were locally controlled for 9 and 10 months, respectively. One is alive with disease, and the other is dead with disease.

There were 3 patients with failures in the form of regional recurrences associated with extralymphatic disease. These patients were irradiated again with curative intent; local control was achieved and maintained for 7, 14, and 30 months, respectively. Two of these patients are alive with disease, and 1 is dead with disease.

Relapse-free rates. Figure 5 is an actuarial curve showing relapse-free rates at 6 month intervals for 25 patients with Stage I and II H.D. who received extended mantle irradiation alone. Three Stage I and II cases were excluded from this analysis because they received total lymphoid irradiation

TABLE IV

HODGKIN'S DISEASE

ANALYSIS OF FAILURES BY STAGE AFTER EXTENDED MANTLE IRRADIATION ALONE (6 CASES)* OR AFTER TOTAL LYMPHOID IRRADIATION (17 CASES)

Pathologic Stage	Interval in Years			Total Failures
	0–1	1–2	2–3	
	Number of Patients (*Case No.*)			
IA and IIA	1(*1*)	2(*2, 3*)	0	3
IB and IIB	0	2(*4, 5*)	0	2
IIIA and IIIsA	2(*6–7*)	4(*8–11*)	1(*12*)	7
IIIB and IIIsB	8(*14–20*)	3(*21–23*)	0	11
Subtotal	11	11	1	23

Case No.	Code†	
(*1*)	D	Died of pulmonary embolism near termination of therapy.
(*2*)	TR	Axillary recurrence irradiated with local control for 1 year.
(*3*)	TR	Diseased mediastinal node removed during pericardiectomy.
(*4*)	TR	Submental recurrence irradiated with local control.
	EL	Osseous metastases, left knee; palliated with additional radiation.
(*5*)	TR	Axillary recurrence.
	EL	Lung, pericardium, kidney.
(*6*)	EL	Liver.
(*7*)	EL	Breast, lung, liver.
(*8*)	TR	Left neck recurrence treated with radiation; since status of recurrence is unknown, this patient is classified as alive with disease.
(*9*)	TR	Pelvic recurrence in previously irradiated pelvis. Not irradiated a second time.
	EL	Pleural and rib metastases. Irradiated with local control for 9 months.
(*10*)	RR	Recurrence in parotid at upper end of extended mantle. This was irradiated with local control until death 7 months later, when the patient died from extralymphatic disease.
	EL	Liver, bone marrow.
(*11*)	RR	Extension to right arm. This was irradiated with control for 14 months.
	EL	Subcutaneous nodule right chest wall, irradiated with local control for 14 months.
(*12*)	RR	Extension to untreated pelvic lymph nodes, which were irradiated with local control for 6 months.
(*13*)	EL	Liver.
(*14*)	TR	Submental recurrence irradiated with local control for 28 months.
(*15*)	TR	Left axillary recurrence irradiated with local control for 6 months.
(*16*)	TR	Recurrence in neck irradiated for palliation.
	EL	Osseous (femur and base of skull) with cranial nerve involvement.
(*17*)	RR	Extension to left arm irradiated with local control for 30 months.
	EL	Subcutaneous chest wall nodule irradiated with local control for 30 months.
(*18*)	EL	Osseous disease (T-12).
(*19*)	EL	Liver and bone marrow.
(*20*)	EL	Liver.
(*21*)	RR	Extension to unirradiated pelvic lymph nodes in patient who refused treatment after extended mantle. Alive with disease.
(*22*)	EL	Liver.
(*23*)	EL	Osseous disease (T-10).

* Cases (*1, 2, 3, 5, 12*), and (*21*) received extended mantle irradiation alone; the remainder received total lymphoid irradiation (extended mantle followed by lower inverted-Y).

† Key: TR = True recurrence.
RR = Regional recurrence.
EL = Extralymphatic.
D = Death.

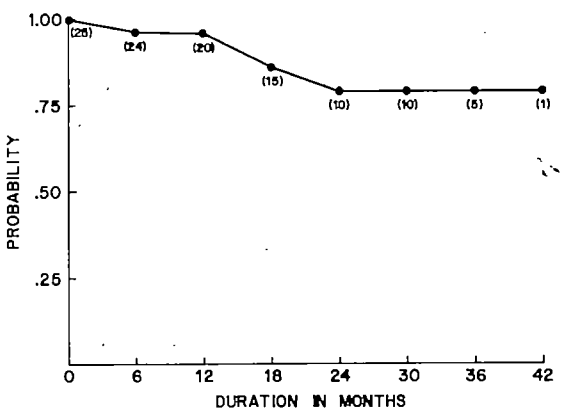


FIG. 5. Actuarial curve showing the relapse-free rates at 6 month intervals for 25 patients with Stage I and II Hodgkin's disease who received extended mantle irradiation alone. The curve was calculated according to the method of Kaplan and Meier.[8] The numbers in parentheses represent number of cases at risk at the start of each interval.

(Table I). The relapse-free rate for Stage I and II disease after extended mantle therapy was 0.79 at 24 months.

Figure 6 is an actuarial curve showing relapse-free rates at 6 month intervals for 21 patients with Stage IIIA H.D., who received total lymphoid irradiation. Two patients with Stage IIIA disease were excluded from this analysis because they received extended mantle irradiation alone (Table I). The relapse-free rate for Stage IIIA disease after total lymphoid irradiation was 0.69 at 24 months. In an attempt to determine the effect of adjuvant chemotherapy on these cases, we calculated separate curves for 5 patients who received chemotherapy and for 16 who did not. There were no relapses among those who received chemotherapy, but among those without chemotherapy the relapse-free rate was 0.60 at 24 months.

An actuarial curve showing relapse-free rates at 6 month intervals for 15 patients with Stage IIIB H.D. who had total lymphoid irradiation is shown in Figure 7. Two patients with Stage IIIB disease were excluded from this analysis because they received extended mantle irradiation alone

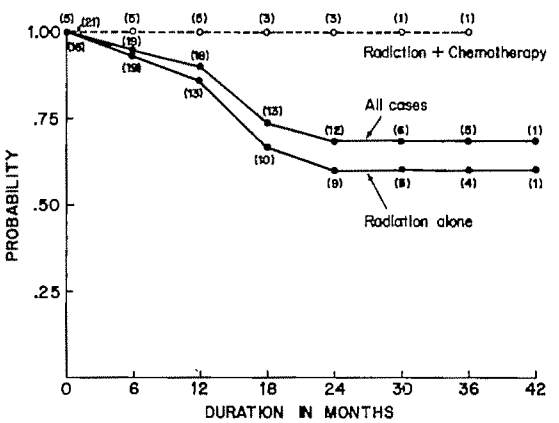


FIG. 6. Actuarial curves showing relapse-free rates at 6 month intervals for 21 patients with Stage IIIA or IIIsA Hodgkin's disease who received total lymphoid irradiation, for 5 patients who also received chemotherapy, and for 16 patients who did not receive chemotherapy. The curves were calculated according to the method of Kaplan and Meier.[8] The numbers in parentheses represent number of cases at risk at the start of each interval.

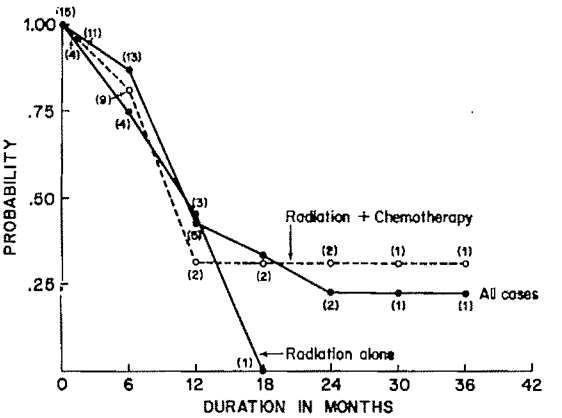


FIG. 7. Actuarial curves showing relapse-free rates at 6 month intervals for 15 patients with Stage IIIB or IIIsB Hodgkin's disease who received total lymphoid irradiation, for 11 patients who also received chemotherapy, and for 4 patients who did not receive chemotherapy. The curves were calculated according to the method of Kaplan and Meier.[8] The numbers in parentheses represent number of cases at risk at the start of each interval.

(Table I). The relapse-free rate for Stage IIIB disease after total lymphoid irradiation was 0.23 at 24 months. Separate curves were calculated for 11 cases who received chemotherapy and for 4 cases who did not. The patients who received chemotherapy had a relapse-free rate of 0.31 at 18 months, whereas all 4 patients who did not receive chemotherapy had relapsed at that time.

NON-HODGKIN'S LYMPHOMA

Types of radiation therapy versus stage. Among 13 patients with Stage I and II disease, 5 received extended mantle irradiation alone and 8 received total lymphoid irradiation. All 14 patients with Stage III disease received total lymphoid irradiation.

Current status of patients (Table V). Of 27 patients with non-Hodgkin's lymphoma who completed extended mantle therapy, 16 are alive without disease, 2 are alive with disease, and 9 are dead with disease (Table V). As indicated in the table, 2 patients previously treated suboptimally with radiation elsewhere were surgically staged here, and were then re-irradiated with curative intent. One is alive without disease and the other is dead with disease. Twenty patients were followed for periods

TABLE V

NON-HODGKIN'S LYMPHOMA

FOLLOW-UP OF EXTENDED MANTLE IRRADIATION ALONE (5 CASES) AND EXTENDED MANTLE PLUS LOWER INVERTED-Y IRRADIATION (22 CASES)

ALL STAGES

Status	Interval (Years)				Total Patients
	0–1	1–2	2–3	3–4	
Alive without disease	3	7	5(1)*	1	16
Alive with disease	2	0	0	0	2
Dead with disease	5(1)*	3	1	0	9
Subtotal	10	10	6	1	27

* Number in parentheses indicates number of patients previously treated with radiation who were qualified to receive extended mantle irradiation with curative intent.

of less than 2 years, whereas 7 were followed for 2 to 4 years. Eight of 9 patients dead with disease died during the first 2 years.

Table VI shows the relationship of stage and histopathology to the status of the patient. There were 14 patients with diffuse histiocytic, 4 with diffuse lymphocytic, 4 with nodular lymphocytic, 2 with diffuse composite, and 3 with nodular composite lymphoma. Thirteen patients were Stage IA or IIA, 11 were Stage IIIA or IIIsA, and 3 were Stage IIIB or IIIsB. Seven of 9 patients dead with disease were Stage IIIA or IIIB; 4 of them had histiocytic lymphoma.

Analysis of failures. Among 27 patients with non-Hodgkin's lymphoma who completed extended mantle irradiation, 11 were considered radiation failures (Table VII). All 11 had extralymphatic disease.

TABLE VI

NON-HODGKIN'S LYMPHOMA

STATUS OF PATIENTS IN RELATION TO STAGE AND HISTOPATHOLOGY (27 CASES)

		Pathologic Stage											
		IA and IIA			IIIA and IIIsA			IIIB and IIIsB			Total		
Histology	Status	AsD	AcD	DcD	AsD	AcD	DcD	AsD	AcD	DcD	AsD	AcD	DcD
Histiocytic diffuse	AsD*	7†									7		
	AcD		1									1	
	DcD			2			2			2			6
Lymphocytic diffuse	AsD	2									2		
	AcD					1						1	
	DcD						1						1
Lymphocytic nodular	AsD												
	AcD				4						4		
	DcD												
Composite diffuse	AsD	1									1		
	AcD												
	DcD						1						1
Composite nodular	AsD				2						2		
	AcD												
	DcD									1			1
Subtotal	AsD	10			6						16		
	AcD		1			1						2	
	DcD			2			4			3			9
Total		13			11			3			27		

* For abbreviations, see Table III.

† One case with histopathology of histiocytic lymphoma and Hodgkin's disease.

TABLE VII

NON-HODGKIN'S LYMPHOMA

ANALYSIS OF FAILURES BY STAGE AFTER EXTENDED MANTLE IRRADIATION ALONE (1 CASE)* AND TOTAL LYMPHOID IRRADIATION (10 CASES)

Pathologic Stage	Interval in Years		Total Failures
	0–1	1–2	
	Number of Patients (*Case No.*)		
I and IIA	3(*1–3*)	0	3
I and IIB	0	0	0
IIIA and IIIsA	3(*4–6*)	2(*7, 8*)	5
IIIB and IIIsB	3(*9–11*)	0	3
Subtotal	9	2	11

Case No.	Code†	
(*1*)	TR	Recurrence in treated neck.
	TDR	Extension to untreated pelvic lymph nodes.
	EL	Liver, bone marrow.
(*2*)	EL	Liver.
(*3*)	EL	Orbital metastasis with proptosis and subcutaneous nodule of thigh.
(*4*)	TR	Right axillary recurrence not irradiated a second time; placed on chemotherapy.
	EL	Liver, bone marrow, subcutaneous nodules.
(*5*)	TR	Recurrence in previously irradiated pelvis not irradiated a second time; placed on chemotherapy.
	EL	Bone marrow, peripheral blood.
(*6*)	EL	Liver, osseous disease in C-4.
(*7*)	TR	Left axillary recurrence not retreated with radiation; placed on chemotherapy.
	EL	Bone marrow, peripheral blood.
(*8*)	TR	Recurrence in irradiated mediastinum.
	EL	Malignant pleural effusion, liver.
(*9*)	TR	Left axillary recurrence; irradiated again for palliation.
	EL	Testicle.
(*10*)	RR	Extension to right and left upper extremities.
	EL	Liver, bone marrow.
(*11*)	EL	Bone marrow, central nervous system.

* Only Case No. (*3*) was treated with extended mantle irradiation alone; the remaining 10 cases either completed total lymphoid irradiation or had dissemination during treatment of the lower inverted-Y.

† Key: TR = True recurrence.
RR = Regional recurrence.
TDR = Transdiaphragmatic recurrence.
EL = Extralymphatic disease.

TABLE VIII

COMPARISON OF SYMPTOMS AT TWO RATES OF TREATMENT FOR EXTENDED MANTLE IRRADIATION

Symptoms	Fraction of Cases	
	150 rads/day	200 rads/day
Nausea	12/19 (63%)	38/76 (50%)
Vomiting	9/19 (47%)	25/76 (33%)
Esophagitis	5/19 (26%)	25/76 (33%)
Skin reactions	4/19 (21%)	26/76 (34%)
Diarrhea	0	4/76 (5%)

Six of these failures were due to true recurrences associated with extralymphatic disease; 1 was due to a regional recurrence associated with extralymphatic disease, and 4 to extralymphatic disease unassociated with true or regional recurrence. Details of each failure are shown in the footnote to Table VII.

Relapse-free rates. The probability of being relapse-free at 24 months was 0.62 for lymphocytic lymphoma in all stages, 0.48 for histiocytic lymphoma in all stages, 0.69 for histiocytic lymphoma Stage I and II, and 0.53 for all types and stages of non-Hodgkin's lymphoma.

COMPLICATIONS OF EXTENDED MANTLE IRRADIATION

The general morbidity associated with therapy is summarized in Table VIII. Symptoms at 2 different rates of treatment are compared. It was initially expected that patients treated with 150 rads per day would have fewer radiation-induced symptoms; review of our records failed to support this expectation, however. Extended mantle irradiation of 95 patients was associated with nausea in 50 patients (53 per cent), vomiting in 34 (36 per cent), esophagitis in 30 (32 per cent), severe skin reactions in 30 (32 per cent), and diarrhea in 4 cases (4 per cent).

MILD COMPLICATIONS

These are summarized in Table IX. Eight patients became dehydrated at termination of therapy because of radiation esophagitis and inadequate oral intake, and had to be hospitalized for admin-

TABLE IX

MILD COMPLICATIONS RESULTING FROM EXTENDED MANTLE IRRADIATION

Type	No. of Cases	Per Cent
Dehydration	8	8
Hypothyroidism (clinical)	3	3
Radiation caries	2	2
Thrombophlebitis	2	2
Complete xerostomia	1	1

istration of intravenous fluids. Several others received intravenous fluids as outpatients, with prompt correction of dehydration. Three patients became hypothyroid and required therapy with thyroid extract. In 2 patients, radiation-induced caries developed several years after treatment; one patient underwent total dental restoration, and the other had several lower teeth extracted. Partial xerostomia occurred in the majority of patients and complete xerostomia in 1 patient who received 4,000 rads in 4 weeks. Two patients were hospitalized for thrombophlebitis, received anticoagulants, and recovered without developing clinical evidence of pulmonary emboli.

Other mild sequelae noted in conjunction with extended mantle field irradiation were pulmonary fibrosis and gastric pain. In most patients, radiation-induced paramediastinal pulmonary fibrosis developed in areas corresponding to the portal margins. In 1 case, we suspected that a recently developed, smoothly rounded dense area in the region of the hilum represented recurrent disease, and subjected the patient to a thoracotomy which revealed only fibrosis. Epigastric pain during the course of irradiation was noted in 4 patients who received 4,000 rads in 4 weeks, but the pain did not persist and no clinical evidence of gastric injury ensued in these or any other patients.

No patient developed spinal cord injury or peripheral nerve damage. As previously reported,[11] an unseparated splice is used when the pelvis is treated in addition to the extended mantle field, so that total lymphoid irradiation is achieved. This splice is made at a level below the spinal cord, and over peripheral nerves and mobile segments of bowel. Among 61 patients so treated, no nerve injuries were recorded, and only 1 injury of the large bowel was noted at a site distant from the splice. This injury occurred in a patient with ulcerative colitis who developed a sigmoid obstruction and perforation associated with an inflammatory mass after radiation to the pelvis.

SEVERE COMPLICATIONS

Table x summarizes the severe complications.

Radiation pneumonitis. Five patients developed severe radiation pneumonitis within the first 2 months following completion of the extended mantle therapy. In 4 patients, the presentation was similar: they developed tachypnea and a low arterial pressure of oxygen. Chest roentgenography (Fig. 8) demonstrated bilateral paramediastinal and hilar infiltrates corresponding to the portal margins, and left lower lobe infiltrates corresponding to the splenic pedicle extension. In 2 of these cases, infiltrates were later noted beyond the portal margins in the peripheral portions of the lung. Sputum cultures in both cases revealed mixed flora with *Staphylococcus aureus* predominating in 1 case and *E. coli*, *enterococcus* species, and *Candida albicans* in the other. Staphcyllin was used in the

TABLE X

SEVERE COMPLICATIONS RESULTING FROM EXTENDED MANTLE IRRADIATION

Type	No. of Cases	Per Cent
Radiation pneumonitis	5	5
Severe esophagitis	2	2
Tracheoesophageal fistula	1	1
Constrictive pericarditis	1	1
Pulmonary embolism	1	1

first case with minimal success, and prednisone, 100 mg. daily, in the second with rapid symptomatic improvement. The diffuse infiltrates resolved in both cases, leaving the patients asymptomatic. The other 2 patients in this group did not develop peripheral infiltrates and became asymptomatic over 3 months of observation on steroid therapy, which was gradually tapered. The fifth patient was a woman with massive mediastinal H.D., nodular sclerosis type. It was not possible to shield much of the normal lung, and the patient developed a chronic cough and respiratory insufficiency after a protracted course of irradiation which included multiple rest periods to allow shrinkage of the tumor. She subsequently developed extralymphatic disease and died.

Severe esophagitis. Two patients developed severe esophagitis during the course of extended mantle therapy, with subsequent profound weight loss. Both patients developed extralymphatic disease; 1 died and 1 is alive.

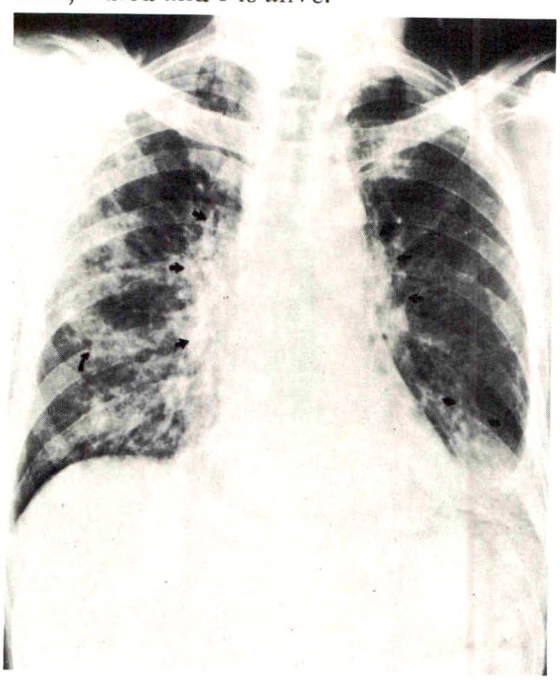


FIG. 8. Radiation pneumonitis after extended mantle irradiation. Paramediastinal and hilar infiltrates correspond to splenic pedicle extension. Peripheral infiltrates (curved arrow) are present beyond margins of irradiated lung.

Tracheoesophageal fistula. One patient, a 26 year old male with massive mediastinal disease involving the pericardium and left lung, developed a tracheoesophageal fistula midway through the course of irradiation. After the patient had received adequate nutritional intake through a gastrostomy, the fistula healed spontaneously and the patient is alive without disease after 2 years.

Constrictive pericarditis. One patient, a 25 year old male with H.D., nodular sclerosis type, was retreated by us and subsequently developed constrictive pericarditis. This patient had received 2,736 rads of kilovoltage irradiation to the mediastinum over 6 weeks at another hospital. Three months later, he was referred to our institution with obvious roentgenographic evidence of recurrent mediastinal disease. He was treated with an extended mantle field, receiving 4,000 rads in 4 weeks to the midmediastinum with shielding of the entire thoracic and cervical spinal cord. The pericarditis developed 18 months later, necessitating pericardiectomy. Residual disease was found in a fibrotic encapsulated lymph node. The patient is presently on chemotherapy and doing well. This was our only case of clinical pericarditis among 8 patients whose mediastinum was retreated. None of the 87 patients who received their initial treatment at our institution developed radiation pericarditis.

Blood cell counts. On the basis of the distribution of marrow in adults as described by Ellis,[2] we estimate that approximately 36 per cent of the total marrow is included in an extended mantle field. Figures 9 and 10 show white blood cell counts and platelet counts *versus* midplane dose in rads for all patients during extended mantle irradiation. The status of blood cell counts during lower inverted-Y irradiation is not represented in these graphs. In 12 patients, irradiation of the extended mantle field had to be interrupted because of thrombocytopenia or leukopenia; extralymphatic disease later occurred in 10 of these 12. Of the 10 patients who had rest periods during ex-

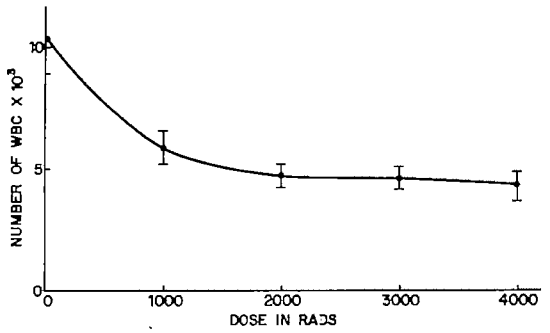


FIG. 9. Effect of extended mantle irradiation on white blood cell count (WBC). Points on the solid line represent mean white blood cell count values; the vertical lines are ±2 standard errors of the mean.

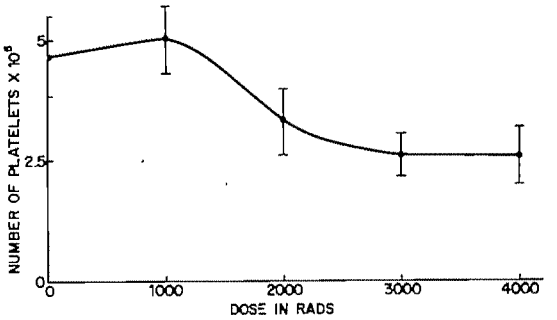


FIG. 10. Effect of extended mantle irradiation on platelet count. Each point on the solid line represents the mean platelet count; the vertical lines are ±2 standard errors of the mean.

tended mantle irradiation and later developed extralymphatic disease, 6 had histiocytic lymphoma, 2 had lymphocytic lymphoma, and 2 had H.D. Two of the 10 patients in this group had previously received chemotherapy. It should be emphasized that all patients had undergone staging laparotomy and splenectomy, and that splenectomy in all likelihood was an important factor in permitting completion of radiotherapy without hematopoietic depression in most patients.[16]

Mortality. One death attributable to radiotherapy occurred in a young woman who was near completion of treatment with the extended mantle field, and who had esophagitis, dehydration, and hemoconcentration, resulting in a massive pulmonary embolus.

DISCUSSION

The rationale for the use of the extended mantle field is to eliminate the need for splicing at the level of the diaphragm in total lymphoid radiotherapy of H.D. and M.L. Such splicing can result in overlap of the mantle and inverted-Y fields, causing excessive dose in the region of the spinal cord. Transverse myelitis can ensue and the patient may be left with the sequelae of paresis and of dysfunction of bladder and bowel. Alternatively, excessive separation can result in an inadequate dose to diseased regions, and thus in recurrence of disease.[5] Retreatment is difficult because of the danger of exceeding the radiation tolerance of normal organs. Stewart and Kaplan[17] reported an incidence of 0.15 per cent (1 case in 592 patients treated) for radiation-induced transverse myelitis, and cautioned that meticulous technique and treatment planning are required. On retrospective review of their case, they found that inadequate separation of fields had probably occurred. There are few documented reports of this complication in the literature, but we continually hear of such injury from other therapists in major treatment centers. Glenn *et al.*[5] reported on a patient who developed recurrence in the anterior mediastinum in the region of the splice separation. We have not seen a recurrence at the diaphragm with such splicing, but we are very familiar with the patient who presents with recurrent disease between portals after "patchwork radiotherapy" over multiple fields.

We are aware of the potential for error in our own facility, which has a heavy workload and limited staff; therefore, we became convinced that elimination of the splice at the level of the diaphragm by the formation of what came to be called the "extended mantle" field would be advantageous. Such a field eliminates the splice and thus the possibility not only of overlap, but also of excessive separation of fields. When we began to use this field in late 1968, there

were 2 uncertainties: (1) whether such an extensive field would be tolerated by the patients; and (2) whether treatment results would be acceptable. We now feel that, in general, the field is tolerated well despite the increase in integral dose, and that our treatment results are acceptable. The aged, the medically infirm, and those subjected to previous chemotherapy, however, are less tolerant of such extensive irradiation and are not good candidates for the extended mantle field.

In our series of 68 cases with H.D. treated with extended mantle fields or with total lymphoid irradiation, there were 19 cases still without disease within the first 2 years of follow-up when, in the general experience, the majority of recurrences are noted. Some of these patients may fail at a future date. The fact that almost all failures after extended mantle irradiation (22 of 23 cases, Table IV) occurred within the first 2 years indicates that follow-up during this period should be frequent, especially since some patients can be salvaged with further radiotherapy. Kaplan[9] reported significant relapses in the third year of follow-up as well, a finding that may become apparent in further studies of our case material. As was expected, most of the failures in our series (18 of 23 cases, Table IV) were noted among patients with Stage III disease. Thirteen of these 18 patients had extralymphatic disease. The relapse-free curve calculated for Stage I and II H.D. treated with extended mantle irradiation alone (Fig. 5) is comparable to that published by Kaplan,[9] whose cases were treated with classic mantle fields. It would seem from examination of our actuarial relapse-free curves for Stage IIIA and IIIB H.D. that adjuvant combination chemotherapy might be instrumental in preventing relapse. However, the number of patients in the 2 groups is too small to show statistically significant differences. (In addition, there was a degree of bias involved in the selection of patients who received chemotherapy; it has only been in the past 4 years that all adults with Stage IIIA and IIIB H.D. have been randomized to receive or not to receive adjuvant chemotherapy.) Rosenberg *et al.*[14] reported convincing evidence that relapse-free rates were significantly improved with the use of chemotherapy in a randomized study of patients with different stages of H.D.

In cases of M.L., most failures (9 of 11 cases) occurred within the first year and were associated with extralymphatic disease in all instances. The probability of being relapse-free was greatest for lymphocytic lymphoma and least for histiocytic lymphoma. The data of Jones *et al.*[7] also indicate that the prognosis for histiocytic lymphoma is relatively poor. It should be noted that patients with Stage I and II M.L. are salvageable with extended field radiotherapy. In our series, 10 of 13 patients with Stage IA or IIA disease are alive without disease at the present time (Table VI); 10 of these 13 patients had diffuse histiocytic lymphoma, and 7 of these 10 are alive without disease. As in H.D., the patients with Stage III M.L. failed with the greatest frequency. Unlike H.D., however, no patients who failed were salvaged with additional irradiation. There were too few cases with nodular histopathology for comparison with cases of diffuse histopathology.

We considered it significant that 3 of 6 cases of H.D. (Table II) and 1 of 2 cases of M.L. (Table V) previously treated with various modalities of radiation were alive without disease after retreatment with extended mantle radiation. For a previously treated patient, it is necessary to obtain the data from the institution where such treatment took place, and to incorporate the findings into treatment planning with meticulous attention to the radiation tolerance of the various tissues encompassed by the field. One of the most frequent problems in the proper portal design for previously treated patients is the lack of skin tattoos, which, if present, would help to indicate where potential splice injuries might occur in relation to the spinal cord. With sufficient data, however, curative irradiation can be undertaken in a previously treated

patient, recognizing the higher potential for radiation injury of various organs. Our case of constrictive pericarditis was the only one with significant injury among the 8 patients who were retreated.

In extended mantle irradiation, nausea and vomiting occur acutely with the first treatment, but may be diminished with phenothiazines administered rectally before and after treatment. As pointed out in Table VIII, the prevalence of acute radiation-induced symptoms in patients treated at 150 rads per day was comparable to that in patients treated at 200 rads per day. We felt that this unexpected finding was best explained by case selection: Cases selected to receive 150 rads per day were more ill than those given 200 rads per day.

Esophagitis generally occurs between 2,000 and 3,000 rads, and may result in inadequate oral intake and subsequent dehydration. The latter is potentially the most dangerous complication that may result from this extensive radiation field, since it may lead to severe hemoconcentration and to pulmonary embolism, which was the cause of death in 1 patient treated with the extended mantle field.

The problem of radiation-induced caries and partial xerostomia was not entirely unexpected since the upper margins of our portals (Fig. 4) routinely encompass the parotid glands. The resulting reduction in flow of saliva and alteration in oral pH can lead to radiation caries if appropriate dental care is not given before, during, and after treatment. During radiation therapy our patients are routinely treated with fluoride gel, and they are advised to continue using it permanently. Unfortunately, neither of the 2 patients who developed caries had adhered to the advice given.

Clinical hypothyroidism was observed in 3 per cent of the patients. However, it is possible that laboratory-documented thyroid deficiency without clinical signs may have been high.[4]

Kaplan[9] discussed the problem of radiation pneumonitis in patients with large mediastinal masses or contiguous infiltration of lung tissue by mediastinal lymphoma. In the first instance, only a small volume of lung can be shielded, and in the second, the involved lung is irradiated. In both cases, the risk of pneumonitis is great because large volumes of lung are included in the field. Like the Stanford group,[9] we interrupt the treatment after the administration of midplane doses of 1,500 to 2,000 rads in 1.5 to 2 weeks, to allow regression of disease, so that shielding blocks larger than those used initially can be constructed. Often regression is very slow, particularly in patients with nodular sclerosis H.D., presumably due to the large amount of collagen present in the tumor. In such cases, the treatment is spread over long periods of time and the total dose is usually increased. From a technical standpoint, the problem is to shield as much lung as possible. Sutton[18] has noted that the mediastinum is less wide when the patient is in the upright position, and feels that treating in this position is feasible and probably beneficial. Sequential port films indicate that localization errors are no greater than those noted for patients treated in the supine position.

Except in 1 patient whose mediastinum was retreated for recurrent H.D., radiation-induced injury to the pericardium has not been a problem. Our single case among 8 retreated patients is comparable to the injury rate of 19.5 per cent noted by Kaplan[9] in retreated patients. Among the 87 patients initially treated at our institution, there has been no pericarditis. Kaplan reported a total of 32 cases of radiation-induced heart disease among 592 patients treated daily to a total dose of 4,000 to 4,400 rads (a rate of 5.4 per cent), and pericardiac injury was detected only by virtue of roentgenographic evidence of cardiomegaly.[9] We have routinely roentgenographed our patients and have not seen asymptomatic cardiomegaly. We regard the absence of injury among our patients treated *de novo* as possibly being due

to our schedule of 200 rads per day, 5 days per week, and daily treatment of both fields.

The 2 cases of severe esophagitis were most probably of infectious etiology; *Pseudomonas aeruginosa* was cultured in one case, and *Candida albicans* was suspected, but not cultured in the other. Both patients ultimately proved to have disseminated disease; although no proof exists, both may have suffered immunologic impairment, allowing infection to become superimposed upon a radiation-injured esophagus.

The hematopoietic system tolerated extended mantle irradiation rather well, except in those patients who ultimately developed disseminated disease. Quite possibly, the hematopoietic intolerance in these cases is due to disease involving the bone marrow itself. Improved means of sampling or scanning bone marrow are needed to detect those patients who are more likely to benefit from use of systematic chemotherapy than from radiotherapy. The fact that many patients require a rest period during treatment of the lower portion of the inverted-Y field indicates that the irradiated marrow in the extended mantle field has not sufficiently recovered, and that the pelvic bone marrow is functionally borderline. Treatment of the lower inverted Y is often protracted, and the total dose usually cannot be increased to compensate for this. Only 1 patient with H.D. and 1 with poorly differentiated lymphocytic lymphoma developed true recurrences in the pelvis after a total dose of 4,000 rads. Both recurrences were associated with extralymphatic disease and may have been due to retrograde seeding.

SUMMARY

The extended mantle field eliminates the necessity for splicing at the level of the diaphragm and thereby eliminates the potential for radiation-induced transverse myelitis.

Our experience to date with 95 patients with H.D. or M.L. is described.

Modifications in technique, including changes in the geometry of the field, shielding, time, dose, fractionation, and simulation are described.

End results, an analysis of failures, and complications encountered are presented. Morbidity was acceptable despite the increase in integral dose, particularly in young patients who were not debilitated by disease.

The danger of radiation-induced esophagitis, dehydration, hemoconcentration, and pulmonary embolism is stressed.

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REFERENCES

1. Carbone, P. P., Kaplan, H. S., Musshoff, K., Smithers, D. W., and Tubiana, M. Report of committee on Hodgkin's disease staging classification. *Cancer Res.*, 1971, *31*, 1860–1861.
2. Ellis, R. E. Distribution of active bone marrow in adult. *Phys. Med. Biol.*, 1961, *5*, 255–258.
3. Ferguson, D. J., Allen, L. W., Griem, M. L., Moran, E. M., Rappaport, H., and Ultmann, J. E. Surgical experience with staging laparotomy in 125 patients with lymphoma. *A.M.A. Arch. Int. Med.*, 1973, *131*, 356–361.
4. Glatstein, E., McHardy-Young, S., Brast, N., Eltringham, J. R., and Kriss, J. P. Alterations in serum thyrotropin (TSH) and thyroid function following radiotherapy in patients with malignant lymphoma. *J. Clin. Endocrinol. & Metabol.*, 1971, *32*, 833–841.

5. GLENN, D. W., FAW, F. L., KAGAN, A. R., and JOHNSON, R. E. Field separation in multiple portal radiation therapy. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *102*, 199–206.
6. HAUS, A. G., STRUBLER, K., and MARKS, J. E. Film technique for determining absorbed dose at exit from patient during radiation exposure. *Radiology*, 1972, *104*, 197–200.
7. JONES, S. E., FUKS, Z., BULL, M., KADIN, M. E., DORFMAN, R. F., KAPLAN, H. S., ROSENBERG, S. A., and KIM, H. Non-Hodgkin's lymphomas: clinicopathologic correlation in 405 cases. *Cancer*, 1973, *31*, 806–823.
8. KAPLAN, E. L., and MEIER, P. Nonparametric estimation from incomplete observations. *Am. Statis. A. J.*, 1958, *53*, 457–481.
9. KAPLAN, H. S. Hodgkin's Disease. Harvard University Press, Cambridge, 1972.
10. LUKES, R. J., BUTLER, J. J., and HICKS, E. G. Natural history of Hodgkin's disease as related to its pathologic picture. *Cancer*, 1966, *19*, 317–344.
11. MARKS, J. E., PINSKY, S. M., and GRIEM, M. L. Extended mantle field in radiotherapeutic treatment of malignant lymphoma. *Radiology*, 1971, *100*, 423–425.
12. POWERS, W. E., KINZIE, J. J., DEMIDECKI, A. J., BRADFIELD, J. S., and FELDMAN, A. New system of field shaping for external-beam radiation therapy. *Radiology*, 1973, *108*, 407–413.
13. RAPPAPORT, H. Tumors of hematopoietic system. In: Atlas of Tumor Pathology. Section III. Fascicle 8. Armed Forces Institute of Pathology, Washington, D. C., 1966.
14. ROSENBERG, S. A., MOORE, M. R., BULL, J. M., JONES, S. E., and KAPLAN, H. S. Combination chemotherapy and radiotherapy for Hodgkin's disease. *Cancer*, 1972, *30*, 1505–1510.
15. SALZMAN, J. R., and KAPLAN, H. S. Effect of prior splenectomy on hematologic tolerance during total lymphoid radiotherapy of patients with Hodgkin's disease. *Cancer*, 1971, *27*, 471–478.
16. SCHROEDER, A. F., SCHER, A. J., and MEURK, M. L. Rapid method of localization for Hodgkin's mantle. *Radiology*, 1970, *94*, 701–702.
17. STEWART, J. R., and KAPLAN, H. S. Neurologic complications of Hodgkin's disease and its treatment in 593 patients. Presented at the annual meeting of the American Society of Therapeutic Radiologists, Phoenix, Arirona, November 13, 1970. Unpublished data.
18. SUTTON, H. S. Personal communication, 1973.
19. ULTMANN, J. E. Report on workshop: clinical evaluation, staging and prognosis. *NCI Monograph*, 1973, *36*, 355.

Vol. 121, No. 4

ANGIOGRAPHY OF THE LIVER AND SPLEEN IN HODGKIN'S DISEASE*

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IN Hodgkin's disease, the spread of tumor to the liver is an indication for chemotherapy. Curative treatment by radiotherapy is not possible in these cases, as the liver does not tolerate a radiation dose of 4,000 rads.[7] The diagnosis of liver involvement is thus of great importance in the choice of treatment. To test the accuracy of angiography of the liver and spleen concerning involvement in Hodgkin's disease, a prospective study has been carried out. The results of the angiographies have been correlated to the findings at liver biopsy and splenectomy at staging laparotomy.

MATERIAL AND METHOD

In 18 patients with Hodgkin's disease proven by lymph node biopsy, celiacography was performed in 17 patients. Of these, selective hepatic angiography was performed in 9, and selective splenic angiography in 2. In 1 patient with stenosis of the celiac axis, only aortography was performed. The laparotomy, which included splenectomy and wedge biopsy of the liver (or biopsy of suspected infiltrations in the liver), was performed within 1 month after angiography in 13 patients and after 46–76 days in 4 patients. In 1 patient the laparotomy was performed prior to angiography.

RESULTS

The angiography of the liver was pathologic in 2 patients, with deviation of intrahepatic arteries and multiple filling defects in the parenchymal phase in one (Fig. 1, *A* and *B*), and a single filling defect in the other. In 1 patient nodular infiltrations were palpated at laparotomy, corresponding to the filling defects seen at angiography, but in the other patient the liver was normal at palpation during operation, and no biopsy was undertaken from the suspected area. In the remaining patients the liver angiography was normal and the liver was interpreted as normal at operation, but in 2 of these cases histology revealed Hodgkin's disease.

At angiography the size of the liver was judged as normal in 11 cases, enlarged in 5, and small in 2. In the 3 patients with Hodgkin's disease in the liver, the size of the liver was enlarged in 1 and normal in 2.

The spleen could be evaluated in 15 patients at celiacography. In 2 patients it was not visualized and in 1 patient it had been extirpated earlier. Filling defects 5–30 mm. in diameter (Fig. 2, *A* and *B*) were found in 7 patients and in 1 a defect of 40×50 mm. was detected, with corresponding deviation of the arteries. In 7 patients the angiography of the spleen was normal without filling defects or deviation of the intrasplenic arteries. In no case were tumor vessels, tumor blush, or arteriovenous shunting seen. The histologic examination of the extirpated spleens revealed Hodgkin's disease in 7 of the 8 patients with filling defects, and in 2 of the 7 patients with normal findings. Filling defects were found in 1 patient with normal histology. At angiography the involved spleens were considered to be enlarged in 7 cases and of normal size in 2. The uninvolved spleens were enlarged in 1 case and of normal size in 5.

Histologic examination revealed epithelioid cell granulomas without Hodgkin's disease[2,8] in 4 livers and in 4 spleens. The angiographies of the liver in these cases were all normal. In 2 patients the granulomas were combined with Hodgkin's dis-

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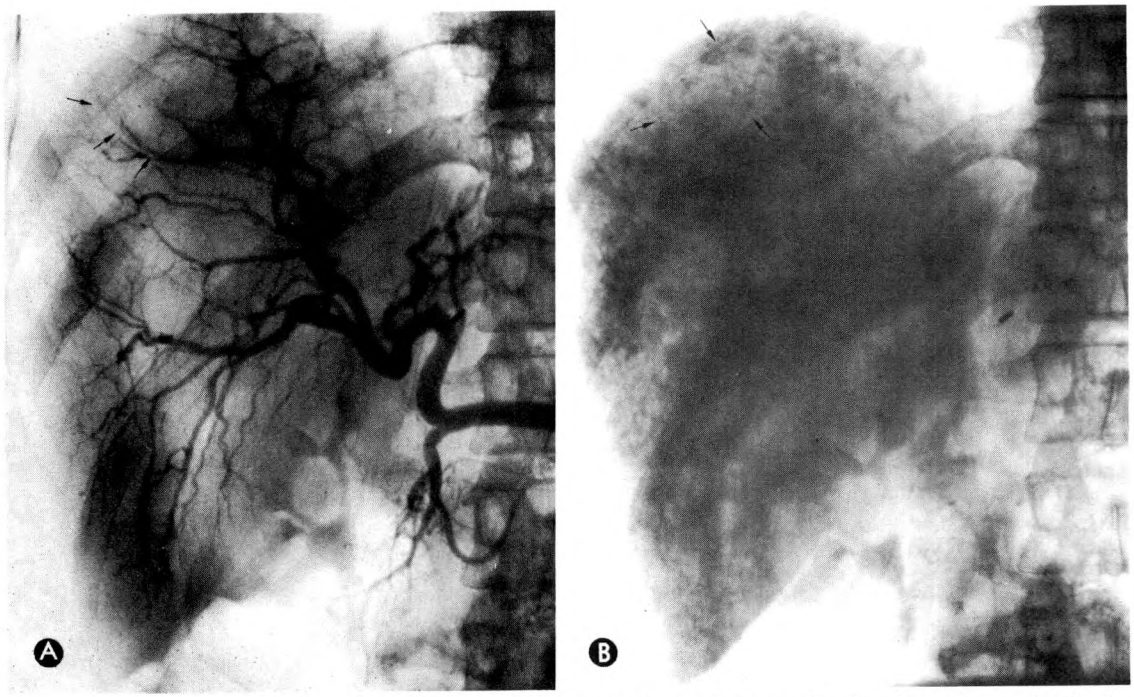


FIG. 1. Angiography of the liver in Hodgkin's disease. (*A*) Arterial phase. The intrahepatic arteries in the right lobe are displaced (arrows). (*B*) Parenchymal phase. A large filling defect is seen corresponding to the displaced arteries (arrows). Smaller filling defects are also seen.

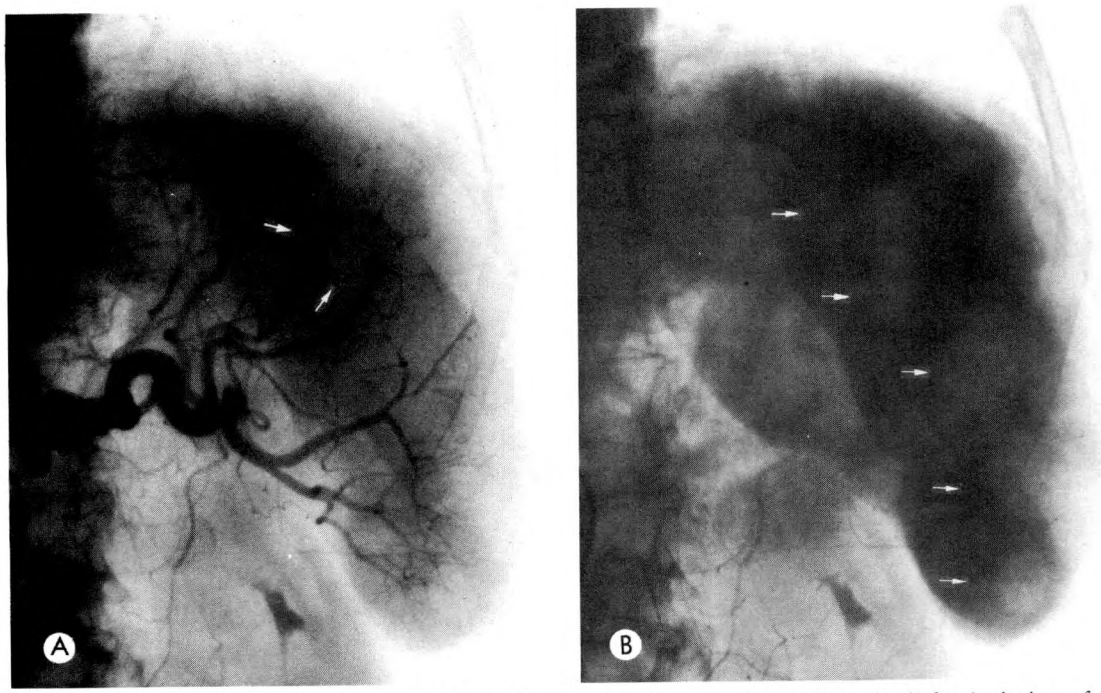


FIG. 2. Angiography of the spleen in Hodgkin's disease. (*A*) Arterial phase. There is slight deviation of the intrasplenic arteries (arrows). (*B*) Parenchymal phase. Several filling defects are seen (arrows).

ease in the spleen and filling defects were seen at angiography. In the other 2 patients with granulomas in the spleen, the angiography was normal in one and showed filling defects in the other.

DISCUSSION

It has been shown[1,4–6,9–11] that palpation and laboratory tests are of very limited value in the judgment of liver or spleen involvement in Hodgkin's disease. As the liver is in a way a key organ in the choice of treatment between radiotherapy and chemotherapy, the diagnosis of liver involvement is extraordinarily important. Involvement of the liver does not occur in the absence of splenic involvement in Hodgkin's disease.[5]

Percutaneous needle aspiration biopsy of the liver has been shown to be of value,[1] but in negative cases the results improved when additional biopsy was made at laparotomy or through laparoscopy. Percutaneous fine needle aspiration biopsy has shown excellent correlation, when compared with biopsy at staging laparotomy, but may be false negative.[10] In the same report a high accuracy of the fine needle aspiration biopsy of the spleen was noted. However, when fine needle aspiration biopsy, palpation and biopsy of the liver at operation are taken as final proof, we must also be aware of the limitations of these methods. Blind fine needle aspiration biopsy as well as biopsy at operation from an apparently normal liver may be positive only when microscopic changes are scattered throughout the organ. When localized deep in the parenchyma, even large scale solitary infiltration may escape detection at both palpation and biopsy. This may have happened in 1 of the cases in the present material, where deviated vessels of the liver and a filling defect were seen.

Scintigraphy of the liver and spleen is an easy procedure for the patient. In cases with Hodgkin's disease it has shown a relatively good correlation concerning spleen involvement, but less accuracy in liver diagnosis.[10] The reverse experience, however, has also been reported.[12,13]

Angiography is a nontraumatic method to examine the liver and spleen. There is no risk of rupture or bleeding as at biopsy. However, the angiographic studies failed to give a correct diagnosis in 2 of 3 patients with involvement of the liver. The findings of the third patient revealed a dislocation of the arteries and filling defects in the parenchymal phase. No other angiographic signs of malignancy were seen. These filling defects corresponded to palpable nodules of Hodgkin's disease in the liver. A similar finding was noted in another liver, but in that case it could not be verified as Hodgkin's disease. The same type of angiographic findings was seen in the spleens in which palpable nodules were noted at operation; *i.e.*, deviation of intrasplenic arteries and nonspecific filling defects in the parenchymal phase. These filling defects are quite nonspecific in the spleen and may occur in normal cases as well as in involved spleens.[3] The use of angiography of the liver and spleen has thus given disappointing results concerning the spread of lymphoma to these organs. The reason for the failure of angiography in so many cases is the scattered microscopic spread of the disease; the same was the case in the epithelioid cell granulomatous reactions. It is only in the cases with large palpable nodules of lymphoma that the angiography is positive.

An enlarged liver and spleen demonstrated by palpation or at angiography are no proof of involvement, and, conversely, a normal-sized liver and spleen may well be involved, although in the present material enlargement of the spleen has been demonstrated in most cases with diseased spleen.

SUMMARY

Angiography of the liver and spleen was performed in 18 patients with Hodgkin's disease.

The results of the angiographies have been correlated with the findings at staging

laparotomy, which included wedge biopsy of the liver and splenectomy.

At angiography nonspecific filling defects and dislocation of intrahepatic arteries were found in 1 patient with lymphoma in the liver. Similar filling defects in the spleen are nonspecific and may occur in both involved and uninvolved cases. No specific angiographic pattern was seen in cases with epithelioid cell granulomas in the liver or spleen.

Angiography is thus of limited value in the preoperative evaluation of the liver and spleen. The only reliable method of establishing spread of the lymphoma is laparotomy.

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REFERENCES

1. BAGLEY, C. M., ROTH, J. A., THOMAS, L. B., and DEVITA, V. T. Liver biopsy in Hodgkin's distase. *Ann. Int. Med.*, 1972, *76*, 219–225.
2. BRINCKER, H. Epithelioid-cell granulomas in Hodgkin's disease. *Arch. path. & microbiol. scandinav.*, 1970, *A-78*, 19–32.
3. CASTELLINO, R. A., SILVERMAN, J. F., GLATSTEIN, E., BLANK, N., and WEXLER, L. Splenic arteriography in Hodgkin's disease: roentgenologic-pathologic study of 33 consecutive untreated patients. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1972, *114*, 574–582.
4. ENRIGHT, L. P., TRUEBLOOD, H. W., and NELSEN, T. S. Surgical diagnosis of abdominal Hodgkin's disease. *Surg., Gynec. & Obst.*, 1970, *130*, 853–858.
5. GLATSTEIN, E., GUERNSEY, J. M., ROSENBERG, S. A., and KAPLAN, H. S. Value of laparotomy and splenectomy in staging of Hodgkin's disease. *Cancer*, 1969, *24*, 709–718.
6. HASS, A. C., BRUNK, S. F., GULESSERIAN, H. P., and GIVLER, R. L. Value of exploratory laparotomy in malignant lymphoma. *Radiology*, 1971, *101*, 157–165.
7. INGOLD, J. A., REED, G. B., KAPLAN, H. S., and BAGSHAW, M. A. Radiation hepatitis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *93*, 200–208.
8. KADIN, M. E., DONALDSON, S. S., and DORFMAN, R. F. Isolated granulomas in Hodgkin's disease. *New England J. Med.*, 1970, *283*, 859–861.
9. KADIN, M. E., GLATSTEIN, E., and DORFMAN, R. F. Clinico-pathologic studies of 117 untreated patients subjected to laparotomy for staging of Hodgkin's disease. *Cancer*, 1971, *27*, 1277–1294.
10. LANDBERG, T., MÖLLER, T., BENGMARK, S., BÖRJESSON, B., OLSSON, A., CAVALLIN-STAHL, E., AHLSTRÖM, C. G., AKERMAN, M., JONSSON, K., LUNDERQUIST, A., and NAVERSTEN, Y. Staging laparotomy with splenectomy in Hodgkin's disease. *Acta chir. scandinav.*, 1974, *140*, 205–213.
11. LEVITAN, R., DIAMOND, H. D., and CRAVER, L. F. Liver in Hodgkin's disease. *Gut*, 1961, *2*, 60–71.
12. LIPTON, M. J., DeNARDO, G. L., SILVERMAN, S., and GLATSTEIN, E. Evaluation of liver and spleen in Hodgkin's disease. I. Value of hepatic scintigraphy. *Am. J. Med.*, 1972, *52*, 356–361.
13. SILVERMAN, S., DeNARDO, G. L., GLATSTEIN, E., and LIPTON, M. E. Evaluation of liver and spleen in Hodgkin's disease. II. Value of splenic scintigraphy. *Am. J. Med.*, 1972, *52*, 362–366.

KAPOSI'S SARCOMA*

EXPERIENCE WITH TEN CASES

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KAPOSI's sarcoma is a neoplastic condition characterized by multiple skin and subcutaneous tumors which are slow growing and histologically unique. It is a common malignancy in South Africa, but uncommon in the U.S.A. The largest series reported in the U.S.A. is 100 cases from New York City.[5]

Ten cases seen over a 5 year period at the Zellerbach Saroni Tumor Institute of Mount Zion Hospital prompted this report and a review of the literature.

REVIEW OF LITERATURE

The discoverer of "Idiopathic Multiple Hemorrhagic Sarcoma" was Moricz Kaposi (1837–1902).[23] He was an exceptional dermatologist, who first described xeroderma pigmentosum and named the disease. In 1872 he reported 5 cases which he designated "Idiopathic Multiple Pigment Sarcoma of Skin."[15] In 1882 Tommass de Amicis of Naples reported 12 further cases, but after this report 75 years passed before interest in the disease was renewed, in the 1950s, when it was realized that in Africa the disease was relatively common. In May, 1971, a symposium on Kaposi's sarcoma was held in Kampala, Uganda, providing a major body of information.

At the University of Chicago from 1946 to 1960 only 8 cases were reported in the clinics out of a total of 13,700 malignancies.[24] At the South African Institute for Medical Research 66 of 15,000 cancers registered from 1949 to 1953 were Kaposi's sarcoma,[21] comprising 15 per cent of all skin tumors. Melanoma comprised 44 per cent, squamous cell carcinoma 34 per cent, and basal cell carcinoma only 2 per cent. During the same period in Tanzania, Kaposi's sarcoma was 4 per cent of all malignancies.[29] Recently, Brownstein *et al.*[5] reported 100 cases from outpatients collated from a private dermatopathology laboratory in New York City, indicating that the disease may be more common than has been thought in this country.

In most studies males were affected more frequently than females. Cook[10] found 59 males affected and only 4 females in Uganda. Bluefarb,[3] in his extensive review, found between 6 and 10 per cent of the cases to be females. Brasfield and O'Brien's series,[4] which updates McCarthy and Pack's review[17] from Memorial Hospital in New York City, had a male to female ratio of 4.25 to 1.

The age peak was between 50 and 70 years, although in South Africa the average age for the disease is in the 40s (due to the generally younger population).[21] Oettle[21] found that the incidence increased from birth to puberty, decreased during the teens and then slowly increased with age in Africa. This is similar to the age incidence curve for lymphomas.

There is an increased incidence of the disease among Northern Italians and Eastern Europeans.[3] McCarthy and Pack[17] reported a large series of patients with a high percentage of Jews, but their material was drawn from New York City. Bluefarb[3] believed that the predilection of the disease for Jews was fictitious and depended more on country of origin, since most

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Jewish patients were from Russia or Poland. He also reported that up to 95 per cent of these people are laborers and outdoor workers and that there is an increased prevalence in occupations involving prolonged standing; but neither of these reports has been confirmed.

There has been no correlation with occupational carcinogens. No infectious agent has been found, although Greco[3] observed fungus particles within the cells of lesions and named them; these "fungi" were later shown to be artifacts. The etiology is unknown.

Because of its rarity, the presence of Kaposi's sarcoma in a higher frequency than would normally be expected with other diseases is of interest. Brasfield and O'Brien[4] found that 22 per cent of their patients had a second primary malignant lesion. It occurs in association with Hodgkin's disease[6] and non-Hodgkin's lymphoma[13] with greater frequency than would normally be expected. (Of 1,200 cases reported in the literature, there are 9 with Hodgkin's disease, or 1 per cent; in the general population the incidence of Hodgkin's disease is .002 per cent.[6]) In a case where Kaposi's sarcoma was found in the same lymph node with Hodgkin's disease an abrupt change took place from one histologic type to another.[6]

The early lesions are round or oval macules.[25] They are blue or purplish due to a combination of vascular dilatation and hemosiderosis. If untreated, lesions become confluent and may measure up to 15 cm. Tumors may involute spontaneously, while others appear. Ulceration is infrequent in the U.S.A., and when present suggests another diagnosis. There is a tendency to develop edema, even before the appearance of the nodules. The edema is non-pitting and the fluid accumulation is in the dermis, not subdermis and might be due to dermal lymph stasis. Pruritus is occasionally experienced by the patient over a clinically normal area just before the eruption of a new lesion. The clinical differential diagnosis includes granuloma pyogenicum, glomus tumor (very tender), or angioma (soft and round).

Kaposi's sarcoma can present with skin lesions alone, or with generalized lymphadenopathy.[2,26] The disease tends to fall into 2 patterns. The much more common type affects the skin, grows slowly and may metastasize to lymph nodes. Out of 81 lesions present in 59 patients, 50 per cent were on the lower extremities, 35 per cent were on the upper extremities and the remainder scattered on the head, neck, abdomen and thorax.[4] It has been reported in almost every other organ of the body.

The second, and less common type, occurs in children, involves the lymph nodes, generally without skin involvement, and is associated with a poor prognosis.[2] Of 281 cases from Uganda, 11 were in children.[11] Of these, 9 were under 11 years of age and 2 were between 11 and 20. All were male. Skin lesions were sparse or absent. Unfortunately, none of the children came to autopsy, but typically they had generalized lymphadenopathy with anemia and hepatosplenomegaly. Among African children, this form of the disease is characteristic. Among children from other countries, the "benign" type is more common.[12] Rylwin *et al.*[26] in this country reported 3 adults with the "malignant" presentation. All 3 died within 12 weeks of diagnosis. All had characteristic Kaposi's sarcoma involving lymph nodes. When the lymph nodes are enlarged in patients with Kaposi's sarcoma, however, they need not be involved.

Palmer[22] has reviewed the roentgenologic findings based on 200 cases from Uganda. The findings are not specific. In general, they are related to the local severity of the disease. Soft tissue roentgenograms of involved areas may show solitary or multiple (grape-like bunches) deep seated tumors. The chest roentgenogram is usually normal even in widespread disease, except for children in whom there are frequently increased hilar and mediastinal lymph nodes. Skeletal changes are found in areas in which soft tissue is involved. They include cysts up to 5 cm. in diameter, without

a pericystic reaction and no cortical breakdown. In addition, one may see rarefaction and cortical erosions. The former is the most common change. Percutaneous angiography can be useful and shows a characteristic appearance.[19] Each nodule receives blood supply from one artery, which divides into smaller vessels distributed in a haphazard direction. The venous channel is often a single channel too; arteriograms taken before and after treatment can show normal findings sometimes to a greater extent than indicated by the clinical response.[30]

A series of 19 autopsy cases from Africa has been presented by Lothe *et al.*[16] In 10 of 19 cases the cause of death was Kaposi's sarcoma; in the others, it was tropical infection or other malignancy. The skin lesions are in the lower or upper extremities usually. Many follow the superficial venous distribution and can occasionally be palpated when not seen.[18] They are usually located in the middle or lower dermis. When the lesions affect the viscera, the most commonly involved organs are small intestine, followed by liver, adrenal and large intestine. In the small intestine the lesions are found in the submucosa; occasionally, they grow through and ulcerate the mucosa. Lymph nodes are exceeded only by skin in incidence. In lymph nodes early lesions are usually subcapsular; those in lung are perivascular, and in the liver the earliest ones are in the portal tract.[18]

The lesions are uniform microscopically. They have 2 basic constituents: spindle cells and vascular structures.[18] The spindle cells are present in sweeping bundles; the vascular structures vary from a multitude of cleft-like spaces in which red blood cells are in direct contact with tumor cells, to areas which look almost exactly like capillaries. There is a moderate number of mitoses. Fibrous tissue trabeculae frequently give the lesions a lobulated appearance. Dilated thin walled lymphatics and hemosiderin deposits occur most frequently around the outside of the tumor. The spindle cell has been studied by electron microscopy.[20] It is ovoid with a large ovoid nucleus and a prominent nucleolus with evenly distributed chromatin. Niemin and Mustakallio[20] found that in younger angiomatous lesions, in areas where red blood cells were being phagocytized by histiocytes, the latter resembled spindle cells.

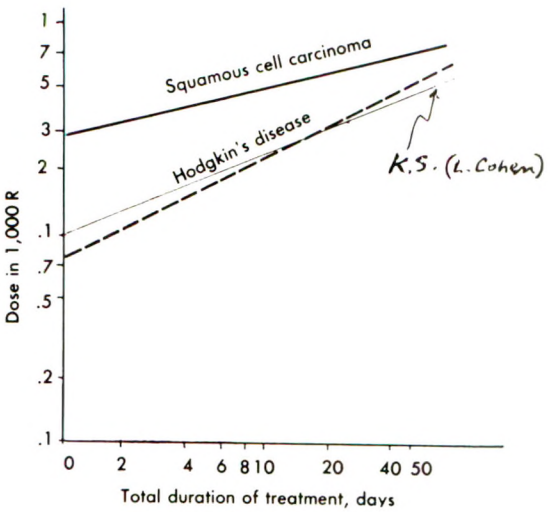


Fig. 1. The tumoricidal dose for Kaposi's sarcoma plotted on a log-log scale conforms closely to Hodgkin's disease (data for Kaposi's sarcoma superimposed on chart from Moss, William T., and Brand, William N.; Therapeutic Radiology, Ed. 3, St. Louis, 1969, The C. V. Mosby Co. Data for Hodgkin's disease taken from Scott and Brizel and for squamous cell carcinoma from Andrews and Moody).

There is very indirect evidence that Kaposi's sarcoma is a form of reticuloendothelial system neoplasia. (1) The incidence curve for Kaposi's sarcoma is similar to that of lymphoma, in that there is a small initial peak in the first decade, a drop during the teens, followed by a slowly steady rise after 20 years following the same slope.[21] (2) Isolated lesions regress while others spontaneously appear, reminiscent of the fluctuant nature of lymph nodes in lymphoma. (3) Its radiosensitivity, as determined by a dose time curve whose end point is control of clinical lesions, is similar to lymphomas (Fig. 1). (4) Electron microscopy studies have shown the possible morphologic transition of histiocytes to fibro-

blasts in sections from nodules of Kaposi's sarcoma.[20] Finally, the disease has a definite variation consisting of generalized lymph node involvement. Burkitt has compared the similarities of Burkitt's lymphoma to Kaposi's sarcoma also.[7]

Radiation therapy is the treatment of choice. In the past, arsenic, ultraviolet light, steroids, antireticular serum of Bogomolets and other methods have been used without success.[3] The lesions are very radiosensitive. Early vascular soft nodules and macules are more radiosensitive than the older infiltrated plaques.[17] Numerous articles using orthovoltage with doses from 75 r to 4,000 r have been reported.[17] Much of today's data is based on work by Lionel Cohen.[8] Generally, 135 kv., 2 mm. Al filter, and 240 kv., HVL 1.5 mm. Cu were used. Cohen plotted a dose time curve and found that for 85 per cent control rate the dose varied from a single 1,000 rad exposure to 2,500 rads in 4 weeks, depending on the size of the field.[8] Post irradiation gangrene occurred only in patients whose field length was greater than 20 cm. From these data a set of tolerance curves for irradiation of a limb was derived.[8] Other reports in the literature are anecdotal.

Systemic and intra-arterial chemotherapy have been used. Cook[9] has reported the use of intra-arterial nitrogen mustard with regression of nodules in 2 to 3 months with dosages of 0.4 mg. per kg. giving 15 per cent complete remission and 15 per cent improvement. Scott and Voight[28] reported 3 cases in whom they used vinblastine and obtained excellent results.

Goldman[14] reported use of laser irradiation on selected lesions with "fair" results.

Information about prognosis is almost absent from the African experience, since many patients live in the "Bush" and follow-up is poor. The usual course of the disease is a chronic one with varying periods of disease flare-ups. Brasfield and O'Brien[4] found a mean survival of 9 years. The "lymphoma-type" of Kaposi's sarcoma is rapidly fatal, death occurring as swiftly as within several months.

ZELLERBACH SARONI TUMOR INSTITUTE EXPERIENCE

Ten patients with Kaposi's sarcoma were seen between August 1966 and April 1972 at the Zellerbach Saroni Tumor Institute of Mount Zion Hospital and their records have been reviewed and updated

TABLE I

SUMMARY OF PERTINENT DATA OF 10 PATIENTS

Patient	Sex	Age (yr.)	Time from Histol. D_x (mo.)	Religion	Country of Origin	Sites Involved	Status
MS	M	59	47	Jewish	Unknown	Rt. foot	Alive—NED
BS	F	70	56	Jewish	Canada (Russia and Poland)	Rt. and Lt. feet and calves	Alive—with disease
MD	F	77	70	Jewish	Nebraska (Russia and Poland)	Rt. calf	Alive—NED
RS	F	75	26	Jewish	Hungary	Rt. calf	Alive—NED
JO	M	84	5	Catholic	Poland	Rt. foot, Lt. foot and knee	Dead (MI)—NED
JT	M	100	22	Jewish	Russia	Rt. and Lt. feet	Dead (GI bleeding) with disease
CP	M	67	40	Catholic	Italy	RLE, LLE, Rt. and Lt. forearms, trunk, ear, penis, inguinal lymph nodes	Dead—with disease (MI)
BZ	F	78	31	Jewish	Poland	Lt. foot	Alive—with disease
MS	M	85	2	Jewish	Poland	Rt. and Lt. feet and ankles and calves	Alive—NED
BR	M	88	2	Jewish	Romania	Lt. knee, buccal mucosa	Alive—NED

Rt. = right; Lt. = left; RLE = right leg; LLE = left leg; NED = no evidence of disease; MI—myocardial infarction; GI—gastrointestinal.

to May 1972. All slides have been reviewed by Mount Zion Hospital pathologists and verified as Kaposi's sarcoma. Time from histologic diagnosis to the most recent updating varied from 1 month to 5 years (Table 1).

Ages ranged from 59 to 100 years. The median age was 77 years. There were 4 females and 6 males.

Eight of the 10 patients were Jewish.

Countries of origin were: Poland (3); Russia (1); Italy (1); Romania (1); and Hungary (1). Two patients from Canada and Nebraska each had parents who had emigrated from Russia and Poland.

Not one of the 10 patients had ever smoked, either recently or in their youth.

The 4 women were all housewives; 3 men were tailors; there was 1 barber, 1 butler and 2 construction workers. Some had retired many years before their illness began.

Associated diseases were those expected in an elderly population. One patient had a second primary malignant lesion (carcinoma of the tongue treated in 1960) and he was without evidence of disease. One patient had varicose veins of the lower extremities, antecedent to the onset of Kaposi's sarcoma.



Fig. 2. (*A*) The left knee is markedly involved with Kaposi's sarcoma. (*B*) The same area 10 weeks after completion of a course of irradiation (2,000 rads in 2 weeks MPD, Co^{60} with bolus) with resolution of the lesions.

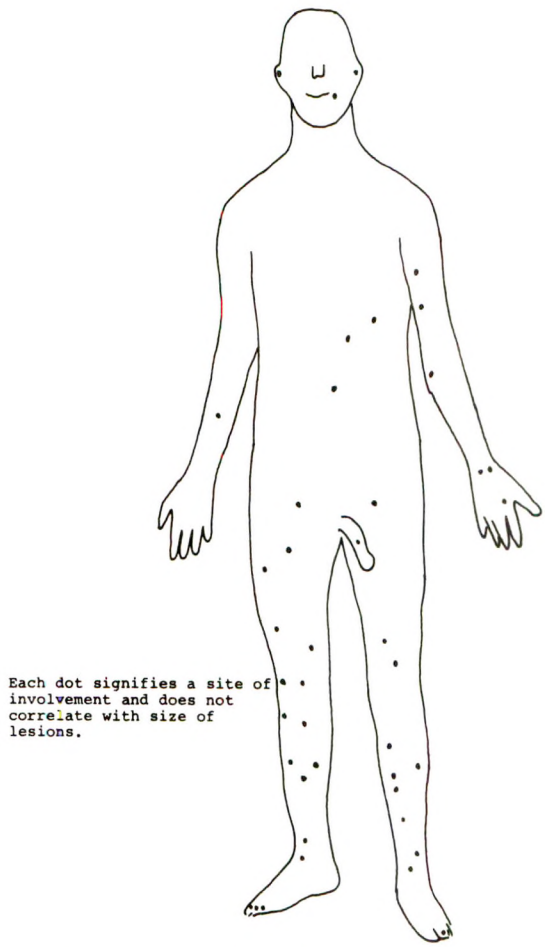


Fig. 3. Distribution of lesions in patients with Kaposi's sarcoma.

The lesions were generally violaceous, well demarcated, nontender papules, nodules or sometimes macules, ranging from less than 1 cm. to up to 15 cm.; they were usually multiple and when large had gotten that way by coalescence (Fig. 2*A*).

In each case the presenting sign was painless discoloration of a lower extremity. Most lesions were on the lower or upper extremities. In addition, lesions were found on the penis (1), inguinal lymph nodes (2), ears (2), buccal mucosa (1), and trunk (2) (Fig. 3). Seven patients had lesions confined to the extremities.

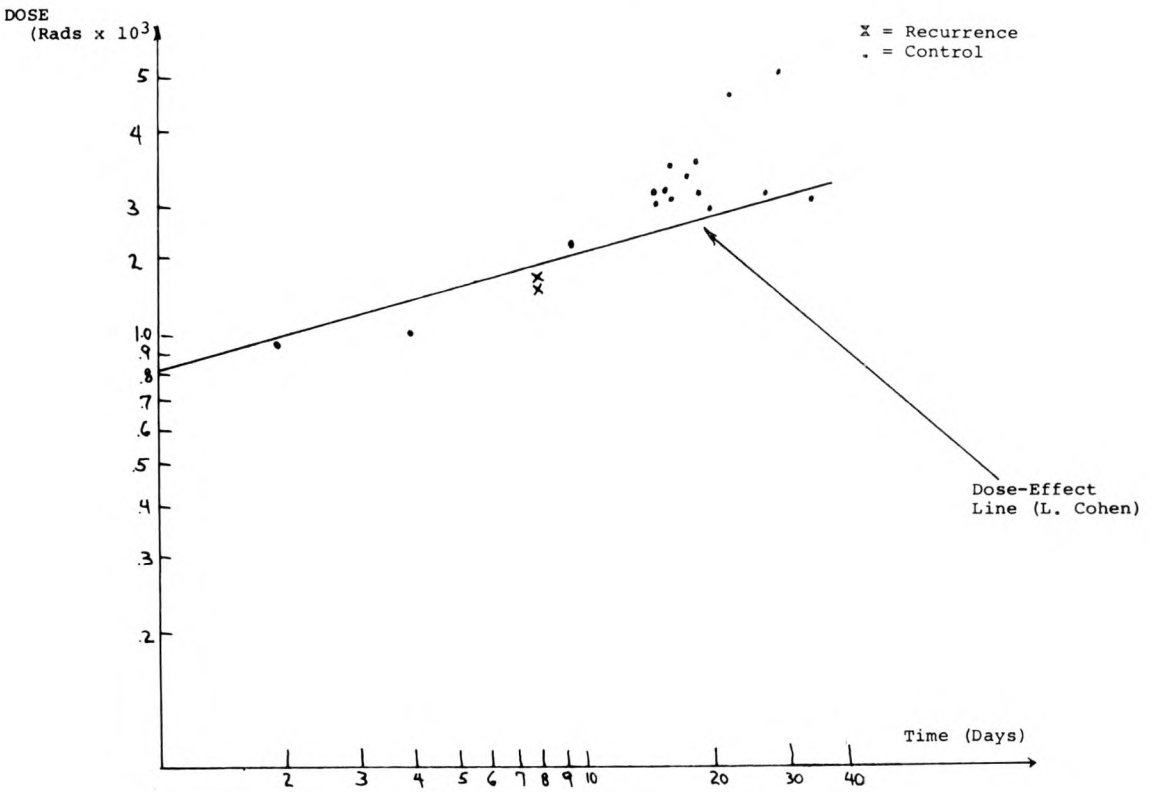


FIG. 4. Dose time curve for Kaposi's sarcoma.

The dose and time for recurrent and controlled lesions have been plotted and are superimposed on the iso-effect line as derived by Cohen[8] (Fig. 4). Two lesions recurred in 1 patient. In general, lesions began regressing in the middle or near the end of therapy and persisted in resolving up to 4 months post therapy (Fig. 2*B*).

There were 25 lesions treated by cryosurgery. All were 1 cm. or less in diameter. Each was treated under local anesthesia with a single application of liquid nitrogen (−160°C.), lasting from less than 30 seconds up to 1 minute. One lesion recurred, but was controlled with a second application. All healed without complication (Fig. 5, *A* and *B*). Generally, by 1 week a



FIG. 5. (*A*) A lesion of the popliteal fossa. (*B*) The same lesion 9 weeks post cryosurgery is smaller and retracted.

crust formed over the lesion and in 3 weeks it totally healed with residual hyperpigmentation.

Of 10 patients, 7 are still alive and 3 have expired. Only 1 of the 3 had an autopsy. One of the 2 without autopsy died from complications of an acute myocardial infarction, and the second had a gastrointestinal hemorrhage. He had a previous polypectomy for a colonic polyp; it was not known if his gastrointestinal tract was involved. The patient who had an autopsy had visceral involvement limited to his stomach; cause of death was an acute myocardial infarction.

Except for the older age group and the larger number of women than would otherwise be expected, the epidemiology of our patients compares similarly to other studies in the literature. The small number and short follow-up preclude conclusions about mortality.

Of interest, however, is the indolent course of the disease in the 4 women. They have been followed for 2 to 5 years. None have lesions above the knees and they all received 1 to 3 courses of irradiation only. An indolent course of the disease in women has been found also by Brasfield and O'Brien.[4]

CONCLUSION

Kaposi's sarcoma is a rare disease in the United States, but should be suspected when one is presented with purplish discoloration of a lower limb in a geriatric patient. A skin biopsy invariably yields the diagnosis.

The disease can be expected to follow a chronic course and can be treated very effectively with irradiation in low doses, with essentially no morbidity. If it occurs in the female, there is only a small chance that it will affect the patient's life expectancy. Cryosurgery is a rapid, safe, and highly successful method of controlling local lesions.

Further evidence is required to establish Kaposi's sarcoma as a lymphoma.

SUMMARY

Kaposi's sarcoma is uncommon in the U.S.A. and common in Africa. Men are affected at least 4 times more frequently than females. It occurs simultaneously with lymphomas and resembles them in several ways. Radiation therapy controls the disease well.

Ten patients were seen with the disease. Most had a relatively benign course, especially the 4 women.

Treatment with supervoltage irradiation and cryosurgery was effective in controlling local disease.

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REFERENCES

1. Andrews, J. R. Dose-time relationships in cancer radiotherapy: clinical radiobiology study of extremes of dose and time. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *93*, 56–74.
2. Bhana, D. Kaposi's sarcoma of lymph nodes. *Brit. J. Cancer*, 1970, *24*, 464–470.
3. Bluefarb, S. M. Kaposi's Sarcoma. Charles C Thomas, Publisher, Springfield, Ill., 1957.
4. Brasfield, R., and O'Brien, P. Kaposi's sarcoma. *Cancer*, 1966, *19*, 1497–1502.
5. Brownstein, M. H., Shapiro, L., and Skolnik, P. Kaposi's sarcoma in community practice. *A.M.A. Arch. Dermat. & Syph.*, 1973, *107*, 137–138.
6. Brunning, R. D., Folley, J. S., and Fortuny, J. F. Hodgkin's disease and Kaposi's sarcoma. *A.M.A. Arch. Int. Med.*, 1963, *112*, 363–369.
7. Burkitt, D. Host defense mechanisms in lymphoma and Kaposi's sarcoma: clinical evidence. *Brit. M. J.*, 1970, *4*, 424–426.
8. Cohen, L. Dose, time and volume parameters in radiation therapy of Kaposi's sarcoma. *Brit. J. Radiol.*, 1962, *35*, 485–488.
9. Cook, J. Kaposi's sarcoma treated with nitrogen mustard. *Lancet*, 1959, *1*, 25–26.
10. Cook, J. Clinical features in East African Bantu of Kaposi's sarcoma. *Acta Unio internat. contra cancrum*, 1962, *18*, 75–80.
11. Davies, J. N., and Lothe, F. Kaposi's sarcoma

in African children. *Acta Unio internat. contra cancrum*, 1962, *18*, 81–85.
12. Dutz, W., and Stout, A. P. Kaposi's sarcoma in infants and children. *Cancer*, 1960, *13*, 684–694.
13. Ehrenfield, I. D. Kaposi's sarcoma and lymphoblastoma. *A.M.A. Arch. Dermat. & Syph.*, 1970, *10*, 610–611.
14. Goldman, L. Laser radiation of malignancy in man. *Cancer*, 1965, *18*, 533–534.
15. Kaposi, M. Idiopathisches multiples Pigmentsarkom der Haut. *Arch. Derm. u. Syph.*, 1872, *4*, 265–273.
16. Lothe, S., and Murray, J. Kaposi's sarcoma: autopsy findings in Africa. *Acta Unio internat. contra cancrum*, 1962, *18*, 116–139.
17. McCarthy, W. D., and Pack, G. T. Malignant blood vessel tumors. *Surg., Gynec & Obst.*, 1950, *91*, 465–482.
18. Murray, J. F., and Lothe, S. Histopathology of Kaposi's sarcoma. *Acta Unio internat. contra cancrum*, 1962, *18*, 100–115.
19. Naylick, A. Angiography in Kaposi's sarcoma. *Clin. Radiol.*, 1963, *14*, 304–309.
20. Niemin, M., and Mustakallio, K. K. Fine structure of spindle cell in Kaposi's sarcoma. *Acta path. microbiol., scandinav.*, 1965, *63*, 567–575.
21. Oettle, A. G. Geographical and racial differences in frequency of Kaposi's sarcoma as evidence of environment or genetic causes. *Acta Unio internat. contra cancrum*, 1962, *18*, 17–22.
22. Palmer, P. E. S. Radiological changes of Kaposi's sarcoma. *Acta Unio internat. contra cancrum*, 1962, *18*, 87–91.
23. Rothman, S. Some remarks on Moricz Kaposi and on history of Kaposi's sarcoma. *Acta Unio internat. contra cancrum*, 1962, *18*, 1–2.
24. Rothman, S. Remarks on sex, age and race distribution of Kaposi's sarcoma and on possible pathogenetic factors. *Acta Unio internat. contra cancrum*, 1962, *18*, 13–15.
25. Rothman, S. Some clinical aspects of Kaposi's sarcoma in European and North American population. *Acta Unio internat. contra cancrum*, 1962, *18*, 8–12.
26. Rylwin, A. M., Recher, L., and Hoffman, E. Lymphoma-like presentation of Kaposi's sarcoma. *A.M.A. Arch. Dermat. & Syph.*, 1966, *93*, 554–561.
27. Scott, R. M., and Brizel, H. E. Time-dose relationships in Hodgkin's disease. *Radiology*, 1964, *82*, 1043–1048.
28. Scott, W. P., and Voight, J. A. Kaposi's sarcoma management with vincaleukoblastine. *Cancer*, 1966, *19*, 557–564.
29. Slavin, G., Cameron, H. McD., and Singh, H. Kaposi's sarcoma in mainland Tanzania: report of 117 cases. *Brit. J. Cancer*, 1969, *23*, 349–357.
30. Wechsler, H. L. Kaposi's sarcoma: angiographic findings before and after orthovoltage treatment. *A.M.A. Arch. Dermat. & Syph.*, 1967, *96*, 69–70.

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UROGRAPHIC MANIFESTATIONS OF METASTATIC MELANOMA*

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THE frequency of genitourinary tract metastases from malignant melanoma is comparable to the more widely recognized gastrointestinal tract involvement. In one autopsy series of 100 patients with metastatic melanoma renal involvement was noted in 45 per cent, while the bladder was affected in 18 per cent.[5] In another postmortem series from the M. D. Anderson Hospital and Tumor Institute, the kidneys were involved in 58 per cent of the cases. Only the lungs, liver, and lymphatic system were more frequent sites of metastases. The bladder was affected in 14 per cent.[6]

As with other metastatic neoplasms to the urinary tract, melanoma is not as frequently recognized clinically as at autopsy.[3] In part, this is due to a reluctance to investigate patients with widely disseminated disease. In addition, there appears to be a lack of familiarity with urinary tract involvement and especially the roentgenologic appearance.[1,4,11–13]

The purpose of this communication is to present our experience with the roentgenologic manifestations of metastatic melanoma involving the urinary tract, in particular the appearance of urothelial metastases of the renal pelvis, ureters, and bladder, which has not been previously described in the radiologic literature.

CLINICAL MATERIAL

Seventy-one patients with metastatic malignant melanoma to the urinary tract were evaluated at the M. D. Anderson Hospital and Tumor Institute from January, 1968 to August, 1973. There were 48 males and 23 females, ranging in age from 18 to 81 years. The skin was the primary malignant site in the vast majority of cases. Virtually every patient had widely disseminated disease, and all patients have since expired. In 55 of the cases, the presence of urinary tract involvement was confirmed at autopsy, while the diagnosis in the remaining 16 patients was based on surgical, cystoscopic, and/or clinical evidence.

Of the 71 patients, the kidneys were affected in 55, the urinary bladder in 20, the ureters in 5, and the renal pelvis in 4.

In approximately half the cases, urinary tract involvement was an incidental finding of widespread disease clinically or at postmortem examination, and no symptoms were apparent. The most common finding that prompted urinary tract investigation in approximately one-fourth of the patients was gross or microscopic hematuria. Evaluation of a suspected abdominal mass, symptoms of urinary tract infection, and azotemia accounted for most of the remainder.

ROENTGENOGRAPHIC FINDINGS

Intravenous pyelographic studies were performed in 22 patients and available for review in 20. At autopsy or clinically, the renal parenchyma was involved in 9, the urinary bladder in 8, the ureters in 5, and the renal pelves in 4. As a reflection of the widely disseminated metastases, the mean survival following pyelographic examination was 68 days. Of the 20 patients with urographic examinations, there were definite abnormalities present in 14.

RENAL PARENCHYMA

Melanoma, as does other metastatic diseases affecting the renal parenchyma,

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typically produces multiple small cortical nodules, usually not of sufficient size to become clinically apparent.[1,3] Of our 9 patients with renal metastases, 6 had small macroscopic lesions measuring from several millimeters to 2 centimeters, while 3 had only microscopically detectable deposits. There was no example of parenchymal metastases large enough to produce a renal mass diagnosed by urography. To our knowledge, there is only one roentgenographic example of metastatic melanoma mimicking a renal carcinoma that has been described.[2] In several of our cases, one or both kidneys were depressed inferiorly by involved adrenal glands. However, similar to renal metastases, the adrenals are usually not enlarged when involved with melanoma.

RENAL PELVES AND URETERS

The least commonly affected areas in the urinary tract were the renal pelves and ureters. Subepithelial metastases provided the most striking and potentially confusing roentgenographic appearance. All 4 cases with renal pelvic deposits were detected roentgenographically. In 2 of these cases, single filling defects were noted in the renal pelvis and resembled a primary epithelial neoplasm, calculus, or blood clot. Bilateral nonobstructing ureteropelvic junction filling defects were present in another (Fig. 1). In the 4th case, multiple bilateral crescentic filling defects were present in the renal pelves and ureters, not unlike pyeloureteritis cystica or multiple papillary tumors (Fig. 2, *A* and *B*).[8,9]

Of the 5 cases with intrinsic ureteral disease, pyelographic abnormalities were present in 4. In 2 of these patients, the lesions were almost totally obstructing and resulted in hydronephrosis (Fig. 3, *A–C*). In the other 2 patients, the filling defects were nonobstructing. One of these patients with multiple bilateral small deposits involving pelves and ureters was discussed above. The other patient demonstrated prominent polypoidal filling defects confined to both distal ureters.

Three additional patients had extrinsic obstruction of one or both ureters from retroperitoneal nodal involvement. Deviation of the ureters without obstruction was present in 2 other cases.

BLADDER

Accuracy in roentgenologic diagnosis of bladder metastases was less than the upper collecting structures, with 3 of 8 cases demonstrating abnormalities. Two of the patients were among those with ureteral involvement. One of these patients had numerous round, irregular filling defects throughout the bladder (Fig. 4). Differential diagnostic considerations for this appearance would include cystitis cystica, bullous edema, and malacoplakia.[7] The third patient had direct extension to the bladder neck from a primary melanoma of the vulva.

DISCUSSION

Metastatic melanoma spreads to the genitourinary tract *via* several routes. Direct extension to urinary tract structures

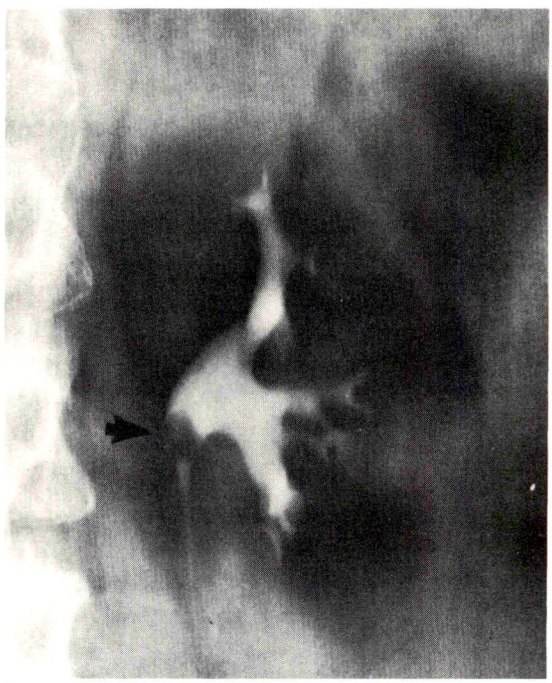


FIG. 1. Nephrotomogram demonstrates a smooth filling defect at the ureteropelvic junction (arrow) secondary to metastatic melanoma.

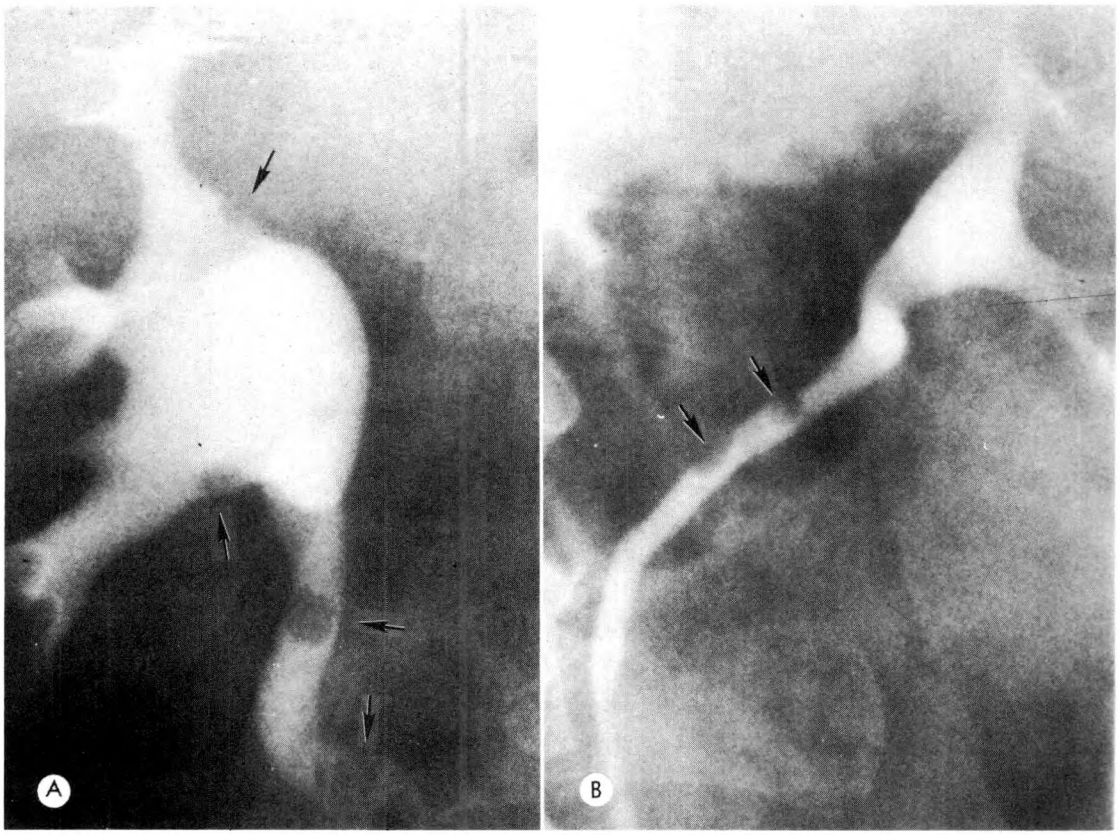


Fig. 2. Bilateral metastatic melanoma resembling pyeloureteritis cystica. (*A*) Intravenous pyelogram shows several crescentic and round filling defects of the right pelvis and proximal ureter (arrows). (*B*) Two discrete crescentic filling defects are seen in the proximal left ureter (arrows).

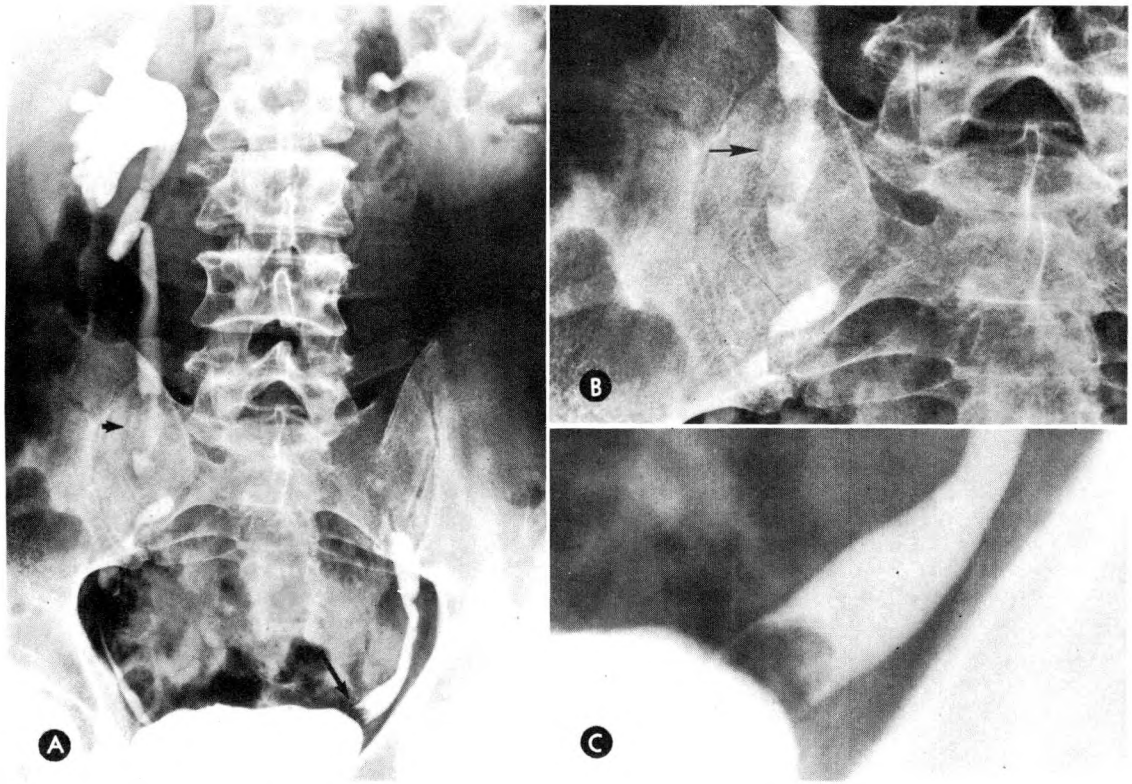
may occur from adjacent invaded lymph nodes or adrenal glands. Dissemination *via* lymphatics may also occur, especially when there is involvement of retroperitoneal lymph nodes with obstruction to the lymphatic flow. The diffuse nodular metastases which are usually present indicate that the usual mode of dissemination is hematogeneous.

Primary melanoma arising in the genitourinary tract is rare. In one series of 1,200 cases of malignant melanoma, only 6 cases originated in the genitourinary tract; not one originated in the kidney parenchyma or urinary tract epithelium.[5] Two of these cases actually arose from the skin of the penis, while the others were of mucocutaneous urethral origin in women.

Hematuria was most common in patients with metastases to the renal pelves and/or ureters, being present in all but 1 of these cases. Half of the patients with bladder metastases had hematuria, and 1 patient had dark-colored urine with melanin pigment.

Cystoscopic findings of bladder metastases may vary with lesions ranging in size and configuration from tiny black or blue-black areas confined to the mucosa to pedunculated or sessile invasive tumors (Fig. 5). In the latter situation, necrosis and inflammation may obscure the natural blue-black coloration and result in the lesion being confused with a primary bladder neoplasm. Double contrast barium cystography may complement cystoscopy and enhance the accuracy in the diagnosis of bladder metastases.[10]

In virtually all instances when metastatic melanoma involves the urinary tract, it is part of an already widely disseminated process with cure most unlikely. Famil-



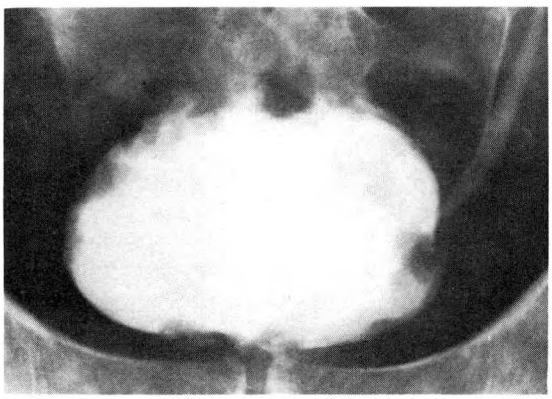


FIG. 4. Multiple crescentic and irregular metastases involve the entire bladder.

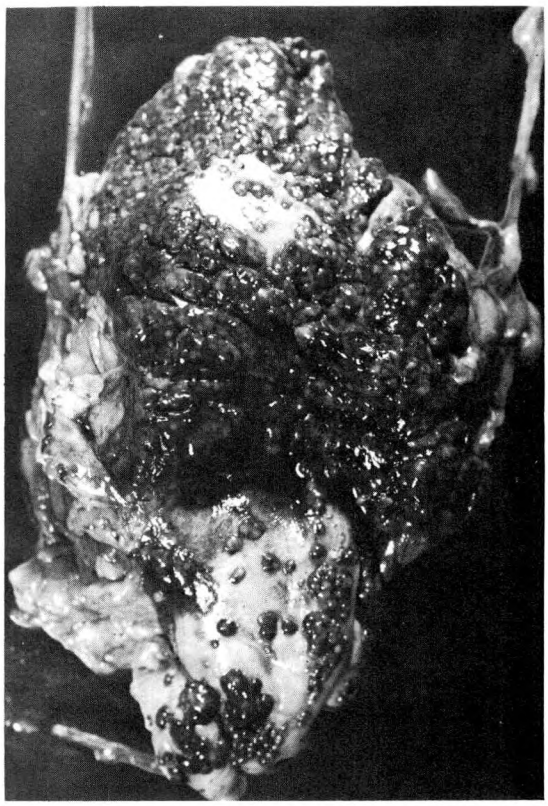


FIG. 5. Gross specimen of an open bladder showing numerous black, solitary and conglomerate metastatic melanoma deposits.

Fig. 3. Metastatic melanoma involving both ureters. (*A*) Intravenous pyelogram demonstrates a constricting lesion of the right mid-ureter (short arrow), causing moderate hydronephrosis and a polypoidal deposit in the distal left ureter (long arrow). (*B*) Close-up view of the annular melanoma metastasis of the right ureter (arrow). (*C*) Close-up view of the smooth, ovoid deposit in the very distal left ureter.

iarity with the possible roentgen appearances, particularly of urothelial melanoma, may prevent a time-consuming and expensive evaluation for other etiologies, or prevent unnecessary surgical resection for a suspected primary epithelial tumor. On the other hand, recognition may occasionally aid in the palliative management of a clinical situation such as intractable hematuria. Although the treatment of metastatic melanoma to the urinary tract is the treatment of the primary disease itself, localized symptoms may occasionally warrant surgical intervention.

SUMMARY

Urinary tract involvement with metastatic malignant melanoma occurs much more commonly than generally thought. It is usually part of a disseminated process with a poor life expectancy.

The renal parenchyma is the most common site affected, although this is infrequently detected roentgenographically. Urothelial involvement of the renal pelves, ureters, and bladder manifests as single or multiple filling defects at pyelography. This appearance of metastatic melanoma of the urinary tract has not been previously described in the roentgenologic literature.

Recognition is important to prevent unnecessary intervention and to guide palliative treatment.

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REFERENCES

1. Abeshouse, B. S. Primary and secondary melanoma of genitourinary tract. *South. M. J.*, 1958, *51*, 994–1006.
2. Agnew, C. H. Metastatic malignant melanoma of kidney simulating primary neoplasm. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1958, *80*, 813–816.
3. Bosniak, M. A., Stein, W., Lopez, F., Tehranian, N., and O'Connor, S. Metastatic neoplasm to kidney. *Radiology*, 1969, *92*, 989–993.
4. Brown, R. C., Jones, M. C., Boldus, R., and Flocks, R. H. Lesions causing radiolucent defects in renal pelvis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 770–778.
5. Das Gupta, T., and Grabstald, H. Melanoma of genitourinary tract. *J. Urol.*, 1965, *93*, 607–614.
6. Einhorn, L. H., Burgess, M. A., Vallejos, C., Bodey, G. P., Sr., Gutterman, J., Mavligit, G., Hersh, E. M., Luce, J. K., Frei, E., III, Freireich, E. J., and Gottlieb, J. A. Advanced metastatic melanoma: prognostic correlations and response to treatment in 426 patients. To be published.
7. Elliott, G. B., Maloney, P. J., and Clement, J. G. Malacoplakia of urinary tract. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 830–837.
8. Kaplan, J. H., McDonald, J. R., and Thompson, G. J. Multicentric origin of papillary tumors of urinary tract. *J. Urol.*, 1951, *66*, 792–804.
9. Loitman, B. S., and Chiat, H. Ureteritis cystica and pyelitis cystica: review of cases and roentgenologic criteria. *Radiology*, 1957, *68*, 345–351.
10. Pochaczevsky, R., and Grabstald, H. Double contrast barium cystography utilizing carbon dioxide. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 365–374.
11. Samellas, W., and Marks, A. R. Metastatic melanoma of urinary tract. *J. Urol.*, 1961, *85*, 21–23.
12. Tsuau, S., and Prince, C. L. Melanoma of bladder. *J. Urol.*, 1962, *87*, 365–367.
13. Weston, P. A. M., and Smith, B. J. Metastatic melanoma in bladder and urethra. *Brit. J. Surg.*, 1964, *51*, 78–79.

THE INCREASED INCIDENCE OF CARCINOMA OF THE COLON FOLLOWING URETEROSIGMOIDOSTOMY*

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WHILE the occurrence of hydronephrosis secondary to scarring at the anastomotic site is a common complication of ureterosigmoidostomy,[17,19] urinary obstruction and other complications secondary to a presumably induced[6] neoplasm of the colon[1–5,7–14,16,18,20] are rather uncommon, and to our knowledge have not been reported in the roentgenologic literature.

We have found 21 previously reported cases and wish to present a case of our own and review the reported roentgenologic features.

REPORT OF A CASE

A 34 year old Caucasian male was admitted in December, 1972, with left inguinal intermittent colicky pain of 2 weeks' duration. The patient denied any bowel complaints. A shotgun wound to the patient's pelvis at 8 years of age had necessitated removal of his urinary bladder and establishment of a bilateral ureterosigmoidostomy. The patient had been seen numerous times in the interim for pyelonephritis and various plastic surgical procedures. The patient was afebrile and no costovertebral angle tenderness was noted. Serum creatinine was 0.7 mg. per cent. The complete blood cell count was unremarkable.

Intravenous urography revealed bilateral obstruction (Fig. 1). A barium enema examination was performed (Fig. 2) which strongly suggested carcinoma of the colon.

At surgery numerous peritoneal and hepatic nodules were noted. A left nephrectomy and descending loop colostomy were performed. The final pathologic diagnoses were metastatic mucinous adenocarcinoma and hydronephrosis of the left kidney. The patient was discharged after a somewhat stormy postoperative course.

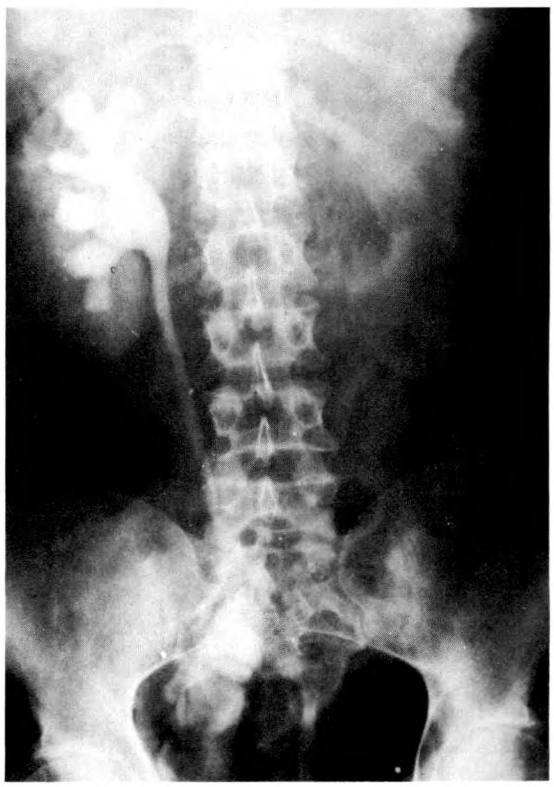


FIG. 1. A drip intravenous urogram shows prompt excretion on the right. The diuretic effect of high dose urography accentuates the dilatation of the collecting system secondary to partial obstruction. On the left, there is delayed excretion. The nephrogram effect shows large radiolucent areas surrounded by a rim effect. These dilated calyces subsequently showed faint filling confirming the obstructive uropathy. Contrast material in the rectum and rectosigmoid failed to reflux into the descending colon.

DISCUSSION

The first ureterosigmoidostomy was performed by Simon[15] in 1852, while Hammer

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in 1929[7] described the first patient with a colonic tumor obstructing the implanted ureters. Since then, 20 other cases of tumors of the colon in such patients have been reported: the present case brings the total to 22.

Tables I and II outline the reported material demonstrating the original reason for ureterosigmoidostomy and the type of tumor found in the colon. One other patient developed a transitional cell carcinoma at the ureterocolic anastomosis 17 years after surgery for exstrophy of the urinary bladder. Although this lesion presented roentgenographically as a colonic polyp, it was derived from urinary epithelium and is not included in our Tables.[16]

These primary colon neoplasms were almost surely induced in some way by the ureterosigmoidostomy and resulting urine flow through the sigmoid, as evidenced by the very young age of several of the patients. The 19 patients developing carcinoma that were reviewed averaged 40 years of age; 5 were under 26 years of age. Urdaneta *et al.*[18] reviewed 23 cases with ureterosigmoidostomy that survived 10 years or more. Three of these developed sigmoid carcinoma, yielding an incidence of 13,300 per 100,000 in this group compared to an expected incidence of 2.3 per 100,000 for carcinoma of the sigmoid colon for patients at large (National Health Statistics). It is important for the physician to be aware of this 6,000 fold increased incidence of colon neoplasms in these patients, especially those of young age in

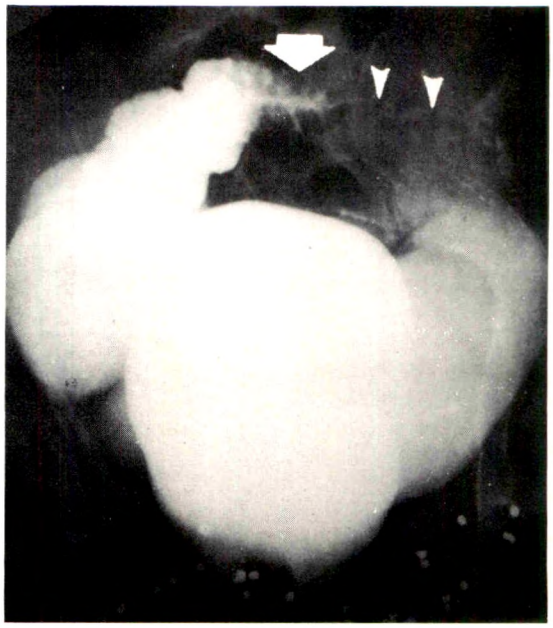


FIG. 2. A barium enema examination demonstrates a narrowed area in the sigmoid colon. The area below the large arrow shows irregular mucosa with small polypoid defects. The area under the smaller arrows reveals a narrow, irregular lumen with loss of mucosa. This appearance is indicative of colonic cancer. Note the multiple metallic pellets.

TABLE I

PRIMARY COLON ADENOMAS

Author—Year	Reason for Original Surgery	Age Sex	Years after Surgery	Type of Tumor	Site of Tumor	Roentgenologic Findings
Dixon and Weismann, 1948[4]	Exstrophy	33/M	30	Adenomatous polyps (2)	Near both ureteral anastomoses	IVU—Nonfunctioning left kidney
Ellis, 1962[5]	Chronic cystitis (abacterial)	55/M	15	Adenomatous polyp	Left ureteral anastomosis	
Gillman, 1964[6]	Transitional cell papilloma	82/M	7	Adenomatous polyps (3)	Left ureteral anastomosis	BEE—Several small polyps in lower descending colon

IVU = Intravenous urography.
BEE = Barium enema examination.

TABLE II

PRIMARY COLON CARCINOMA

Author—Year	Reason for Original Surgery	Age Sex	Years after Surgery	Type of Tumor	Site of Tumor	Roentgenologic Findings
Hammer, 1929[7]	Exstrophy	50/M	10	Colloid carcinoma	Both ureteral anastomoses	
Wilson, 1957[20]	Congenital urinary incontinence	47/M	30	Anaplastic carcinoma	Both ureteral anastomoses	
Aldis, 1961[1]	Exstrophy	33/F	15	Scirrhous carcinoma	Left ureteral anastomosis	IVU—Left kidney showed poor function with dilated ureter and pelvis
Hanley, 1962[8]	Persistent urinary fistulae	22/M	15	Mucinous adenocarcinoma	Left ureteral anastomosis	
Kozak *et al.*, 1966[10]	Exstrophy	17/M	15	Mucinous adenocarcinoma	Left ureteral anastomosis	IVU—Nonvisualization of left kidney. BEE—Questionable defect in sigmoid attributed to previous surgery
Urdaneta *et al.*, 1966[18]	Chronic cystitis	40/F	17	Mucinous adenocarcinoma	Right ureteral anastomosis	BEE—"Applecore" lesion in sigmoid with sinus tract to abscess
Urdaneta *et al.*, 1966[18]	Squamous cell carcinoma of bladder	64/M	9	Adenocarcinoma	Right ureteral anastomosis	IVU—"Sudden unilateral hydronephrosis"
Urdaneta *et al.*, 1966[18]	Exstrophy	34/F	30	Undifferentiated carcinoma	Diffuse	Abdomen—Colonic obstruction Gastrografin enema—Obstruction in distal sigmoid
Mass. Gen. Hosp., 1958[11]	Transitional cell carcinoma of bladder	63/M	8	Adenocarcinoma	Right ureteral anastomosis	IVU—Nonfunction of right kidney. BEE—Filling defect at anastomosis
Scheinman, 1961[14]	Transitional cell carcinoma of bladder	51/M	9	Mucinous adenocarcinoma	Left ureteral anastomosis	IVU—Negative. BEE—Filling defect at anastomosis
Amar, 1961[2]	Transitional cell carcinoma of bladder	64/M	6	Mucinous adenocarcinoma	Left ureteral anastomosis	
Oetjen *et al.*, 1970[13]	Exstrophy	23/M	23	Adenocarcinoma	Left ureteral anastomosis	IVU—Left hydronephrosis at age 15. Nephrectomy at age 15. BEE—Colonic polyp at age 23
Mueller and Thornbury, 1973[12]	Exstrophy	55/M	46	Adenocarcinoma at each site	Separate tumors at each anastomosis	IVU—Progressive hydronephrosis for 3 years. BEE—Polypoid tumor at each anastomotic site
Brekkan *et al.*, 1972[3]	Exstrophy	23/F	20	Anaplastic carcinoma and adenocarcinoma	Separate tumors at each anastomosis	IVU—Nonfunctioning right kidney and left-sided hydronephrosis. BEE—Tumor at each anastomotic site
Brekkan *et al.*, 1972[3]	Exstrophy	44/M	38	Adenocarcinoma	Immediately below right ureterostomy	IVU—Silent right kidney and slight dilatation on the left 8 months before discovery of the sigmoid tumors
Brekkan *et al.*, 1972[3]	Exstrophy	47/M	41	Mucinous adenocarcinoma	Surrounding ureterosigmoid anastomosis	IVU—Nonfunctioning left kidney. BEE—Polypoid lesion at anastomotic site, first interpreted as trigone, but 2 months later suspected as being malignant
Kille and Glick, 1967[9]	Incontinence secondary to epispadias	19/M	15	Cystic mucinous polyp-malignant?	Near ureterocolic anastomosis	IVU—Small left kidney with dilated calyces and little function; normal 3 years prior. BEE—Large sigmoid polyp

TABLE II—(*Continued*)

Author—Year	Reason for Original Surgery	Age Sex	Years after Surgery	Type of Tumor	Site of Tumor	Roentgenologic Findings
Kille and Glick, 1967[9]	Incontinence secondary to epispadias	26/M	16	Adenocarcinoma	Left ureteral anastomosis	IVU—Minimal dilatation of pelvicocalyceal systems; normal prior study
Current Report	Gunshot wound	34/M	26	Mucinous adenocarcinoma	Sigmoid colon	IVU—Left hydronephrosis with poor function. BEE—Obstruction in sigmoid with mucosal destruction

IVU = Intravenous urography; BEE = Barium enema examination.

whom this diagnosis ordinarily might not be considered.

Since ileal loop diversion has, to a large extent, replaced ureterosigmoidostomy, we will probably be seeing less of this problem in the future. However, there are a considerable number of patients with ureterosigmoidostomies who will continue to receive longterm follow-up examinations, and ureterosigmoidostomy is still of use in selected patients, such as patients with skin sensitivity to urine. Development of cancer can be expected in some of these patients.

Lack of appreciation of this complication with resulting delay in obtaining a barium enema examination, possibly resulting in a "delay in diagnosis of adenocarcinoma," was reported in 1 case.[12] Almost every case report described urinary or large bowel complaints, some for a considerable duration before the diagnosis of neoplasm was made. Although the occurrence of ureteric obstruction, diarrhea, blood per rectum, or colon obstruction is not especially remarkable in patients with a recent ureterosigmoid transplant, their development many years after operation should alert the physician.

Presenting renal complaints will usually necessitate intravenous urography. The findings suggestive of obstruction at the anastomotic site should not be dismissed as being due to fibrosis, since this complication usually appears within 1 year and not later than 18 months postoperatively.[19] A barium enema examination should be obtained to rule out neoplastic involvement. A presenting bowel complaint such as melena will lead to proctoscopic examination and barium enema examination followed by intravenous urography to evaluate ureteral patency.

SUMMARY

Presumably induced tumors of the sigmoid colon, often in young patients, are now a well documented complication of ureterosigmoidostomy.

The physician must be aware of this association and should obtain appropriate roentgen studies.

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REFERENCES

1. Aldis, A. S. Carcinoma of colon following transplantation of ureters, and at site of transplantation. *Proc. Roy. Soc. Med.*, 1961, *54*, 159–160.
2. Amar, A. D. Neoplastic obstruction of ureterosigmoid anastomosis. *J. Urol.*, 1961, *86*, 334–335.
3. Brekkan, E., Colleen, S., Myrvold, H., du Rietz, B., Schnürer, L. B., and Fritjofsson, A. Colonic neoplasia: late complication of ureterosigmoidostomy. *Scandinav. J. Urol., Nephrol.*, 1972, *6*, 197–202.
4. Dixon, C. F., and Weismann, R. E. Polyps of sigmoid occurring 30 years after bilateral ureterosigmoidostomy for exstrophy of bladder. *Surgery*, 1948, *24*, 1026–1034.
5. Ellis, F. G. Adenocarcinoma complicating ureterosigmoidostomy. *Proc. Roy. Soc. Med.*, 1962, *55*, 100.
6. Gillman, J. C. Adenomatous polyp of bowel fol-

lowing ureterocolic anastomosis: case report and review of literature. *Brit. J. Urol.*, 1964, *36*, 264–270.

7. HAMMER, E. Cancer du colon sigmoide dix ans après implantation des uretères d'une vessie extrophiée. *J. urol. Paris*, 1929, *28*, 260–263.
8. HANLEY, H. G. Personal communication to Gillman.[6]
9. KILLE, J. N., and GLICK, S. Neoplasia complicating ureterosigmoidostomy. *Brit. M. J.*, 1967, *4*, 783–784.
10. KOZAK, J. A., WATKINS, W. E., and JEWELL, W. R. Neoplastic stomal obstruction: complication of ureterosigmoidostomy. *J. Urol.*, 1966, *96*, 691–696.
11. Massachusetts General Hospital, 1958. Case records (Case No. 44052). *New England J. Med.*, 1958, *258*, 244–247.
12. MUELLER, C. E., and THORNBURY, J. R. Adenocarcinoma of colon complicating ureterosigmoidostomy: case report and review of literature. *J. Urol.*, 1973, *109*, 225–227.
13. OETJEN, L. H., CAMPBELL, J. L., THOMLEY, M. W., and PARSONS, R. L. Carcinoma of colon following ureterosigmoidostomy: report of case. *J. Urol.*, 1970, *104*, 535–537.
14. SCHEINMAN, L. J. Tumor at site of ureterosigmoidostomy nine years postoperatively. *J. Urol.*, 1961, *85*, 934–939.
15. SIMON, J. Ectopia vesicae (absence of anterior walls of bladder and pelvic abdominal parieties): operation for directing orifices of ureters into rectum; temporary success; subsequent death; autopsy. *Lancet*, 1852, *2*, 568–570.
16. SUGG, W. L. Tumor at site of ureterosigmoidostomy. *Ann. Surg.*, 1962, *155*, 572–576.
17. THOMPSON, I. M., KAROW, W. F., and ROSS, G. Long-term results of ureteral reimplantation for trauma. *J. Urol.*, 1969, *102*, 308–315.
18. URDANETA, L. F., DUFFELL, D., CREEVY, C. D., and AUST, J. B. Late development of primary carcinoma of colon following ureterosigmoidostomy: report of three cases and literature review. *Ann. Surg.*, 1966, *164*, 503–513.
19. WILLIAMS, D. F., BURKHOLDER, G. V., and GOODWIN, W. E. Ureterosigmoidostomy: 15 year experience. *J. Urol.*, 1969, *101*, 168–170.
20. WILSON, L. L. Carcinomatous ureteric obstruction 30 years after ureterosigmoidostomy. *Australia & New Zealand J. Surg.*, 1957, *27*, 158–159.

DETECTION AND LOCALIZATION OF AN OCCULT VESICOENTERIC FISTULA*

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PNEUMATURIA and fecaluria are usually caused by a vesicoenteric fistula. The diagnosis of such a fistula can be confirmed by giving charcoal or mineral oil by mouth and noting its appearance in the urine. Other methods used to further document the presence of an occult fistula include cystoscopy, cystography and barium enema examination.[2–6] Methylene blue enemas at the time of cystoscopy have also been helpful. Bourne[1] had described a method of collecting urine samples before and after a barium enema examination and then roentgenographing the centrifuged samples. In most instances the presence of a fistula can easily be proved, but its location may remain in doubt.

Since localization of the fistula is important in order to help the surgeon select the best therapeutic approach, a method for localization of the fistulous site would be of value. This report summarizes a simple method for both confirming the presence of a vesicoenteric fistula and determining its location in a patient with chronic inflammatory bowel disease.

REPORT OF A CASE

A 43 year old woman (JHH #1194528) had had chronic nonspecific inflammatory bowel disease for 14 years, for which she had been treated with corticosteroids, ACTH, and Azulfidine. Abdominal surgery had been first performed in 1966 when a feeding tube broke producing intestinal obstruction. In 1969, she had the onset of chronic dysuria and had intractable polymicrobial urinary tract infections. The patient had multiple cystoscopic procedures, barium enema examinations and cystographies which were unsuccessful in revealing a fistula. Charcoal given by mouth, was detected in the urine. In 1971, the patient had a colostomy with bypass of the descending and sigmoid colon with removal of all adherent bowel from the bladder. There was some improvement in controlling the bladder infections, but she did continue to have low grade bacteriuria. In 1972 the patient had menometrorrhagia and underwent a hysterectomy. At this time the colostomy was taken down and some redundant colon resected. Following surgery the patient had progressively worsening dysuria with significant bacteriuria refractory to antimicrobial therapy. The tracer study was performed to localize the site of fistula.

Preoperative Study. The patient was well hydrated (urine flow approximately 10 cc./min.) before the study; 100 μc of Au^{198} colloid was mixed with 100 cc. of water and given to the patient by mouth. An indwelling urinary catheter was inserted and urine was collected at intervals of 15 minutes for 2 hours. The activity in 2 cc. aliquots was immediately determined in a gamma well counter (baseline 350 kev., window 100 kev.) at the end of each collection period. The patient was placed under the detector of a scintillation camera equipped with a high energy diverging collimator, and 50,000 count scintiphotos of the distribution of activity in the gastrointestinal tract were obtained at regular intervals for 2 hours in the anterior view. When the activity appeared in the urine, scans were obtained in oblique and lateral views in addition to the anterior view to determine the location of the Au^{198} colloid in the gastrointestinal tract. Scintiphotos were also obtained at a later time to determine the transit time of tracer past the fistulous site.

Seventy-two hours later when the activity in the urine had returned to background, a barium enema examination was performed. Mixed with the barium was 50 μc of Au^{198} colloid. The patient was hydrated before the study as above and also had a Foley catheter in place. The barium was allowed to flow slowly into the colon. Roentgenograms were taken of the bar-

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ium at fixed anatomic locations (*e.g.*, sigmoid colon, descending colon, splenic flexure, etc.) and urine samples were collected simultaneously. The activity in 2 cc. aliquots of urine was determined immediately after collection in a gamma well counter. When activity was noted in the urine, multiple views were obtained to see if barium could be detected entering the bladder.

Following the radioisotope study, the patient had an exploratory laparotomy and it was noted that the cecum was adherent to the bladder. After separation of the cecum from the bladder, Evans blue dye was put into the bladder under pressure to attempt to visualize the fistula tract. No dye was seen in the area where the cecum was attached to the bladder. The patient had remained free of urinary tract infection for 5 months after surgery, the last time she was examined.

Postoperative Study. Four months following surgery, the patient was again given Au^{198} colloid by mouth. A Foley catheter was not used, but the patient was instructed to clean the genital area with sterile saline before voiding. Activity in both the stool and urine was determined. No attempt was made to obtain gamma camera images.

RESULTS

Preoperative Study. Figure 1, *A–C*, shows the movement of the Au^{198} through the gastrointestinal tract. The Au^{198} appeared in the urine between the image taken at 1 hour and 30 minutes and 1 hour and 45 minutes. This suggested that the fistula was located in the area involving the terminal ileum and ascending colon. When the barium enema was performed (Fig. 2, *A* and *B*), there was no appearance of the tracer in the urine until the barium reached

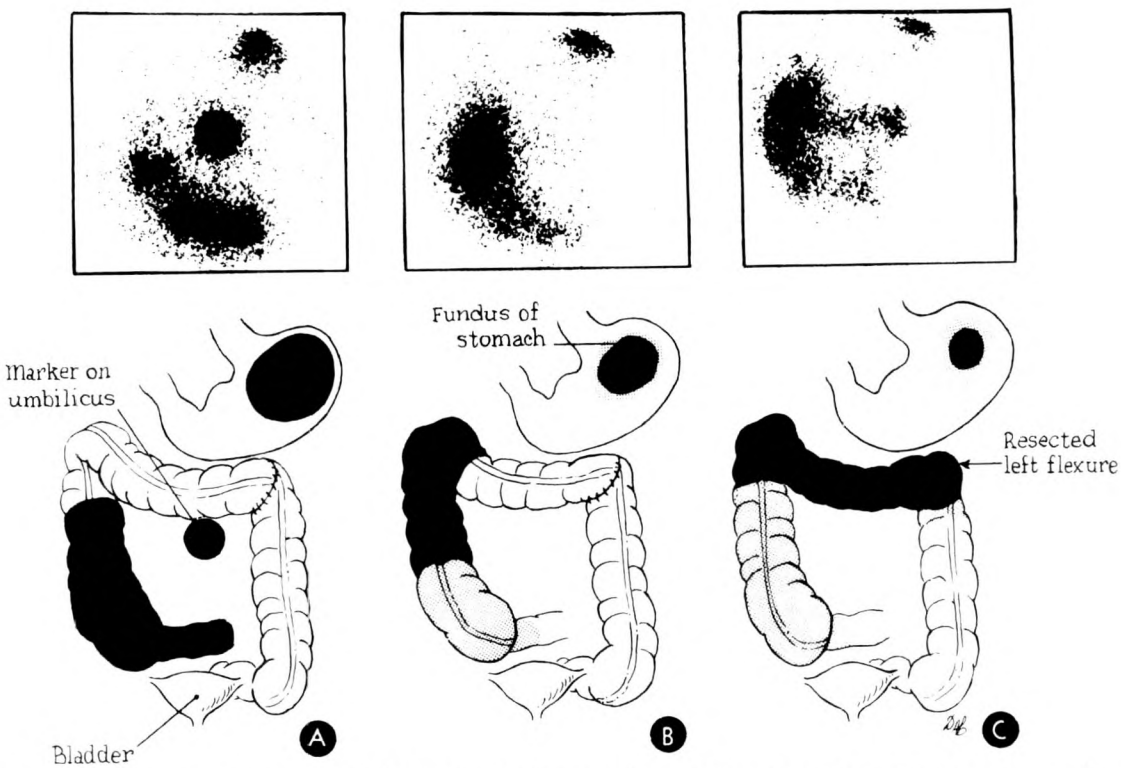


FIG. 1. Scintiphotos of Au^{198} colloid distribution in the gastrointestinal tract. (*A*) Distal ileum and ascending colon. (*B*) Ascending colon and hepatic flexure. (*C*) Hepatic flexure and transverse colon. Below the scintiphotos, the anatomic location of the activity is diagrammed. Activity appeared in the urine between the first and second images. This indicates that the connection between the gastrointestinal tract and the bladder is in the region of the terminal ileum and ascending colon. As can be seen there is still some activity in the stomach, since the emptying time of the stomach in this patient is approximately equal to the transit time in the bowel. Also the latter image helped define the anatomic location more easily.

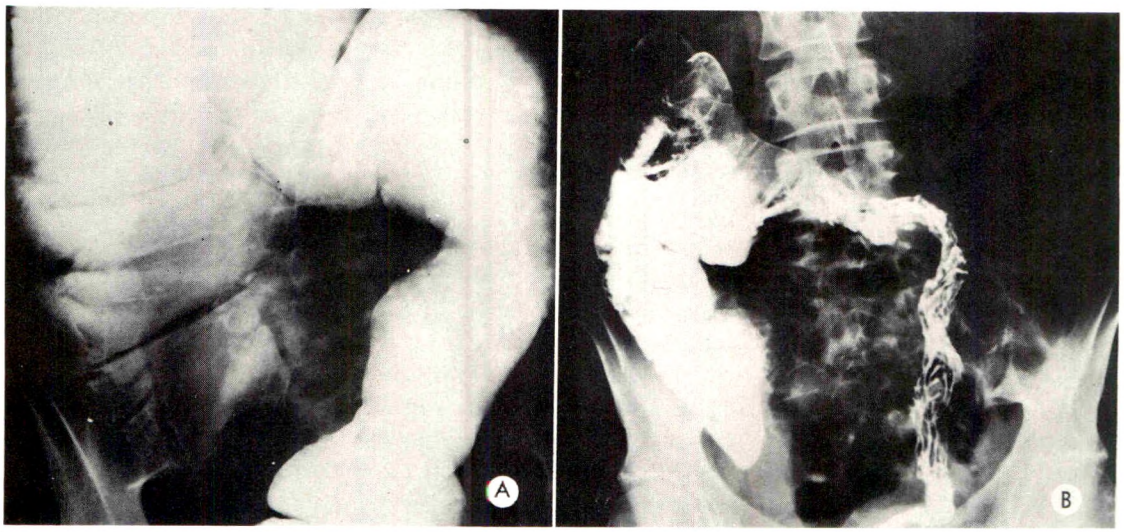


FIG. 2. Enema examination performed with barium and Au^{198} colloid mixture. (*A*) The front of the barium mixture is in the region of the ascending colon. At this point no activity was noted in the urine. (*B*) The postevacuation roentgenogram shows some barium flowing retrograde into the region of the cecum. Activity was noted in the urine at this time.

the area of the cecum (Table I). No fistulous tract was outlined by the barium. Thus from these 2 studies it appeared that the fistula was located in the area of cecum.

Postoperative Study. The results of this study are shown in Table II. As can be seen, activity appeared in the feces at 1 hour and 40 minutes after administration. At this time there was no activity in the urine. In subsequent samples, the urine remained free of activity.

DISCUSSION

The use of a substance, such as charcoal, that can be given by mouth, is not absorbed from the gastrointestinal tract, and is of small enough size to pass through the fistula into the genitourinary system is useful to confirm the presence of an intestinovesical fistula. The choice of a radioactive substance that has the above properties can be an advantage since exceedingly small amounts of the material can be measured in the urine quite easily, and in addition its approximate position in the gastrointestinal tract can be ascertained by external imaging. In this patient less than 1/10,000 of the administered dose was found in the urine on the first examination.

The tracer selected for fistula detection has to fulfill the following criteria: (1) not be absorbed from the gastrointestinal

TABLE I

LOCATION OF BARIUM-AU^{198} COLLOID MIXTURE IN COLON AND ASSOCIATED ACTIVITY IN URINE

Location	Counts (1 min.)/ 2 cc. Urine
No Barium	BKg*
Sigmoid colon	BKg
Descending colon	BKg
Splenic flexure	BKg
Transverse colon	BKg
Hepatic flexure	BKg
Ascending colon	BKg
Cecum	1,200

* Mean BKg was 20 cpm.

TABLE II

ACTIVITY IN FECES AND URINE AFTER ADMINISTRATION OF AU^{198} COLLOID BY MOUTH

Time	Feces	Urine
45 min.	BKg*	BKg
100 min.	15,000 cts/gm.	BKg
150 min.	40,000 cts/gm.	BKg
360 min.	—	BKg

* Mean BKg was 20 cpm.

tract; (2) have small size to easily pass through the fistula; (3) be detected easily in the urine; and (4) be easily imaged while in the gastrointestinal tract.

These criteria limit the selection of a material to a gamma emitting radionuclide, which must be insoluble and nonabsorbable. Technetium labeled compounds while having good counting characteristics cannot be used, since it is difficult to make a completely stable bond between the tracer and the compound and any of the label eluted from the carrier compound would be absorbed by the gastrointestinal tract and partly excreted in the urine.

Other nuclides were considered including: chromium 51; carbonized microspheres; and gold colloid. Chromium was not selected because it has only an 8 per cent incidence gamma photons/100 disintegrations. This is too few to obtain a high resolution image in a reasonable interval of time. An advantage of carbonized microspheres is that they can be firmly labeled with nuclides suitable for imaging with the gamma camera and that a different labeled microsphere could be used for further studies on the same day without having to wait for the other nuclide to decay. However, the smallest particle size available commercially is approximately 5 μ. This size might inhibit the passage of the particle through some small fistula.

The least expensive, off the shelf, radiopharmaceutical which meets the above criteria is Au^{198} colloid. The tracer (95 per cent 410 kev.) is a gamma emitter which is not absorbed from the gastrointestinal tract, the colloidal particles are extremely small (<0.1 μ), readily detected in the urine by counting the specimen in a gamma well counter, and can easily be imaged with a gamma camera.

As illustrated in this study, when the radioactive material was combined with barium, the anatomic resolution increased. This combination of Au^{198} colloid and barium also could probably be given by mouth to determine small intestine fistula tracts. The evaluation of the site of fistula by both the oral and rectal routes permits a degree of confidence in the diagnosis that might not be present were either method used alone.

Postoperatively the examination was repeated without a Foley catheter. With a cooperative patient rinsing of the genital region is adequate for good collections. However, when the patient was having a barium enema it was not possible for the patient to move and in this instance a urinary catheter was necessary. Also when the barium enema examination is performed, there is not much time available for collection of the urine samples as the barium moves through the colon. In this case the Foley catheter and good hydration are necessary.

One consideration in performing this examination is the radiation dose to the gastrointestinal tract from the gold colloid. In this patient, with a rapid transit time, the radiation burden for the gonads is 42 mrads, approximately 1/100 of the gonadal dose from an intravenous pyelography. For patients with normal gastrointestinal transits, the calculated radiation burden is increased, but remains well within safe limits. (See Appendix.)

SUMMARY

Detection and localization of an enterovesicular fistula in a patient with chronic inflammatory bowel disease and recurrent urinary tract infection was accomplished by following a bolus of orally administered gold colloid as it traversed the gastrointestinal tract with serial scintiphotos and simultaneously monitoring the activity in the urine.

When significant tracer was found in the urine, multiple scans of the abdomen were obtained to determine the location of the tracer.

To confirm the site of the fistula, the tracer was mixed with barium and administered per rectum. Radiographs were obtained when significant activity was found in the urine.

At surgery, bowel in the region of the

cecum was adherent to the bladder, but no fistula was demonstrable by methylene blue instillation into the bladder.

Postoperatively, the urinary tract infection cleared, and re-examination with oral Au^{198} colloid revealed no activity in the urine.

The radiation dose to the gonads from this procedure is 42 mrads.

The procedure is safe, simple to perform, well tolerated, and permits an answer to the question of whether a fistula is present, and where it is located.

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REFERENCES

1. Bourne, R. B. New aid in diagnosis of vesicoenteric fistula. *J. Urol.*, 1964, *91*, 340–342.
2. Coures, G. D., and Block, M. A. Intestinovesical fistula. *Surgery*, 1963, *54*, 736–742.
3. Forbes, K. A., and Rose, R. J. Appendicovesical fistula. *Ann. Surg.*, 1964, *160*, 801–805.
4. Goodwin, W., Winter, C. C., and Turner, R. D. Fistula between bowel and urinary tract. *J. Urol.*, 1960, *84*, 95–105.
5. Pennington, J., and Plumb, R. T. Diagnosis and treatment of inflammatory vesicoenteric fistula. *J. Urol.*, 1964, *92*, 679–680.
6. Ward, J. N., Lowengood, K. W., Nay, H. R., and Draper, J. W. Diagnosis and treatment of colovesical fistulas. *Surg., Gynec. & Obst.*, 1970, *130*, 1082–1090.

APPENDIX

GASTROINTESTINAL TRACT DOSIMETRY FOR Au^{198} AND Tc^{99m}

Because of the extremely rapid transit in the gastrointestinal tract of this unusual subject, the ordinary kinetic models described by Bernard and Hayes,[1] Unnikrishan and his colleagues,[5] and Dolphin and Eve[2] for the behavior of insoluble material in the tract do not apply. In the case at hand, the total transit time through the whole tract was determined to be 2 hours. The activity in the bolus (100 μc) was sufficient to permit its shape and length to be revealed by scanning. In the descending loop of the bowel, the bolus was approximately 1 foot long and 2 inches in diameter, which is equivalent to a volume of 618 $cm.^3$ and, assuming unit density, to a mass of 618 grams.

A large portion of the radiation dose to the lumen of the bowel will be that delivered by the very short range (beta and beta-like), "nonpenetrating" radiations. At a maximum, the lumen dose could approach one-half of the self dose to the bolus from these radiations. For Au^{198}, the nonpenetrating emissions deliver a dose of 0.7028 gm.-rads/μc-hr. to the volume in which the radionuclide is distributed as given in MIRD Pamphlet 4.[3] Accordingly, the dose to the lumen from the activity in a 618 gm. bolus could approach:

$$1/2\left(\frac{100\ \mu c}{618\ gm.}\right).7028 = .05686\ rads$$

per hour of residence.

In the unlikely case of complete stasis, the residence time, as a limit, would be equal to the mean life of Au^{198}, which is in hours:

$$1.44(2.70\ days)(24) = 93.31\ hours,$$

and, in that event, the dose from nonpenetrating radiations would reach the limit:

$$.05685\ rads/hr.\ (93.31\ hr.) = 5.306\ rads.$$

Given the measured 2 hour total transit time in this patient, the length of the bolus revealed on the scan and estimating the total length of the gastrointestinal tract to be 24 feet, the bolus would require 2/24 hr. (.0833 hr.) to pass a given point in the bowel. The dose from nonpenetrating radiations at any such point would thus be:

$$.05686\ rads/hr.\ (.0833\ hr.) = .004736\ rads.$$

If this same bolus were to have traveled at the same rate through the narrower small intestine, it would have been elongated by a factor of 4 and its passage would take 4 times as long. The nonpenetrating

dose to the immediately adjacent portion of the small intestine would thus be:

.05686 rads/hr. (8/24 hr.) = .01895 rads.

The dose from penetrating photon radiations can be conservatively estimated by assuming that the entire 100 μc bolus remained within the target organ for the entire 2 hours. Making this assumption separately for the small intestine and large intestine one obtains:

.02392 rads to small intestine, and

.07372 rads to large intestine (lower portion).

A similarly conservative estimate of the gonadal dose in this patient can be made by calculating the penetrating photon close on the pessimistic assumption that the bolus remains for the entire 2 hours in that portion of the gastrointestinal tract from which it most effectively irradiates the ovaries. That assumption yields an estimate of:

.04166 rads to the gonads.

The dose estimates for this patient are summarized in Table I (Appendix).

Of more general interest is the estimated radiation dose from this procedure in subjects with normal gastrointestinal physiology. The data of Dolphin and Eve[2] can be used to estimate the location and mean residence time of insoluble material in the gastrointestinal tract at various times after ingestion.

The beta and beta-like dose to a segment of the lumen of the gut depends directly upon the specific activity of the bolus and the time required for that bolus to pass a particular point on the lumen. Given a designated total activity, the specific activity of a bolus depends inversely upon its size or mass. The mass of the bolus varies according to the net in-flow or out-flow of fluid in the affected portion of the gut, which is not susceptible to accurate prediction. However, if one assumes regular laminar flow (not an unreasonable assumption in the case of an insoluble nonabsorbable substance), then any dilution of the bolus which reduces its specific activity will necessarily prolong its passage by an exactly equivalent amount, to the end that the cumulative exposure (in μc-hr.) remains constant. Given a mean residence time in a segment of the gastrointestinal tract, the mean cumulative exposure to any portion of that segment can be calculated by assuming that the activity is distributed in a mass equal to the volume of that segment for a period equal to the mean residence time. For dosimetric purposes, the gastrointestinal tract is divided into 4 segments, the stomach, the small intestine, the upper large intestine, and the lower large intestine. Using the transit times from Dolphin and Eve,[2] and the volumes from MIRD Pamphlet 5,[4] the cumulative activity (corrected for physical decay) has been calculated to estimate the beta and beta-like dose to the lumen of the several segments in subjects with normal transit times. The results of these calculations are summarized in Table II (Appendix).

Penetrating χ and γ photons, unlike beta and beta-like particles can irradiate not only the segment in which they are then contained, but also other segments of the gastrointestinal tract and the gonads. The dose to the gonads from penetrating radiations emitted from various portions of the tract has been estimated from the MIRD data[4] for the absorbed fraction of such

TABLE I

(Appendix)

RADIATION DOSE TO SUBJECT X FROM THE PROCEDURE DESCRIBED

Organ	Small Intestine	Large Intestine	Gonads
β dose, rads	.01895	.00474	0
γ dose, rads	.02392	.07372	.04166
Total dose, rads	.04287	.07846	.04166

TABLE II
(Appendix)

RADIATION DOSE TO GONADS AND GASTROINTESTINAL TRACT FROM 100 μc OF INSOLUBLE Au^{198} COLLOID

Time (hr.)	Location of Activity	Gamma Doses from Activity in Gastrointestinal Tract				
		Stomach	Small Intestine	Upper Large Intestine	Lower Large Intestine	Gonads (Uterus)
0–1	stomach	.02187	.00083	.00114	.00038	.00191
1–5	small intestine	.00303	.02968	.01581	.00597	.08010
5–13	upper large intestine	.00754	.02647	.10845	.00631	.06682
13–31	lower large intestine	.00574	.02625	.01264	.29923	.20529
Total gamma dose (rads)		.03818	.08323	.13804	.31198	.35412
β self-dose (rads)		.0897	.0793	.5514	1.9023	0
Total dose (rads)		.1279	.1625	.6894	2.2143	.3541

emanations in the uterus, which organ may be taken to be representative of the similarly situated (but untabulated) ovaries. In addition, photons emitted from a bolus resident in one portion of the tract irradiate other portions as well and this dose must be added to the self-dose accumulated when the bolus resides in the affected portion. Since photons are penetrating the resulting dose is relatively independent of assumptions concerning the precise location of the activity within specific portions of the tract. Accordingly, the self-dose and segment to segment dose have been estimated, assuming uniform distribution in each segment for the mean residence times given by Dolphin and Eve.[2] Absorbed fractions were taken from the previously cited MIRD Pamphlet 5.[4] Results of these calculations are also summarized in Table II (Appendix).

For comparison the dosimetry of an insoluble form of technetium 99m is included. This isotope delivers only about one-tenth the dose produced by an equivalent activity of Au^{198}. Dose estimates for the ingestion of 1,000 μc of Tc^{99m} are summarized in Table III (Appendix).

TABLE III
(Appendix)

RADIATION DOSE TO GONADS AND GASTROINTESTINAL TRACT FROM 1,000 μc OF INSOLUBLE Tc^{99m}

Time (hr.)	Location of Activity	Gamma Doses				
		Stomach	Small Intestine	Upper Large Intestine	Lower Large Intestine	Gonads (Uterus)
0–1	stomach	.06764	.00263	.00356	.00118	.00506
1–5	small intestine	.00665	.06364	.03409	.01470	.18837
5–13	upper large intestine	.00191	.04617	.18207	.00937	.12158
13–31	lower large intestine	.00199	.01013	.00487	.11118	.09102
Total gamma dose (rads)		.07819	.12257	.22418	.13643	.40603
β self-dose (rads)		.0424	.0263	.1443	.1100	0
Total dose (rads)		.1206	.1489	.3685	.2464	.40603

REFERENCES

1. Bernard, S. R., and Hayes, R. L. Dose to various segments of gastrointestinal tract. In: Medical Radionuclides: Radiation Dose and Effects. AEC Symposium Series No. 20, 1970.
2. Dolphin, G. W., and Eve, I. S. Dosimetry of gastrointestinal tract. *Health Physics*, 1966, *12*, 163.
3. MIRD. Suppl. No. 2. Pamphlet 4. *J. Nuclear Med.*, 1969, *10*.
4. MIRD. Suppl. No. 3. Pamphlet 5. *J. Nuclear Med.*, 1969, *10*.
5. Unnikrishan, K., Hupkoo, R. K., and Somasundaram, S. Dosimetry of unabsorbed beta emitters. *Health Physics*, 1973, *25*, 141–146.

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Vol. 121, No. 4

INFERIOR VENA CAVAL EXTENSION OF RENAL CARCINOMA: A LOST CAUSE?*

ROENTGENOGRAPHIC AND PATHOLOGIC FINDINGS IN SURGICAL PATIENTS

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AT one time thrombosis of the inferior vena cava due to extension of a renal carcinoma was believed to be an ominous sign and uniformly associated with an extremely poor prognosis.[11] Our recent experience with 4 patients demonstrating inferior vena caval extension of right renal carcinoma reveals that resection of the renal tumor and vena cava with ligation of the left renal vein is possible, but the ultimate prognosis depends on several factors.

Angiography has become increasingly important in determining the extent of intravascular tumor and its usefulness in planning surgical therapy will be illustrated.

REPORT OF CASES

CASE I. R.E.D. A 58 year old man presented with back pain and pitting edema of the lower extremities. A metastatic work-up was negative. The roentgenographic findings are illustrated in Figure 1, *A–C*. An *en bloc* right radical nephrectomy with vena cavectomy and ligation of the left renal vein was performed. Pathologic examination revealed positive renal hilar lymph nodes, caval invasion, and perinephric fat involvement. He is alive and working over a year later but has several nodular lesions on a recent chest roentgenogram.

CASE II. M.M. A 63 year old man was admitted with gross hematuria and was found to have edema of the left leg and a right upper quadrant fullness. Figure 2, *A* and *B*, demonstrates the roentgenographic findings. Osteolytic lesions were demonstrated in the right humerus and in one rib on metastatic bone survey. A palliative operation was performed using a technique similar to Case I. Pathologic examination of the specimen revealed involvement of the diaphragm, vena cava, and lymph nodes. He died 5 months later.

CASE III. R.G. A 66 year old woman presented with fever, fatigue, night sweats, and anemia. Liver function tests were abnormal. The metastatic work-up was negative. Figure 3, *A–E*, demonstrates the roentgenographic findings. An operative procedure was performed (Fig. 5*C*). Pathologic examination of the specimen revealed positive aortic lymph nodes with involvement of the right adrenal gland, vena cava, and perinephric fat. She left the hospital after 10 days but died 6 months later of cerebral metastases.

CASE IV. M.V. A 70 year old man had several episodes of gross hematuria. A metastatic work-up was negative. The roentgenographic findings are demonstrated in Figure 4, *A–D*. An operation similar to the one described in Case I was performed. Although the vena cava was invaded, all resected lymph nodes were negative and there was no invasion of the renal capsule. Azotemia developed postoperatively, but resolved by the ninth postoperative day when he was discharged. At follow-up examination 9 months later, there was no evidence of recurrent tumor. Serum creatinine was 1.9 mg. per cent and the left kidney was normal on an excretory urogram.

DISCUSSION

Tumor thrombus extension into the vena cava is found in less than 10 per cent of renal cell carcinoma nephrectomy specimens.[8,12] Approximately 25 per cent of the cases with caval involvement also exhibit right atrial extension,[11,13] and instances of tumor thrombus extending to the right ventricle and even into the pulmonary

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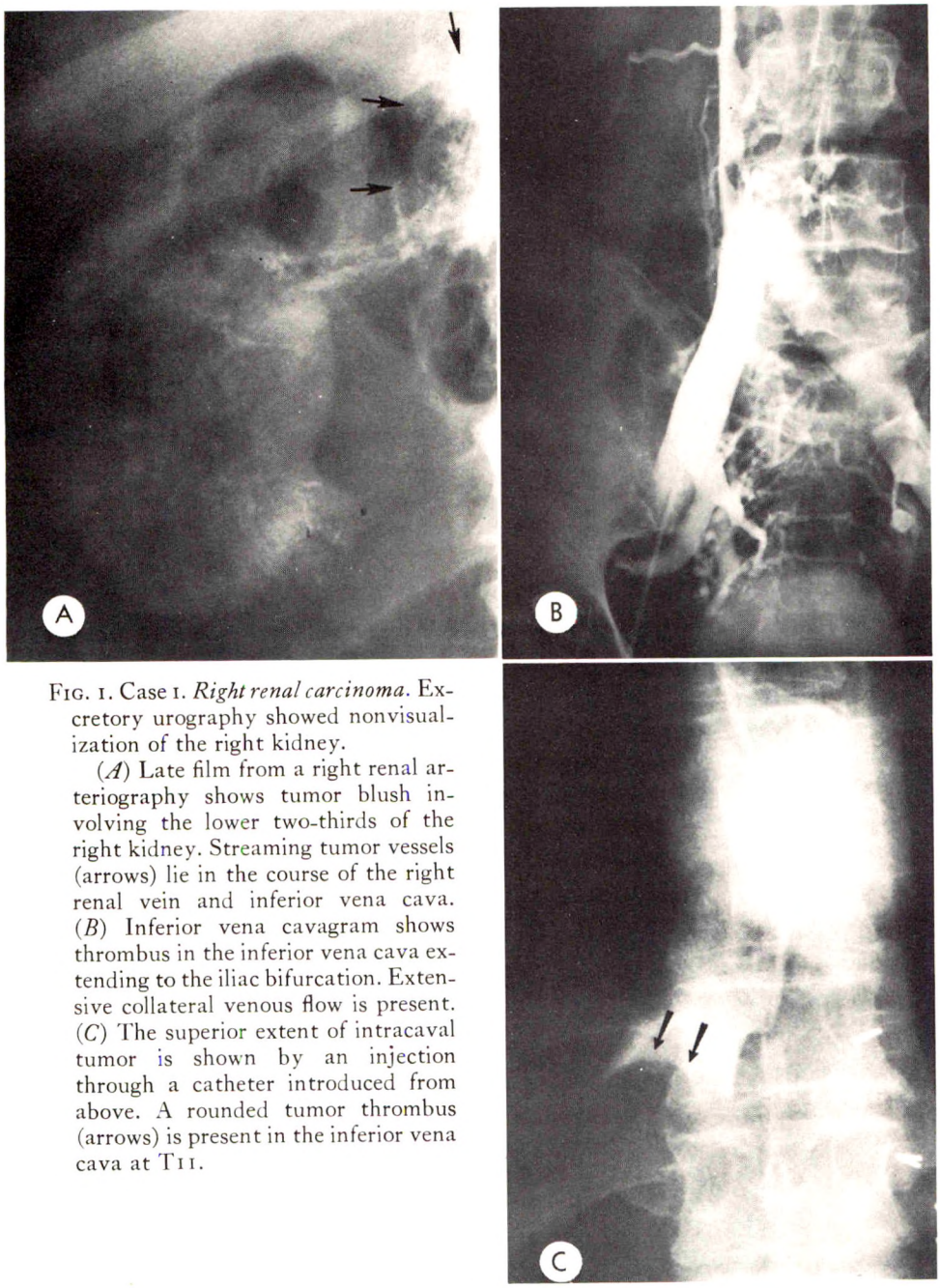


FIG. 1. Case 1. *Right renal carcinoma.* Excretory urography showed nonvisualization of the right kidney.

(*A*) Late film from a right renal arteriography shows tumor blush involving the lower two-thirds of the right kidney. Streaming tumor vessels (arrows) lie in the course of the right renal vein and inferior vena cava. (*B*) Inferior vena cavagram shows thrombus in the inferior vena cava extending to the iliac bifurcation. Extensive collateral venous flow is present. (*C*) The superior extent of intracaval tumor is shown by an injection through a catheter introduced from above. A rounded tumor thrombus (arrows) is present in the inferior vena cava at T11.

artery have been documented.[1] Right renal carcinomas are much more likely to grow into the vena cava than tumors originating on the left due to the short right renal vein. Males with caval extension have outnumbered females by approximately 4 to 1.[11,13]

It is apparent from our case histories and others previously reported that radical surgery including vena caval resection may be justified in some of these patients. Precise preoperative localization of the tumor thrombus permits proper operative planning.

Several roentgenographic signs have correlated well with extension of tumor into

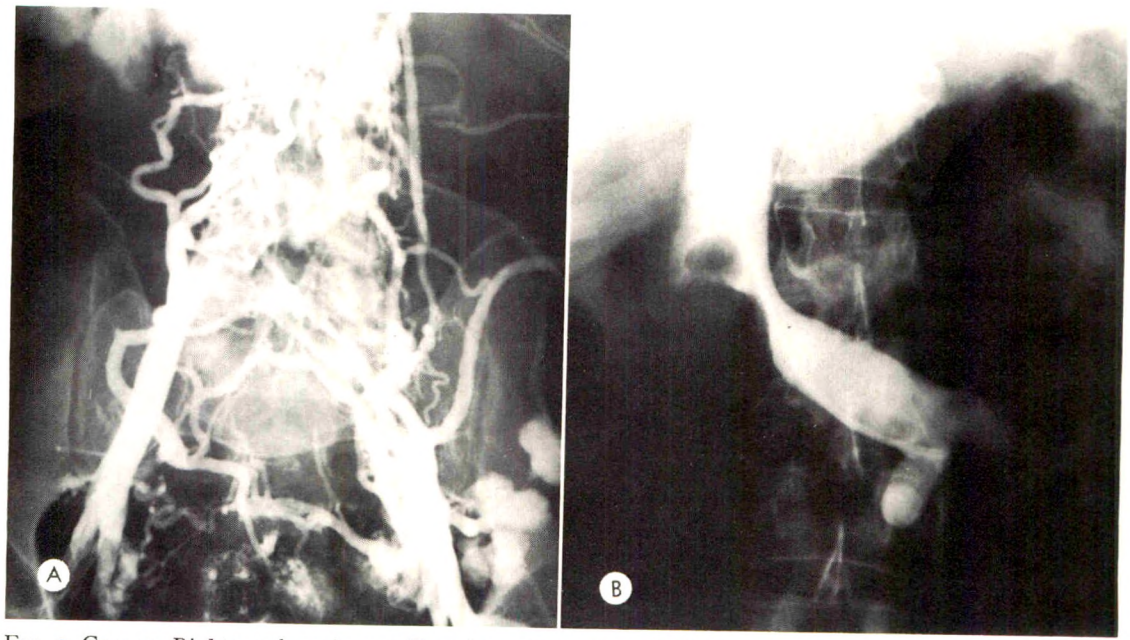


Fig. 2. Case 11. *Right renal carcinoma.* Complete obstruction of inferior vena cava.
(*A*) Bilateral iliac venogram shows caudal extension of thrombus to the right common iliac vein and to the left external iliac vein. An extensive network of collateral venous channels is present. (*B*) Left renal venogram with catheter introduced from above shows intracaval tumor extending slightly above the level of the left renal vein. The orifice of the left renal vein is partially occluded.

the renal vein and inferior vena cava. Lalli[7] indicated that urographic nonvisualization of the kidney containing tumor was an almost certain sign of renal vein invasion. It has been apparent from other studies, however, that patients have had both renal vein and inferior vena caval extension of renal carcinoma, while maintaining excretion of contrast medium on the urogram to a greater or lesser degree.[4] Kahn[6] and Ferris *et al.*[4] showed that a striated vascular pattern on renal arteriography indicates the presence of tumor in the renal vein and/or vena cava. This pattern reflects the supply of intraluminal tumor by vessels originating in the kidney and may be exaggerated by pharmacoangiography with epinephrine. While the presence of this sign always indicates intravascular tumor, its absence, unfortunately, does not exclude venous involvement.[4,9]

In 2 of our patients there was bland thrombosis of the inferior vena cava below the tumor thrombus. In both instances striated vessels were present in the tumor thrombus, but none were visualized in the bland thrombus. Bland thrombosis of the inferior vena cava below the level of the kidney is probably related to partial obstruction.

Angiography may be misleading when collateral venous drainage appears in a kidney with carcinoma. While some authors originally took this finding to indicate renal vein extension of tumor,[3] it has been correctly pointed out by others[2,5] that such collateral venous drainage is seen frequently in the absence of renal vein involvement, and merely reflects parasitization of capsular and retroperitoneal veins by the tumor.

The only means of diagnosing or excluding tumor extension into the renal veins and inferior vena cava is to visualize these structures directly. If the renal vein can be cleared of tumor involvement by means of large dose arteriography (20–40 cc. into the renal artery), a vena cavagraphy need not be performed. If the renal vein is not demonstrated, and this is often the case

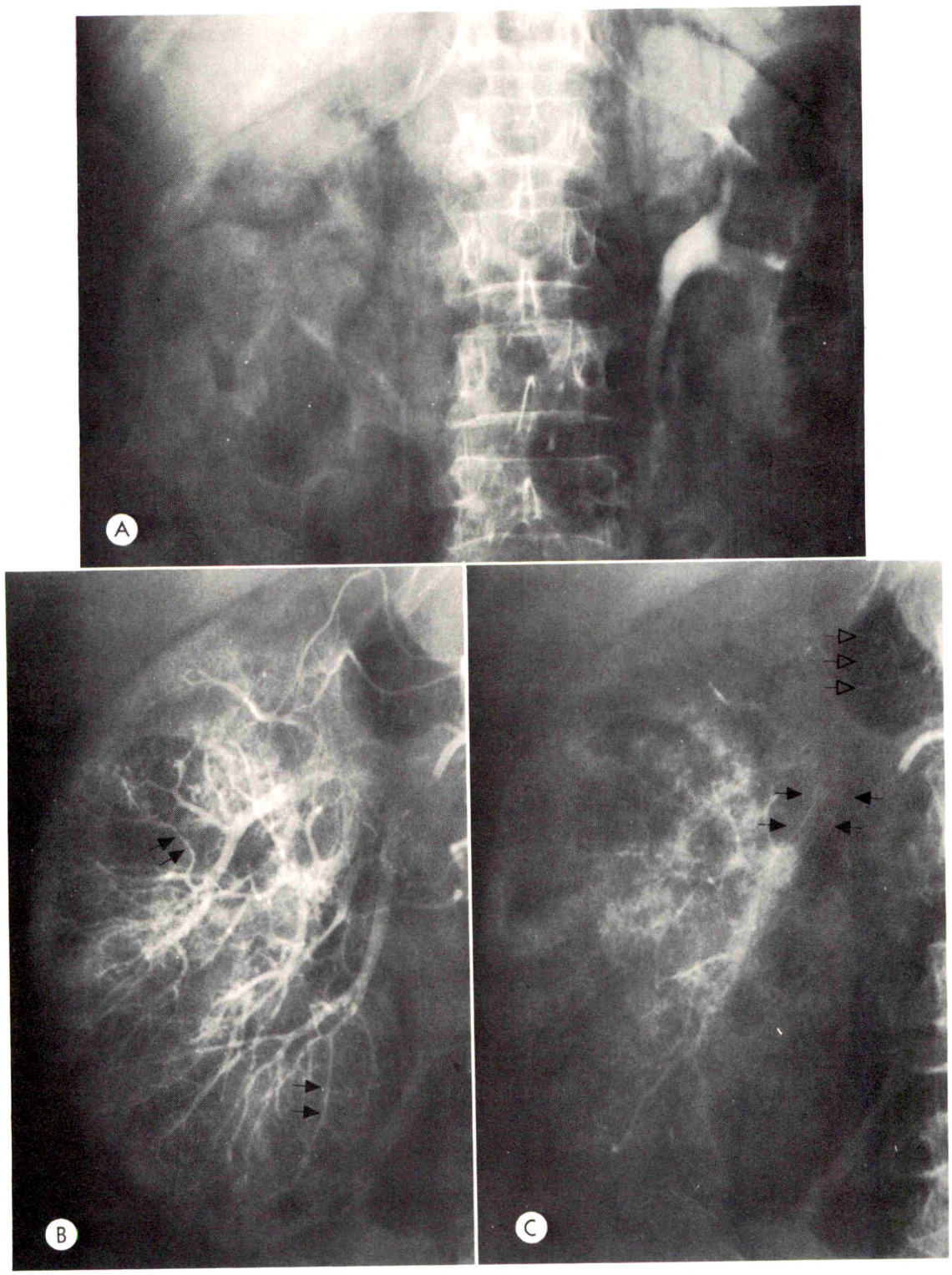


FIG. 3. Case III. *Right renal carcinoma.*

(*A*) Excretory urogram shows an enlarged right kidney with markedly impaired excretion of contrast material. (*B*) Arteriography demonstrates fine tumor vessels throughout the right kidney and several encased intrarenal arteries (arrows). (*C*) Later film from the right renal arteriography shows "streaming" vessels (black arrows) in the course of the right renal vein. A fine network of tumor vessels (open arrows) corresponds to tumor in the inferior vena cava.

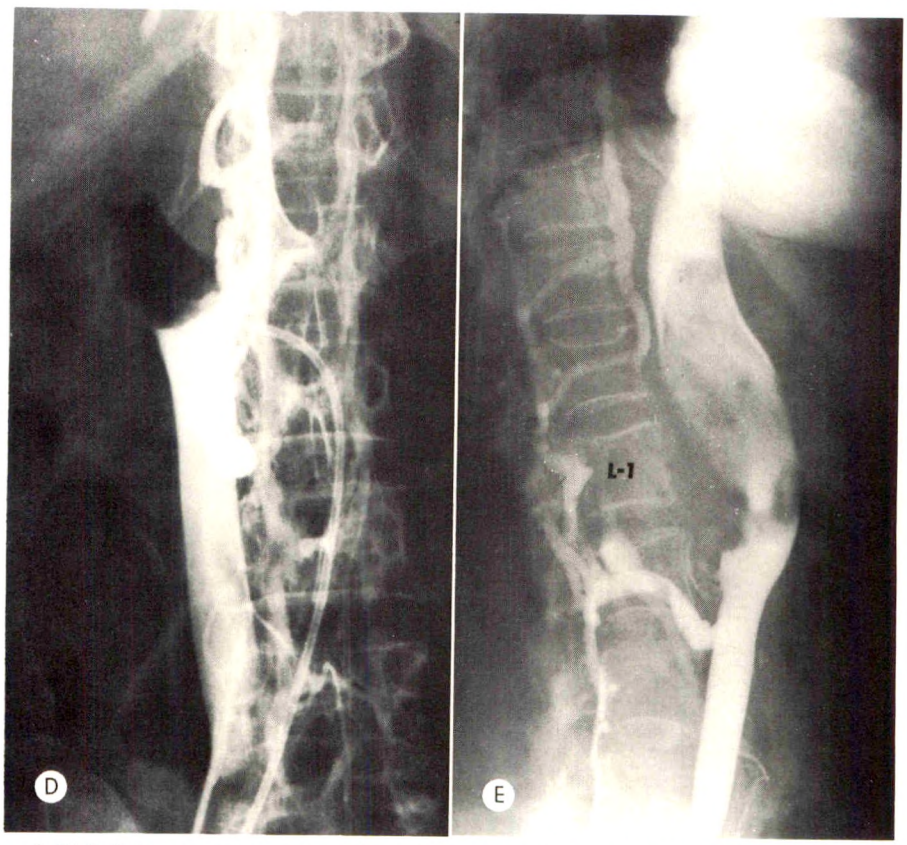


Fig. 3. (*D* and *E*) Inferior vena cavagrams, frontal and lateral views. There is high grade, incomplete obstruction of the inferior vena cava with collateral flow through the lumbar-paravertebral plexus. A large filling defect in the inferior vena cava corresponds to the tumor vessels demonstrated on the arteriogram. The superior extent of intracaval tumor is at T11.

on the right side, a vena cavagraphy should be done to answer the question. In the presence of complete vena caval obstruction, it is necessary to visualize the superior extent of involvement by catheterizing the patient from above, either *via* an antecubital or jugular vein.

If the tumor thrombus extends above the level of the main hepatic veins, the surgeon should be prepared to use cardiopulmonary bypass. When the tumors project only slightly into the vena cava, vena cavotomy with *en bloc* extraction of the tumor may be all that is required. However, if the tumor thrombus is invading or densely adherent to the vena cava (as in all our cases) vena caval resection seems desirable. Figure 5, *A–C*, summarizes the surgical alternatives which have recently been considered in more detail in a separate report.[10]

The left kidney will survive ligation of the left renal vein if this is done close to the vena cava, because the proximal connections of the renal vein (adrenal, gonadal, lumbar, ureteric) provide sufficient collateral drainage. Although temporary renal venous hypertension with renal dysfunction may develop, these resolve as collateral venous drainage improves. Roentgenographic findings in the patient who suffered temporary renal dysfunction were an increase in renal size and compression of the collecting system; both resolved in 9 days as renal function returned to normal. Physiologic changes included temporary proteinuria, hypertension with elevated renin levels, oliguria,

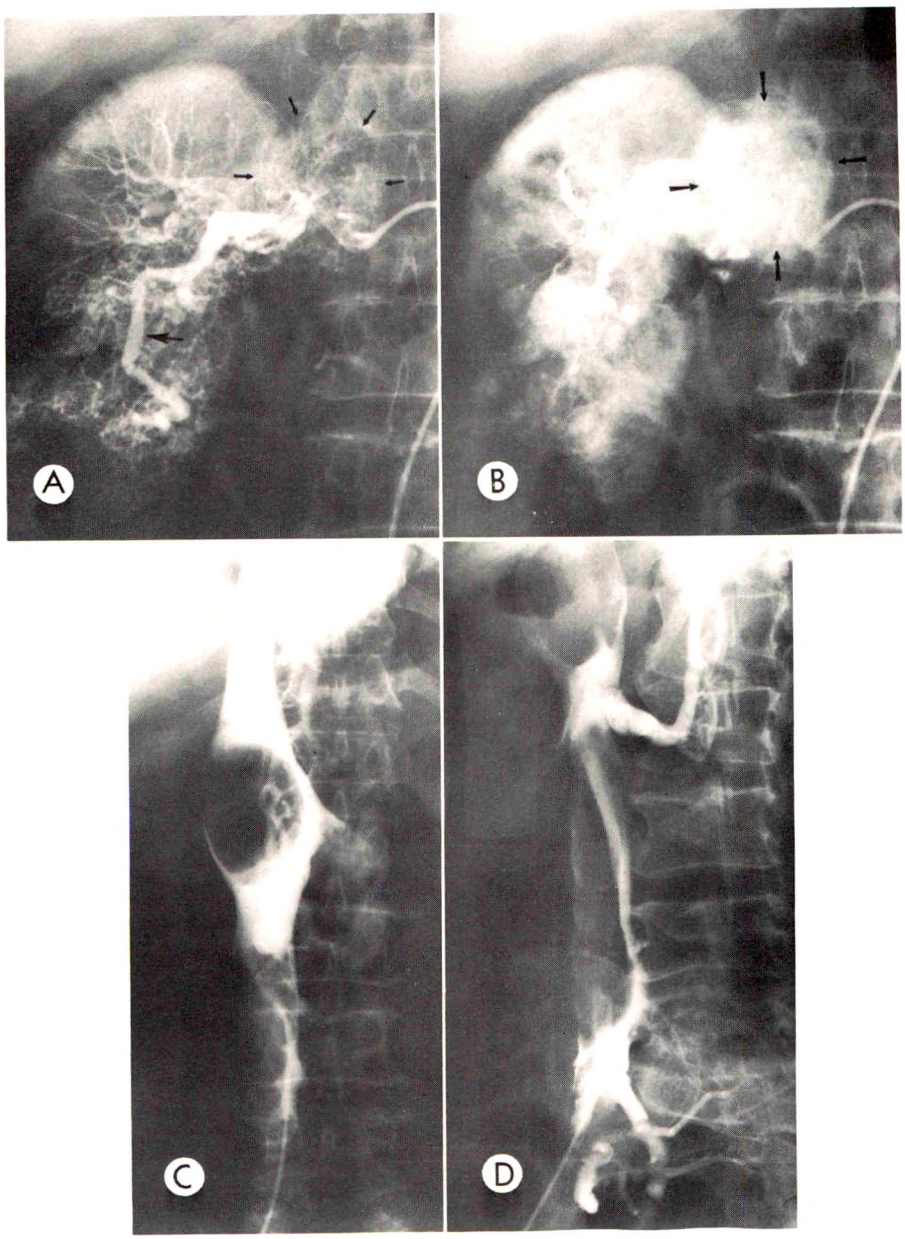


FIG. 4. Case IV. *Right renal carcinoma.* Excretory urography showed diminished excretion of contrast material by the right kidney.

(*A*) Right renal arteriogram, early phase, shows profuse tumor vascularity involving the lower two-thirds of the right kidney. A prominent collateral vein (large arrow) courses towards the renal hilum. A circular collection of fine tumor vessels (small arrows) lies medial to the upper pole of the kidney. (*B*) Right renal arteriogram, late phase. A dense tumor blush (arrows) is partially superimposed on the spine. A collateral vein draining the kidney is still filled with contrast material. (*C*) Inferior vena cavagram, frontal projection, shows an oval tumor thrombus at the level of the right renal vein. The intracaval tumor corresponds exactly to the tumor blush shown on the arteriogram. (*D*) Inferior vena cavagram, right posterior oblique projection. A large thrombus occupies the inferior vena cava below the level of the tumor thrombus seen in *C*. At surgery the lower thrombus contained no tumor. Filling of collateral lumbar paravertebral veins indicates partial obstruction of the inferior vena cava by the tumor thrombus.

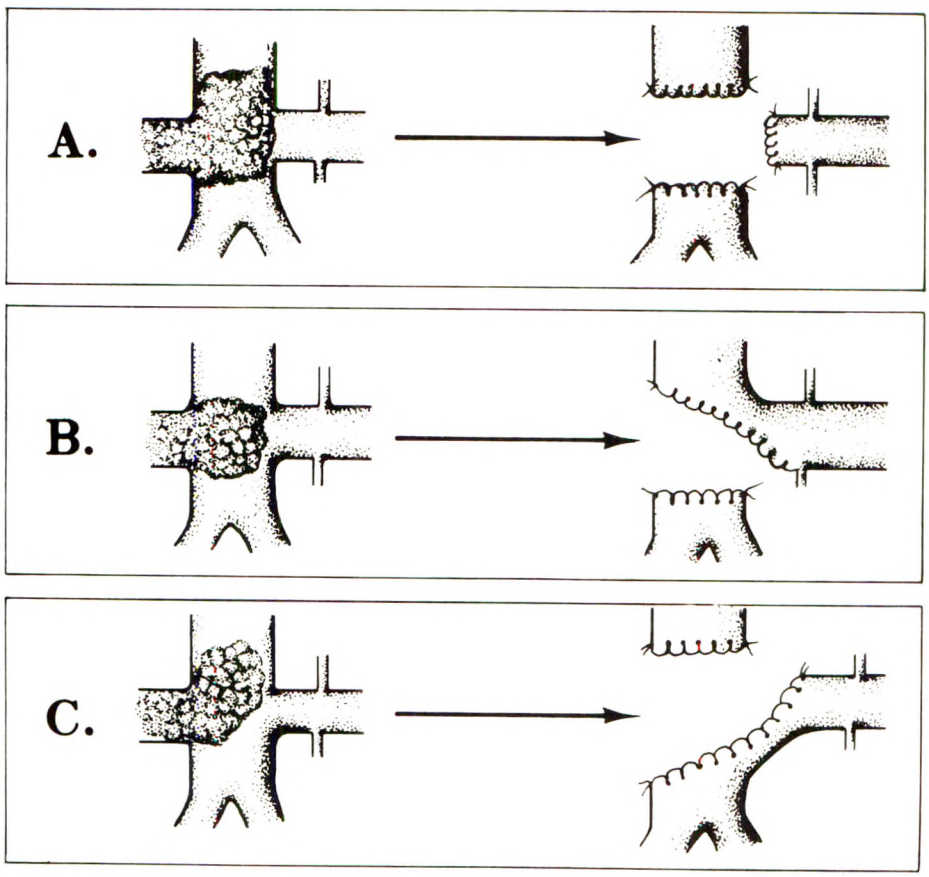


FIG. 5. (*A–C*) Different methods of surgical management of vena cava invasion by renal carcinoma.

and azotemia.

All 4 cases demonstrated tumor invasion of the right renal vein and the inferior vena cava for a variable distance. There was often endothelialization of the tumor thrombi. None of these tumors could have been "shelled out" of the inferior vena cava without leaving tumor cells behind. Figure 6, *A* and *B*, demonstrates invasion of the wall of the renal vein and vena cava by renal cell carcinoma, and is representative of all our patients.

Prognosis apparently depends on the stage of the disease one finds at surgery; *i.e.*, lymph node involvement, extension into perirenal fat, liver, etc. It is often impossible to determine the pathologic stage until the specimen is removed, even with the aid of preoperative panangiography. The role of palliative nephrectomy for renal cell carcinoma is controversial.

The morbidity in these patients was low. All left the hospital in less than 14 days and there was no problem with prolonged edema of the lower extremities.

SUMMARY

Using angiography it is possible to accurately define venous extension of renal carcinomas preoperatively.

Modern surgical techniques permit successful removal of the tumors and vena cava *en bloc*. The left renal vein can be ligated, although a syndrome of temporary renal dysfunction may occur.

The long term prognosis of such patients appears to correlate best with the pathologic stage of the resected specimen and

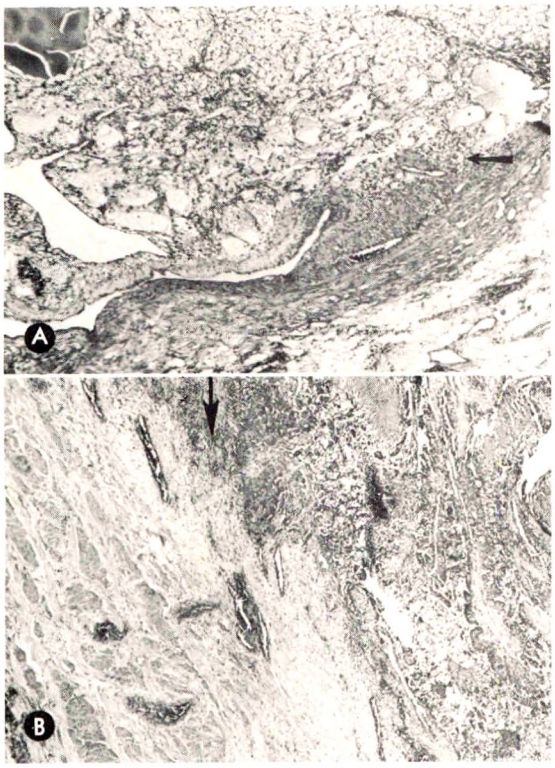


FIG. 6. *Renal cell carcinoma.* Venous invasion.
(*A*) Renal vein—arrow points to site of invasion, tumor is above, vein wall below. (*B*) Vena cava—arrow at site of invasion, tumor is at right, vena caval wall to left.

cannot be predicted by preoperative studies unless these show evidence of metastases.

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REFERENCES

1. Arkless, R. Renal carcinoma: how it metastasizes. *Radiology*, 1965, *84*, 496–501.
2. Brindle, M. J. Alternative vascular channels in renal cell carcinoma. *Clin. Radiol.*, 1972, *23*, 321–330.
3. Eisenman, J. I., Finck, E. J., and O'Loughlin, B. J. Collateral vein sign: angiographic demonstration of renal vein invasion in renal carcinoma. *Radiology*, 1969, *92*, 1256–1261.
4. Ferris, E. J., Bosniak, M. A., and O'Connor, J. F. Angiographic sign demonstrating extension of renal carcinoma into renal vein and vena cava. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 384–391.
5. Folin, J. Angiography in renal tumors. *Acta radiol.*, 1967, Suppl. 267.
6. Kahn, P. C. Epinephrine effect in selective renal angiography. *Radiology*, 1965, *85*, 301–305.
7. Lalli, A. F. Roentgen diagnosis of renal vein invasion by tumor. *J. Urol.*, 1966, *95*, 8–9.
8. Marshall, V. F., Middleton, R. G., Holswade, G. R., and Goldsmith, E. I. Surgery for renal cell carcinoma in vena cava. *J. Urol.*, 1970, *103*, 414–420.
9. McCoy, R. M., Klatte, E. C., and Rhamy, R. K. Use of inferior vena cavography in evaluation of renal neoplasms. *J. Urol.*, 1969, *102*, 556–559.
10. McCullough, D. L., and Gittes, R. F. Vena cava resection for renal cell carcinoma. To be published.
11. Ney, C. Thrombosis of inferior vena cava associated with malignant renal tumors. *J. Urol.*, 1946, *55*, 583–590.
12. Skinner, D. G., Pfister, R. F., and Colvin, R. Extension of renal cell carcinoma into vena cava: rationale for aggressive surgical management. *J. Urol.*, 1972, *107*, 711–716.
13. Svane, S. Tumor thrombus of inferior vena cava resulting from renal carcinoma. *Scandinav. J. Urol. & Nephrol.*, 1969, *3*, 245–256.

CALCIFICATION IN UROEPITHELIAL TUMORS OF THE BLADDER*

REPORT OF 5 CASES AND SURVEY OF THE LITERATURE

By STEPHEN W. MILLER, M.D., *and* RICHARD C. PFISTER, M.D.
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ALTHOUGH calcification within bladder tumors is an uncommon roentgenographic finding, it is important to distinguish tumor calcification from other causes of bladder and pelvic calcifications. Individual case reports indicate that the usual roentgen appearance consists of a hazy group of fine, punctate calcifications,[2,4,7] less frequently as a coarse nodularity,[6] or a mixture thereof. The incidence of roentgenologically demonstrable calcium in uroepithelial bladder tumors has varied widely in the 2 reported series: between 0.69 and 6.7 per cent.[3,6]

This report deals with the incidence, pathology, and roentgenologic features of 5 cases of calcified epithelial bladder cancers encountered at the Massachusetts General Hospital and summarizes the cases reported in the English language literature since 1961.[3,4,6]

MATERIAL

The pertinent features of the 5 cases of calcified uroepithelial tumors of the bladder are summarized in Table 1. Three of the neoplasms were transitional cell, 1 was squamous cell and the fifth a mixture of transitional-squamous cell carcinoma. In 3 patients calcium was located on the surface of the neoplasm, being intermingled with the papillary fronds in 1 of these. The other 2 cases had calcium deposited in the subepithelial region adjacent to the

TABLE I

CALCIFIED EPITHELIAL TUMORS OF THE BLADDER

Case No.	Age	Sex	Pathologic Diagnosis	Histologic Location of Calcium	Roentgenologic Appearance of Calcium	Urine pH	Urine Culture	Serum Calcium (mg. per cent)	Roentgenologic Tumor Size (cm.)
I. J.M.	88	M	Transitional cell carcinoma	Surface of epithelium (within fronds of tumor)	Nodular	8.0	Sterile	9.5	5
II. G.R.	78	F	Squamous cell carcinoma	Surface of epithelium	Granular	7.5	Alpha *Streptococcus*	8.8	5
III. B.A.	69	M	Squamous and transitional cell carcinoma	Surface of epithelium	Granular and Nodular	7.0	Sterile	9.0	4
IV. R.T.	63	F	Transitional cell carcinoma	Subepithelial (in islands of hyaline)	Granular and Nodular	—	Sterile	—	4
V. E.I.	67	F	Transitional cell carcinoma	Subepithelial	Granular and Nodular	6.0	*B. proteus* *E. coli*	—	5

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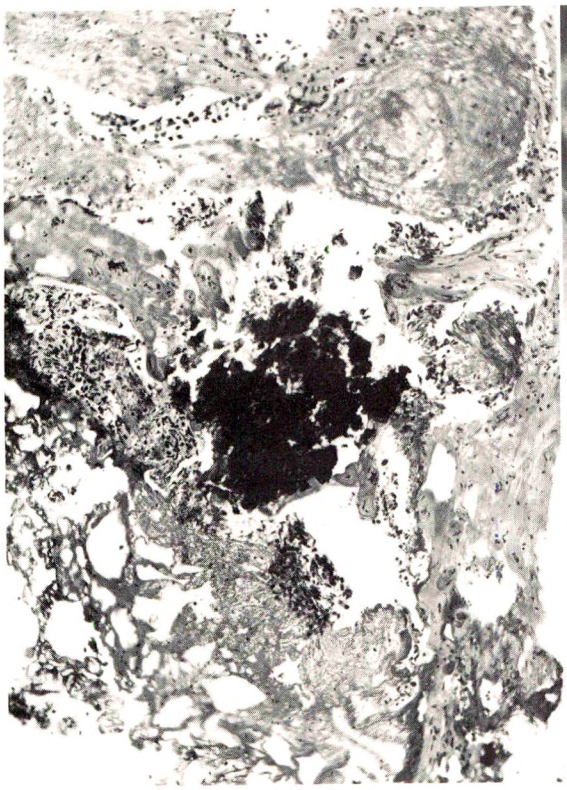


Fig. 1. Case IV. Subepithelial calcification (central dark area), adjacent to infiltrating transitional cell carcinoma.

tumor with the calcification usually in an area of necrosis but also occurring in islands of hyaline (Fig. 1).

The urine was alkaline in 3 of 4 patients in whom data were available, and these had calcium deposited on the surface of the tumor. Two patients had associated urinary tract infections, with 1 having calcium encrusted on the surface epithelium and the other having subepithelial tumor calcification. Three patients in whom serum calcium determinations were made had normal values.

The roentgenologic appearance of tumor calcification was of the fine granular variety (1 case) (Fig. 2), coarsely nodular pattern (1 case) (Fig. 3, *A* and *B*) probably representing conglomerate deposition, or most frequently (3 cases) a mixture of the 2 types (Fig. 4, *A* and *B*; 5, *A* and *B*; and 6, *A* and *B*). Only a portion of the total tumor mass was calcified and in 4 cases it

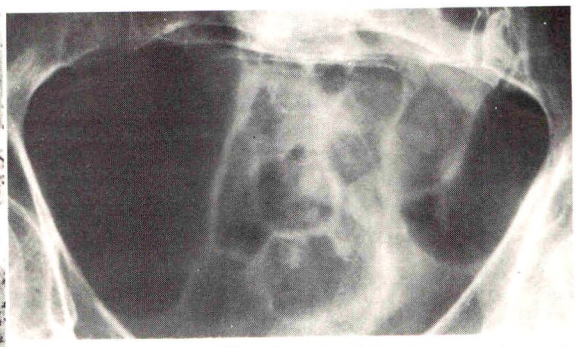


Fig. 2. Case II. Wide band of finely granular calcifications on the surface (encrusted) of squamous cell carcinoma.

was distributed in a hemispherical pattern along the convex outer edge of the tumor (Fig. 2; 3, *A* and *B*; 4, *A* and *B*; 5, *A* and *B*; and 6, *A* and *B*).

To determine the frequency of calcification in uroepithelial bladder tumors, a computer search of medical records between 1967–1972 was made. During this

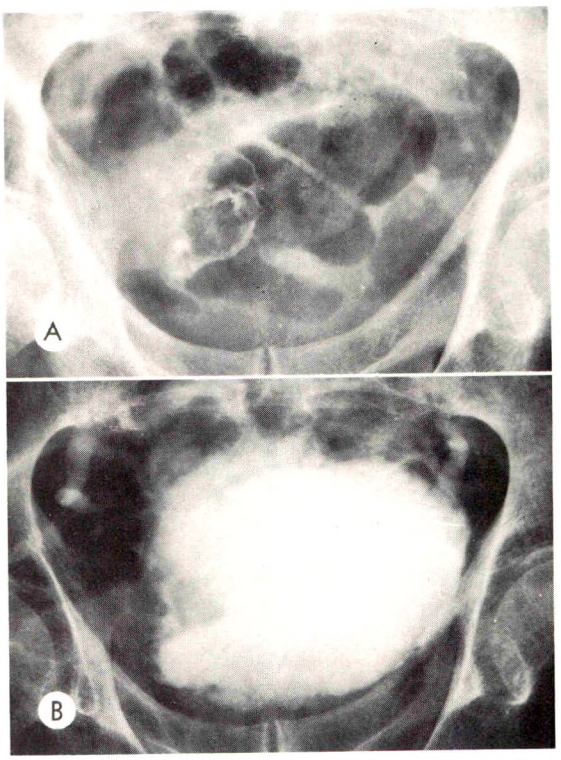


Fig. 3. Case I. (*A*) Nodular calcifications on surface of large transitional cell neoplasm. (*B*) Corresponding opacified bladder showing correlating tumor size.

period, there were 465 patients who had a diagnosis of primary epithelial carcinoma of the bladder. Two cases of calcified bladder tumors were encountered in this population, giving an incidence of roentgenologic calcification in bladder carcinoma of 0.43 per cent.

DISCUSSION

Although histologic calcification occurs rather frequently in bladder tumors,[8] the size of the individual calcium deposit is usually too minute to be seen roentgenologically. Eleven well documented cases of roentgenologically demonstrated calcification in uroepithelial tumors of the bladder have been reported in the English language literature (Table II), although this would not seem to be a true reflection of its occurrence since other illustrations can be found.[2,5,8]

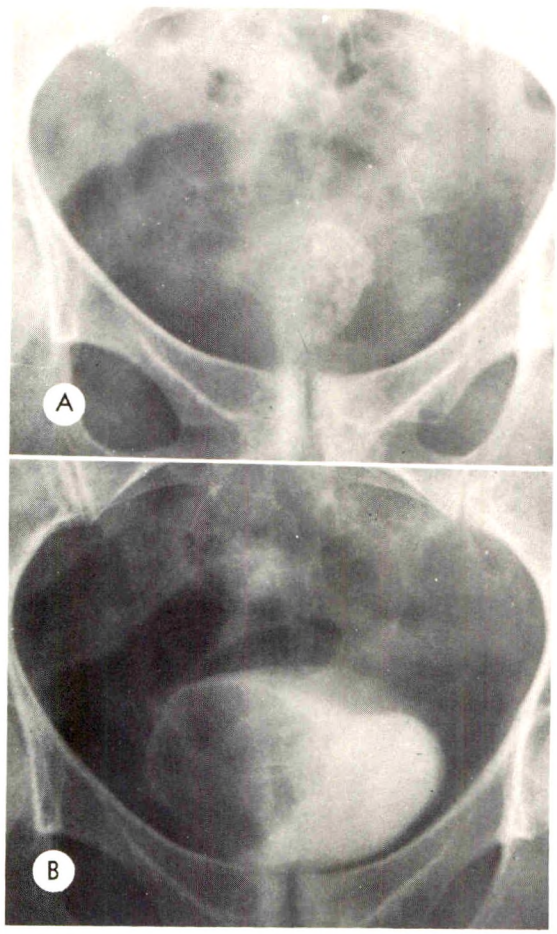


FIG. 4. Case V. (*A*) Mixed granular and nodular calcifications in subepithelial location involving left side of transitional cell carcinoma and (*B*) only a portion of the large neoplasm is calcified.

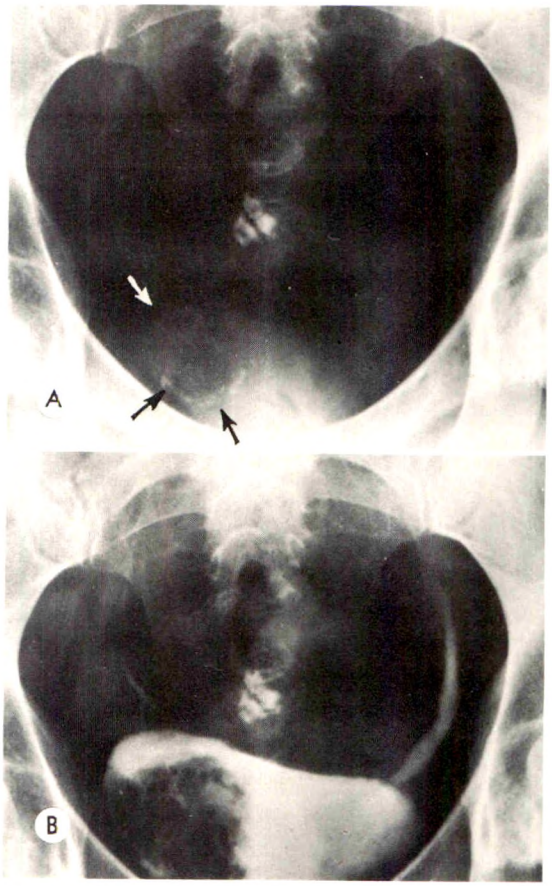


FIG. 5. Case IV. (*A*) Mixed soft granular-nodular calcifications (arrows) in subepithelial position of transitional cell neoplasm. (*B*) Larger tumor volume readily apparent. Note unassociated supravesical calcifications.

The incidence of roentgenologic calcium in bladder cancer has varied between 0.69–6.7 per cent;[3,6] however, the latter figure seems much too high for a large series. In a review of 732 cases of bladder tumors that underwent urography, Braband[3] found 5 cases (0.69 per cent) that contained roentgenographic tumor calcification while Ferris and O'Connor[6] encountered 5 cases among 75 patients with bladder cancer. Our experience indicates an

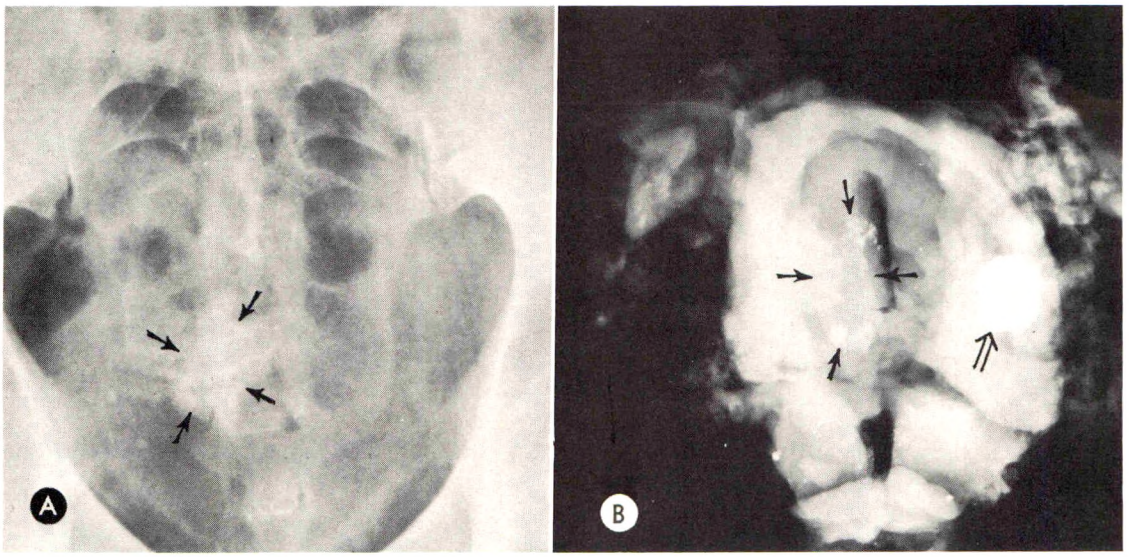


FIG. 6. Case III. (*A*) Dense mixed granular-nodular calcification pattern overlying sacrum (arrows) and (*B*) specimen roentgenogram showing surface calcifications on transitional-squamous cell carcinoma (arrows) compared to a calcified mesenteric lymph node (double-tailed arrow).

incidence of 0.43 per cent which concurs with Braband's findings.

Calcification of bladder tumors may be either within the tissue or on its surface. Calcification in neoplasms, excluding those in which the tumor matrix actually produces calcium or osteoid, has been classified as dystrophic.[1,10] Dystrophic calcification usually is found in injured, degenerating, or necrotic tissue. By definition, dystrophic calcification occurs with normal levels of blood calcium, but may be augmented by hypercalcemic states. In our 2 cases of subepithelial calcification, the deposits occurred in the center of small regions of necrosis and adjacent to areas of hyaline change. Interestingly, dystrophic calcification in bladder carcinoma has also been reported in animals.[9]

The most common location of bladder calcium is on the surface (encrustation) of the tumor epithelium. Encrustation of

TABLE II

ROENTGENOLOGIC CALCIFICATION OF EPITHELIAL BLADDER TUMORS

Author	Year	No. of Cases	Incidence	Pathologic Diagnosis
Braband[3]	1961	5	0.69% (732 tumors)	"Epithelial cell carcinoma"
Davidson *et al.*[4]*	1965	1	—	Transitional cell carcinoma
Ferris and O'Connor[6]	1965	5	6.7% (75 tumors)	Transitional cell carcinoma—4 Squamous cell carcinoma—1
Miller and Pfister	1974	5	0.43% (465 tumors)	Transitional cell carcinoma—3 Squamous cell carcinoma—1 Transitional-Squamous carcinoma—1

* Other cases in this report were nonepithelial in origin (osteogenic sarcoma, leiomyosarcoma).

calcium salts on a non-necrotic neoplasm may reflect a local pH interaction of the tumor and the normal urinary calcium bathing the bladder cancer. Calcium precipitation is favored by an alkaline pH, and this was possibly operative in 3 of our cases in which tumor surface calcification occurred.

The fine granular calcifications that are occasionally found histologically in nonspecific cystitis with an alkaline urine are rarely, if ever, seen roentgenologically. Also, histologically, cystitis calcification with alkaline urine is diffusely and uniformly encrusted on the bladder epithelium. In contrast, the calcium in or on the tumors is localized to the area of the bladder containing the neoplasm (Fig. 3, *A* and *B*; 4, *A* and *B*; 5, *A* and *B*; and 6, *A* and *B*).

Peripherally located calcium deposits in the bladder wall may be encountered in severe tuberculous cystitis and healing phase of schistosomiasis. No intraluminal mass is present in the former, and multiple smooth, small nodular filling defects are features in the initial phase of the latter, while the calcified bladder tumor presents as a large, bulky, irregular eccentric mass on urography or cystography.

The calcification in bladder tumors has sufficient characteristic features to be distinguished on plain film roentgenograms from other more common pelvic calcifications (Fig. 5, *A* and *B*), such as vesical calculi, phleboliths, uterine fibroids, ovarian dermoids, and calcium in vas deferens, seminal vesicles or arterial walls.

SUMMARY

Roentgenologic calcification of transitional and squamous cell cancers of the bladder is an uncommon but certainly not rare finding: in this and another large series it occurs in approximately 0.5 per cent of cases.

The tumors tend to be large, and the calcium may be encrusted on the surface of the neoplasm or lie deeper in a subepithelial position in degenerating or necrotic areas.

Pathologically, the scattered or conglomerate distribution of the calcium results in a roentgenologic granular or nodular appearance usually in a semicircular pattern involving only a portion of the tumor mass as determined by urography or cystography.

Bladder tumor calcification is sufficiently distinct to be differentiated from other more common calcifications occurring in the pelvis.

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REFERENCES

1. Barr, D. P. Pathological calcification. *Physiol. Rev.*, 1932, *12*, 593–624.
2. Bartholomew, L. G., Cain, J. C., Davis, G. D., and Bulbulian, A. H. Misleading calcific shadows in abdomen. *Postgrad. Med.*, 1961, *30*, 51–66.
3. Braband, H. Incidence of urographic findings in tumours of urinary bladder. *Brit. J. Radiol.*, 1961, *34*, 625–629.
4. Davidson, H. D., Witten, D. M., and Culp, O. S. Roentgenologically demonstrable calcification in tumors of bladder: report of three cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *95*, 450–454.
5. Emmett, J. L., and Witten, D. M. Clinical Urography: An Atlas and Textbook of Roentgenologic Diagnosis. Third edition. W. B. Saunders Company, Philadelphia, 1971, pp. 1273–1275.
6. Ferris, E. J., and O'Connor, S. J. Calcification in urinary bladder tumors. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *95*, 447–449.
7. McAfee, J. G., and Donner, M. W. Differential diagnosis of calcifications encountered in abdominal radiographs. *Am. J. M. Sc.*, 1962, *243*, 609–650.
8. Ney, C., and Friedenberg, R. M. Radiographic Atlas of the Genitourinary System. J. B. Lippincott Company, Philadelphia, 1966, p. 541.
9. O'Brien, T. R. Neoplasia with dystrophic calcifications in dog. *J. Am. Vet. Med. A.*, 1970, *157*, 974–976.
10. Widmann, B. P., Ostrum, H. W., and Freed, H. Practical aspects of calcification and ossification in various body tissues. *Radiology*, 1938, *30*, 598–609.

RADIATION THERAPY AND PELVIC NODE DISSECTION IN THE MANAGEMENT OF CANCER OF THE PROSTATE*

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DURING the period 1961 to 1969, 10 patients with Stage C cancer of the prostate were treated at Memorial Hospital by interstitial implantation of encapsulated radioactive sources. Radon 222 and iridium 192 were used in 5 patients up to 1965, and iodine 125 low energy gamma ray encapsulated sources, developed at Memorial Hospital, were used in the other 5 patients. The use of iodine 125 sources resulted in marked reduction of radiation exposure to personnel, while maintaining a high radiation dose throughout the implanted prostate. Five of these 10 patients are alive without evidence of active disease for periods ranging from $3\frac{1}{2}$ to 12 years after implantation with no late rectal or urinary complications. Encouraged by this experience, an investigation was begun in 1970 at Memorial Hospital to determine the effects of localized high radiation with encapsulated iodine 125 sources in clinical Stage B and C cancer of the prostate.

MATERIAL AND METHOD

From February 1970 to February 1973, 60 patients with cancer of the prostate underwent a combined procedure consisting of a retropubic *en bloc* pelvic lymphadenectomy and interstitial implantation of the prostate with iodine 125 seeds; 29 patients with a clinical Stage B lesion, and 31 patients with a clinical Stage C lesion. The Stage B cancers varied from small nodules within one prostatic lobe to lesions occupying most of the gland. The Stage C lesions were generally those with sharply defined extracapsular extension.[5] Cystoendoscopy was performed as part of the staging procedure in most of the patients. In all patients there was no clinical evidence of distant metastases by physical examination, skeletal survey, chest roentgenograms and excretory urograms. The serum acid and alkaline phosphatase values were elevated in 3 patients and 4 patients, respectively. Liver and bone scans, lymphangiograms and bone marrow aspirations were done preoperatively in some patients, but not routinely.

SURGICAL PROCEDURE

The retropubic approach was used because it permits a more accurate determination of the extent of tumor in the prostate, allows inspection and dissection of the nodes in the pelvis, and facilitates the implantation of radioactive sources. The morbidity with the retropubic approach is lower than with the suprapubic approach. The operation is performed under general or spinal anesthesia. The patient is placed in a modified lithotomy position, prepared with an O'Connor drape, and a Foley catheter is inserted into the bladder. Through a midline incision extending from the symphysis to the umbilicus an extraperitoneal bilateral pelvic lymphadenectomy is done. The pelvic nodes are dissected below the distal portion of the common iliac arteries. After completion of the dissection bilaterally, the incision is carried into the endopelvic fascia to facilitate prostatic rotation and mobilization. The prostate is then exposed anteriorly and laterally so that it can be inspected, pal-

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pated, outlined with surgical clips, and implanted with the radioactive seeds.

IMPLANTATION PROCEDURE

First, hollow stainless steel needles, 17 gauge and 15 cm. long, are inserted into the prostate. The needles are placed parallel to each other about 1 cm. apart, starting from the superior medial margin of the gland near the bladder base. Care is taken to avoid the rectal wall or the lumen of the prostatic urethra. The former is accomplished by keeping the index finger inside the rectum during the insertion of the needles so that their tips can be sensed before entering the rectum (Fig. 1); the latter is minimized by avoiding penetration of the urethral catheter with the needles. The dimensions of the volume to be implanted are then estimated from the width and length between the most peripheral needles; the third dimension is estimated by subtracting the average length of the needles projecting outside the prostate from the over-all length of these needles (15 cm.). The average of the estimated 3 dimensions multiplied by an empirically derived factor of 5, indicates the number of millicuries of iodine 125 required to deliver a minimum effective dose within the implanted volume. Preliminary analysis of the available information shows that this dose is in the range of 16,000 rads in 1 year; its equivalent single dose (ESD) has been calculated[2] to be in the range of 5,300 rads. The value in millicuries is divided by the average activity of the seeds on stock to obtain the number of seeds needed. Using a semi-automatic inserter, which was developed at Memorial Hospital, the seeds are inserted at 1 to 1.5 cm. intervals as each needle is withdrawn. After completion of the implantation, the surgical incision is closed leaving a Penrose drain in its lower angle.

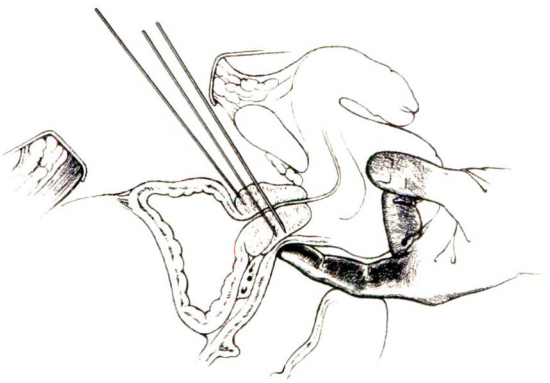


Fig. 1. Implantation procedure of iodine 125 sources. Hollow stainless steel needles, 17 gauge and 15 cm. long, are inserted into the prostate. The needles are placed parallel to each other, about 1 cm. apart. To avoid the rectal wall, the index finger is placed inside the rectum, so that their tips can be sensed before entering the rectum.

RESULTS

PELVIC LYMPHADENECTOMY

On histopathologic examination the resected pelvic lymph nodes were found to be involved by tumor in 26 of 60 patients (43 per cent). Lymph node metastases were more frequent in clinical Stage C patients (20 of 31, 65 per cent) than in clinical Stage B patients (6 of 29, 21 per cent). The obturator lymph nodes were involved in 35 per cent of all patients (8 per cent bilaterally) and the external iliac lymph nodes were involved in 23 per cent (10 per cent bilaterally). The internal iliac (hypogastric) and the common iliac lymph nodes were involved in only 8 and 7 per cent, respectively (3 and 2 per cent bilaterally), but these data are of debatable significance, since systematic dissection of the common iliac and medially situated hypogastric lymph nodes was not performed. No information is available concerning the frequency of involvement of presacral and para-aortic lymph nodes owing to the nature of the lymphadenectomy. Figure 2 shows the frequency of regional pelvic lymph node involvement by clinical stage. None of the Stage B patients had common iliac lymph node metastases.

Correlation of the lymphangiograms with the histopathologic findings. Lymphangiograms were not done routinely in the beginning of this study, but more recently have become routine. Table 1 correlates the lymphangiographic with the histopatho-

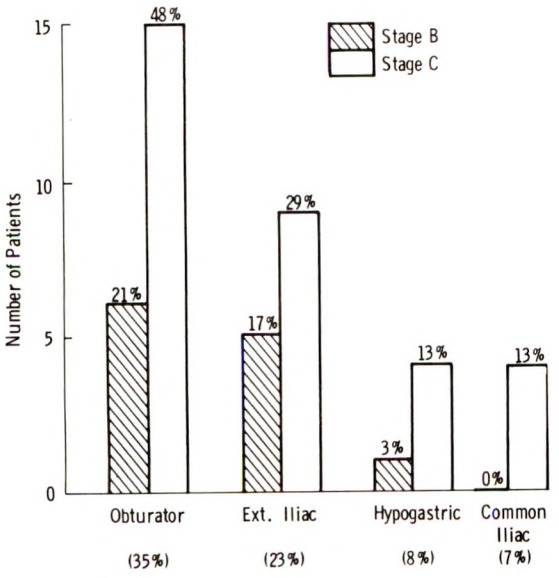


FIG. 2. Frequency of regional pelvic node groups metastases in Stage B and C.

logic findings in 15 patients. Bipedal lymphangiograms are of good diagnostic value in advanced lesions, but of less value in earlier or smaller lesions. This is possibly due to the fact that the obturator lymph nodes, the earliest and most frequently involved lymph nodes, are not consistently visualized by pedal lymphangiography.

PROSTATIC IMPLANTATION

In evaluating prostatic implantation, we have included only 49 patients with carcinoma of the prostate treated by iodine 125 interstitial implantation and pelvic lymphadenectomy up to September 1972, who provide follow-up periods of from 6 to 30 months. Ages ranged from 47 to 77 years with a median age of 60 years. Of the 49 patients, 24 had clinical Stage B tumors and 25 had clinical Stage C tumors. All tumors were histologically classified as adenocarcinomas; the majority were Grade 2 lesions. The interval from diagnosis to treatment ranged from 0 to 46 months with a median of 8.9 months in Stage B patients, and from 0 to 30 months with a median of 5 months in Stage C patients. One patient, previously treated by external irradiation (7,000 rads in 8 weeks), had persistent disease at the time of implantation. Six other patients had been on hormonal therapy for periods of 1 to 3 months prior to referral to Memorial Hospital.

Tumor regression. Decrease in prostatic size and induration does not necessarily indicate tumor eradication but follow-up biopsies have not been performed routinely. Clinical evidence of tumor regression was based on rectal examination 6 to 24 months after implantation. In some patients there was an appreciable increase in the prostatic size and induration a few weeks after the implantation, presumably the result of either the mechanical trauma caused by the insertion of needles, or a radiation-induced edema, or both. Nevertheless, on subsequent examinations there was a gradual decrease in the prostatic size and induration. At 6 months, 44 per cent of the prostatic tumors were smaller and softer. At the end of the first year, when 100 per cent of the nominal dose had been delivered, 57 per cent of the implanted tumors were smaller, while 43 per cent remained unchanged. At the end of the second year after implantation, 78 per cent of the tumors were considered to be reduced in size with variable persistence of induration; 22 per cent showed no apparent change in size despite a softened consistency in some tumors.

Post-treatment morbidity. The median hospital stay after treatment was 8 days; the shortest time was 4 days and the longest was 19 days. The Foley catheter

TABLE I

CARCINOMA OF PROSTATE, STAGE B AND C
CORRELATION OF LYMPHANGIOGRAM (LAG)
AND HISTOLOGIC FINDINGS

Stage	LAG Positive	Histology Positive	LAG Accuracy
B	0/8	1/8	87%
C	4/7	4/7	100%
B and C	4/15	5/15	91%

was removed 1 to 3 days and the Penrose drain 3 to 4 days postoperatively. Blood loss during surgery ranged from 50 to 2,000 cc. with a median quantity of 750 cc. Thirty-five of the 49 patients (71 per cent) had no complications. Thirteen patients had early minor complications, including suprapubic wound infection in 9 patients, dysuria in 1 patient, epididymitis in 2 patients, and rectal burning in 1 patient. The only patient who developed severe complications underwent transurethral resection of the prostate 1 week prior to the implantation of the remaining prostate with iodine 125 seeds; he subsequently had prolonged suprapubic urinary drainage as well as wound infection. Fever was present in 9 patients (20 per cent) for the first 2 to 3 days, but subsided thereafter. Late minor complications which could be attributed to the radiation itself consisted mainly of transient mild rectal pain in 3 patients and temporary rectal bleeding in another; all patients had subsidence of their symptoms without treatment within 3 to 6 months after the implantation. The only severe late complication was noted in the patient who had received 7,000 rads by supervoltage radiation several months prior to the implantation; 3 months after implantation, he experienced severe tenesmus and rectal pain which lasted for 6 months before subsiding with conservative treatment. None of the patients who were followed for periods up to 30 months after implantation developed rectal ulcerations, fistulae or urethral strictures. Late urinary symptoms were observed in 5 patients with pre-existing obstructive symptoms; these were aggravated after implantation, but subsequently subsided within 4 to 6 months in 4 of them. The fifth patient had persistent obstructive urinary symptoms for 1 year.

Sexual potency. The sexual function before and after implantation was known in 47 of the 49 patients. All 26 patients who claimed sexual potency prior to the implantation, maintained their potency postoperatively up to the latest follow-up. Of 7 patients with diminished potency before implantation, 2 became impotent postoperatively and 5 maintained some sexual activity. Of 14 patients who admitted impotency prior to the treatment, 2 resumed some sexual activity after treatment and the others remained impotent.

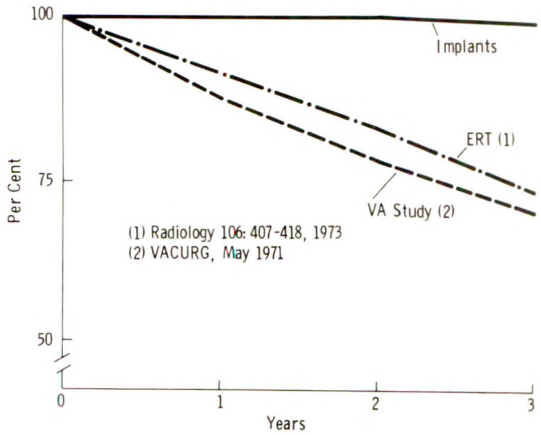


Fig. 3. Actuarial survival in Stage B and C.

Survival and distant metastases. Forty-eight of the 49 patients treated from 6 to 30 months ago are alive at the time of this report. Figure 3 shows the per cent actuarial survival and correlates it with the survival of patients treated by external irradiation only[3] as well as with the survival of patients of similar stages treated by prostatectomy or endocrine therapy.[4] The only death occurred in a 51 year old patient with clinical Stage C cancer who had positive lymph nodes; he developed distant metastases to bone, lungs and upper abdomen 23 months after his initial treatment and expired 7 months later. Distant metastases were observed in 2 patients with clinical Stage B cancer of the prostate and 9 patients with clinical Stage C cancer. All the patients who developed distant metastases had either positive lymph nodes at multiple levels or a Grade 3 primary prostatic cancer. The involvement of pelvic lymph nodes by metastatic cancer emerges as a most significant prognostic factor. This is illustrated in Table II, in which the incidence of distant metastases is related to the status of the pelvic lymph nodes, at various

TABLE II

CARCINOMA OF PROSTATE, STAGE B AND C
INCIDENCE OF DISTANT METASTASES AS RELATED TO NODAL STATUS AT INITIAL TREATMENT

Nodal Status	Interval After Initial Treatment		
	6 mo.	12 mo.	24 mo.
Negative	0/28 (0)	1/16 (6%)	1/8 (12.5%)
Positive	2/21 (9.5%)	3/17 (17.5%)	6/10 (60%)
All Patients	2/49 (4%)	4/33 (12%)	7/18 (39%)

intervals after treatment. All patients with distant metastases have subsequently received endocrine therapy (diethylstilbestrol 1 mg./day) or palliative external radiation therapy, or both to relieve pain caused by bone metastases. They are alive and generally asymptomatic except for the patient mentioned earlier.

Extended field nodal irradiation. Postoperative external irradiation through opposing inverted Y-fields extending from the diaphragm to the inguinal regions was given to 7 of the 21 patients with positive pelvic lymph nodes. It consisted of 4,500 rads delivered in about $4\frac{1}{2}$ weeks with the 6 mev. Linear Accelerator. This supplementary external irradiation was well tolerated and has not led to any late complications up to 24 months following its administration. Distant metastases have developed in 4 of these 7 patients, while distant metastases developed in 6 of 14 patients who had positive lymph nodes but did not receive such irradiation postoperatively. The disease-free mean interval is 18.0 months and 12.5 months, correspondingly. Because of the small number of patients in each group, there is no statistically significant difference either in the incidence of distant metastases or in the disease-free mean interval.

DISCUSSION

The combined approach of interstitial irradiation of the prostate with iodine 125 encapsulated sources, retropubic pelvic lymphadenectomy and, in selected patients, postoperative supervoltage radiation to the regional lymph nodes has been applied in patients with Stage B and C prostatic cancer at Memorial Hospital since 1970.

The retropubic exposure, used in this method of treatment, has allowed a more accurate assessment of the true extent of the disease in the primary site and in the regional lymph nodes, thus improving the accuracy of the staging.

The pelvic lymphadenectomy has diagnostic value by permitting a histologic confirmation of the lymphangiographic findings; it also has therapeutic value by removing the nodes involved by metastatic cancer.

Interstitial implantation of the iodine 125 encapsulated sources has certain advantages over external radiation therapy. The 100 per cent tumor dose represents the maximum dose, if given by rotation or multiple small fields; it represents the minimum effective dose by implantation. The localization of the tumor bearing area is more precise and the dose distribution can be more readily adapted to an irregular tumor shape (Fig. 4). The dose falls rapidly outside of the implanted volume; the bladder and rectum receive 50 per cent or less of the minimum tumor dose (Fig. 5). Finally, the treatment time is much shorter; a permanent implant requires only a single procedure, while a complete course of external radiation therapy usually requires 6 to 7 weeks. The postoperative hospitalization following implantation

has been short in general (about 1 week's duration).

Tumor regression following interstitial irradiation is slow, reaching its maximum in about 2 years after treatment. There is a striking similarity in the percentage of patients who show tumor regression at the end of external supervoltage radiation (59 per cent)[3] and interstitial radiation (57 per cent) allowing, of course, for the time required to complete the treatment with each of these 2 modalities. The given minimum effective dose of 16,000 rads in 1 year, or its single equivalent dose of 5,300 rads, has resulted in approximately 80 per cent local tumor control at 2 years after implantation, as demonstrated by rectal palpation.

The relatively short follow-up does not allow for a meaningful comparison of survival with other types of treatment. The survival rate of 99 per cent at 3 years, calculated by the actuarial method is not significantly different from the 3 year actuarial survival of 84 per cent obtained by small field supervoltage irradiation[3] or the 88 per cent obtained by radical perineal prostatectomy.[1] The V.A. cooperative study (patients who were treated by radical prostatectomy and placebo in Stage B and by hormones in Stage C) shows a 3 year survival of 74 per cent.[4] Survival, however, must be supported by a good quality in order to be meaningful. Maintenance of sexual potency is an important factor for many younger patients. Most of the patients who undergo radical prostatectomy or endocrine therapy become impotent; 70 per cent of patients undergoing external small-field radiation therapy maintained their potency; all of the patients who were potent before implantation of iodine 125 sources have remained potent up to 30 months after treatment. Complications caused by the treatment should also be considered. There was no mortality associated with the combined procedure. None of the treated patients has developed severe early or late complications during the period of follow-up (6 to 30 months), such as rectal ulcerations, fistulae, urinary incontinence, or urethral stricture.

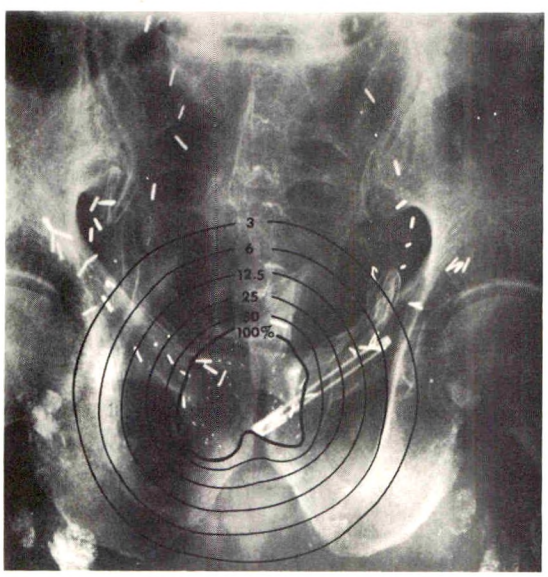


Fig. 4. Localization of tumor bearing area and isodose distribution in an irregular tumor shape.



Fig. 5. The dose falls rapidly outside the implanted volume, and the bladder and rectum receive 50 per cent or less of the minimum tumor dose. Also the treatment time is much shorter.

CONCLUSION

The treatment method presented is thus suitable for patients with clinical Stage B cancer as an alternative method to radical prostatectomy or small-field external supervoltage irradiation. In patients with clinical Stage C prostatic cancer and a high probability of metastases to the regional lymph nodes, it is logical to combine this method with supplemental supervoltage radiation to the paraaortic and common iliac lymph nodes in moderate doses (4,500 rads in 4 to $4\frac{1}{2}$ weeks). Doses exceeding the tolerance of the normal pelvic organs will carry a higher morbidity when combined with an interstitial implant, as it was observed in the patient who received 7,000 rads prior to implantation.

SUMMARY

A technique involving a pelvic lymphadenectomy and a retropubic implantation of the prostate with iodine 125 encapsulated sources is under investigation at Memorial Hospital.

This technique is simple and applicable to patients with localized cancer of the prostate, clinically stage B or C. It has increased the accuracy of staging, carries no mortality and has low morbidity.

The results have been encouraging with substantial local tumor control and no late urinary or rectal disturbances in the 49 patients who have been followed from 6 to 30 months.

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The authors are indebted to Dr. J. S. Laughlin, Dr. G. Holt, Dr. L. Anderson and their staff of the Department of Medical Physics, for their support in carrying out the necessary dosimetric studies during the period of this study.

REFERENCES

1. Jewett, H. J., Bridge, R. W., and Gray, G. F., Jr. Palpable nodule of prostatic cancer. *J.A.M.A.*, 1968, *203*, 403–406.
2. Kim, J. H., and Hilaris, B. S. Unpublished data.
3. Ray, G. R., Cassady, J. R., and Bagshaw, M. A. Definitive radiation therapy of carcinoma of prostate: report on 15 years of experience. *Radiology*, 1973, *106*, 407–418.
4. The Veterans Administration Co-operative Urological Research Group. Carcinoma of prostate: clinical trial results. *Minnesota Med.*, 1971, *5*, 511–514.
5. Whitmore, W. F. Rationale and results of ablative surgery for prostatic cancer. *Cancer*, 1963, *16*, 1119–1132.

Vol. 121, No. 4

PERCUTANEOUS NEEDLE LOCALIZATION OF CLUSTERED MAMMARY MICROCALCIFICATIONS PRIOR TO BIOPSY*

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IN recent years the increasing use of mammography has presented surgeons and radiologists with a distinct problem; *i.e.*, the isolation and excision of suspicious appearing clustered microcalcifications visualized on mammography which have no associated soft tissue mass without removing a major portion of the patient's breast. The incidence of calcification present in breast cancer as determined by clinical mammography varies from series to series (Levitan *et al.*,[7] 29 per cent; Gershon-Cohen *et al.*,[4] 35–40 per cent; Egan,[2] 35–45 per cent; and Zuckerman,[9] 63 per cent). However, in patients with clustered tiny microcalcifications the incidence of cancer, especially of intraductal carcinoma and lobular carcinoma *in situ* is increased.[5,6,8] Therefore, there is a significant possibility in a patient with suspicious clustered microcalcifications that: (1) a carcinoma is present; and (2) an "early" breast cancer (small, noninvasive and without metastases) may be found. Exact localization, excision and pathologic examination of these microcalcifications is imperative.

Radiologists have attempted to localize these microcalcifications within the breast in 2 dimensions by the use of at least 2 film projections; however, this is not always a satisfactory method since the position of the patient during biopsy (recumbent) is quite different from the position for filming (upright). Recently, Frankl and Rosenfeld[3] have reported using strips of numbers taped to the skin, after which mammograms of the breast are obtained. This technique is stated to localize the microcalcifications more closely for a surgeon.

METHOD

At the University of Michigan Hospital we have been using a different technique to accomplish localization of these microcalcifications. This technique—the percutaneous needle localization of suspicious microcalcifications—has been in use since January 1973 for rapid localization of microcalcifications during biopsy. Minimal amounts of breast tissue were removed and roentgenographed from each patient.

A long 25 gauge needle is passed through the skin without anesthesia to the region of the suspicious clustered microcalcification. This is done by gauging or estimating the position of the microcalcification as visualized from the 2 planes of the mammogram and then directing the needle tip toward the supposed area of the microcalcification. The needle is usually passed to its hub; then craniocaudal and lateral mammograms are obtained. Little additional radiation is given to the breast since a low dose screen film combination (Du Pont Lo-Dose) is used. If the needle tip is at, in, or very near the microcalcification, the needle is taped securely into place. This needle placement may be performed immediately prior to biopsy or even the afternoon preceding the biopsy. Our patients have tolerated the introduction and presence of this small needle very well; they were cognizant of the corresponding reduction in size of the biopsy specimen due to precise localization of the microcalcifications by the percutaneous needle localization technique. There were no complications from this procedure.

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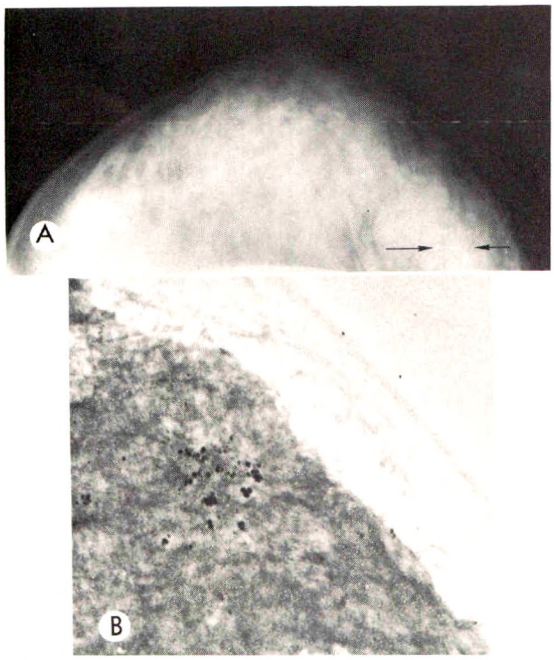


FIG. 1. Case 1. (*A*) Craniocaudal projection with clustered microcalcifications laterally in the right breast. (*B*) Magnification view of the clustered microcalcifications.

Dodd[1] recently reported a somewhat similar technique; however, he uses skin anesthesia and 2 needles for the localization. We have not found either of these to be necessary for accurate localization of the microcalcifications.

REPORT OF CASES

CASE 1. L.S., a 50 year old white female, presented with a nontender mass in the medial aspect of the right breast. Mammography revealed a cluster of suspicious calcifications in the outer aspect of the right breast (Fig. 1, *A* and *B*). Percutaneous needle localization of this cluster of microcalcifications was easily accomplished and confirmed by mammograms in craniocaudal and lateral projections (Fig. 2, *A* and *B*). The needle tip was within the cluster of calcifications. This procedure was performed immediately prior to biopsy. A small specimen was obtained; this was roentgenographed to localize the microcalcifications (Fig. 3). The pathologic diagnosis was extensive fibrocystic disease with multiple tiny intraductal calcifications.

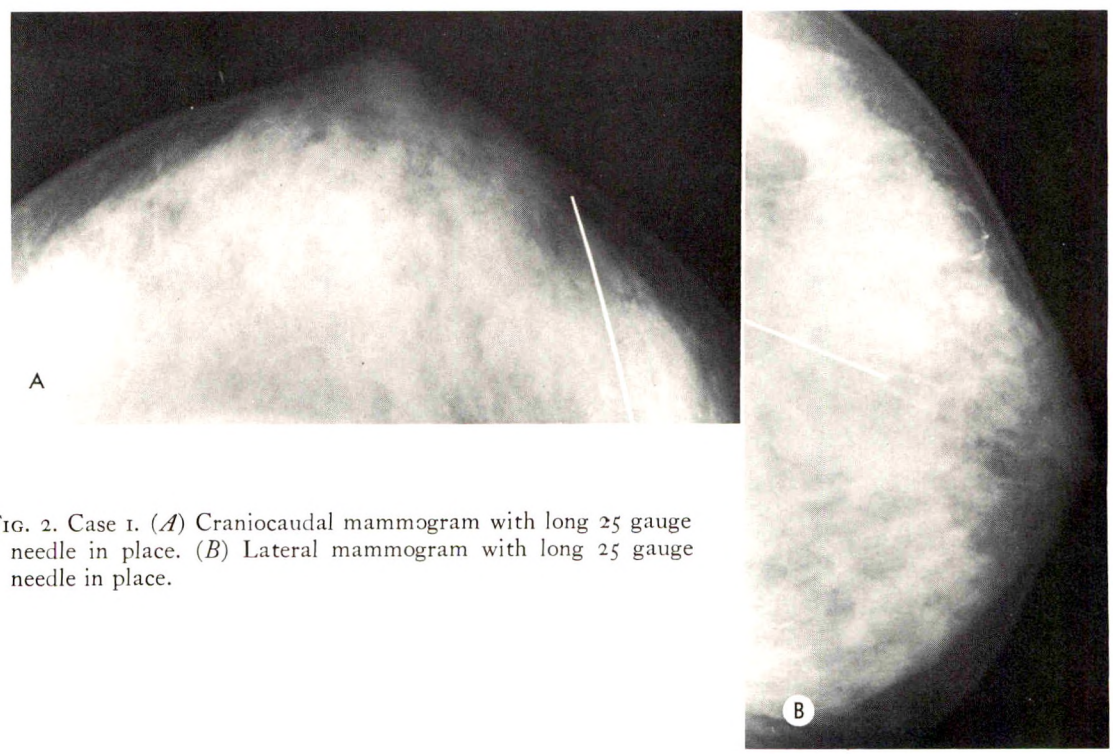


FIG. 2. Case 1. (*A*) Craniocaudal mammogram with long 25 gauge needle in place. (*B*) Lateral mammogram with long 25 gauge needle in place.

Case II. M.S., a 60 year old white female with right breast pain, had suspicious microcalcifications without an associated mass in the left breast on mammography. The afternoon prior to biopsy a long 25 gauge needle was positioned near the microcalcifications in the left breast (Fig. 4, *A* and *B*). This position was monitored by craniocaudal and lateral mammograms. The needle remained taped in position overnight without displacement or significant discomfort. The specimen was roentgenographed to assure removal of the microcalcifications. These were localized for the pathologist. The pathologic diagnosis was periductal fibrosis, fibrocystic disease and a tiny focus of fat necrosis with calcifications.

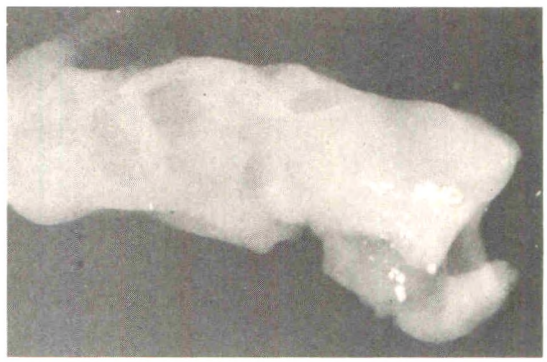


Fig. 3. Case I. Roentgenogram of right breast specimen containing clustered microcalcifications.

Case III. C.C., a 51 year old Black female who presented with multiple pulmonary nodules, had mammograms as part of a medical evaluation. Suspicious clustered microcalcifications without an associated mass were noted in the right breast. Percutaneous needle localization of these microcalcifications was easily accomplished and confirmed by craniocaudal and lateral mammograms. The localization was performed on the morning of surgery. The patient tolerated this procedure without difficulty. The biopsy specimen was roentgenographed and the microcalcifications closely localized for the pathologist. The pathologic diagnosis was intraductal hyperplasia, with slight atypia, and associated scattered calcifications, but without malignant changes.

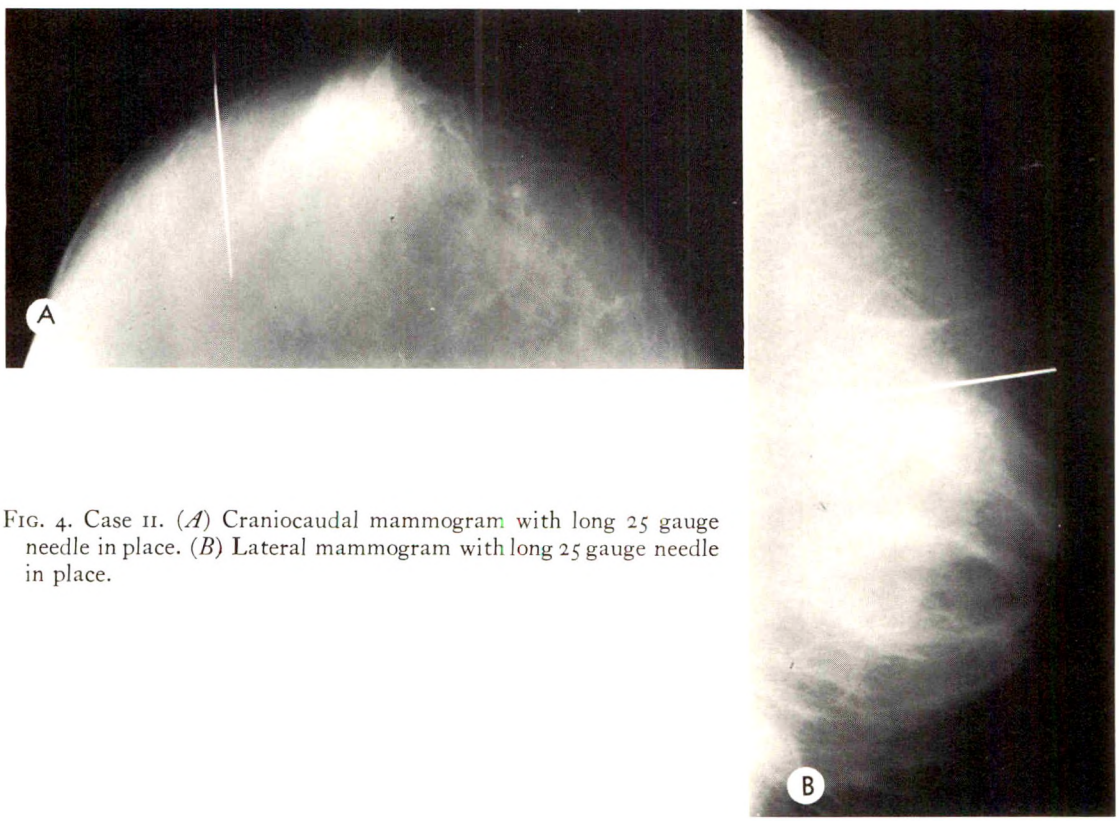


Fig. 4. Case II. (*A*) Craniocaudal mammogram with long 25 gauge needle in place. (*B*) Lateral mammogram with long 25 gauge needle in place.

DISCUSSION

Percutaneous needle localization of microcalcification is recommended prior to surgery for its accuracy, patient acceptance, and reduction in size of the biopsy specimen. We have had no complications from use of this technique. Placement of the needle within the area of the microcalcifications should not be detrimental to a patient's subsequent course since definitive resection of the breast is performed if malignancy is found.

SUMMARY

Mammography occasionally reveals the presence of suspicious appearing clustered microcalcifications without an associated mass. Clinical localization of these microcalcifications within the breast is difficult, even using the 2 dimensional effect of a mammogram.

Percutaneous needle localization of these microcalcifications is recommended for its accuracy, patient acceptance, and reduction in size of the biopsy specimen.

No complications of this procedure have been encountered.

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REFERENCES

1. Dodd, G. Preoperative radiographic localization of non-palpable lesions. Thirteenth Annual Conference on Detection and Treatment of Early Breast Cancer, Lake Buena Vista, Florida, March, 1974.
2. Egan, R. L. Experience with mammography in tumor institution: evaluation of 1,000 studies. *Radiology*, 1960, *75*, 894–900.
3. Frankl, G., and Rosenfeld, D. D. Breast xeroradiography: analysis of our first 17 months. *Ann. Surg.*, 1973, *178*, 676–679.
4. Gershon-Cohen, J., Berger, S. M., and Curcio, B. M. Breast cancer with microcalcifications: diagnostic difficulties. *Radiology*, 1966, *87*, 613–622.
5. Hutter, R. V. P., Snyder, R. E., Lucas, J. C., Foote, F. W., and Farrow, J. H. Pathogenic correlation with mammographic findings in lobular carcinoma in situ. *Cancer*, 1969, *23*, 826–839.
6. Koehl, R. H., Snyder, R. E., Hutter, R. V. P., and Foote, F. W., Jr. Incidence and significance of calcifications within operative breast specimens. *Am. J. Clin. Path.*, 1970, *53*, 3–14.
7. Levitan, L. H., Witten, D. M., and Harrison, E. G., Jr. Calcification in breast disease: mammographic-pathologic correlation. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 29–39.
8. Snyder, R. E. Mammography and lobular carcinoma in situ. *Surg., Gynec. & Obst.*, 1966, *122*, 255–260.
9. Zuckerman, H. C. Mammography in diagnosis of cancer of breast. In: Progress in Clinical Cancer. Volume I. Grune & Stratton, Inc., New York, 1965, pp. 185–212.

VOL. 121, NO. 4

A DEVICE FOR PRECISION NEEDLE BIOPSY OF THE BREAST AT MAMMOGRAPHY*

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THE best diagnostic techniques in diagnosing breast cancer at present are needle biopsy[2] and mammography performed as film mammography,[1,3,4] or xeromammography.[5]

If at mammography a tumor is detected which is not palpable, it is often very difficult to obtain a representative needle biopsy or to localize the tumor at biopsy operation. However, biopsy is of great importance, especially in cases with small tumors. Early correct diagnosis means better prognosis.

DESCRIPTION OF THE DEVICE

To make needle biopsy easier in nonpalpable tumors, the following apparatus was constructed:

The original film-holder table of a Senograph (C.G.R., France) is replaced by a similarly positioned table of the same size made of duraluminum 4 mm. thick. On 2 opposite sides of the table are placed profiled rails of the same material. The profiled rails create 2 slits. The lower slit is just above the table and is intended to hold a film 18×24 cm. The other slit is situated 2 mm. above the lower slit and holds a 1 mm. thick plexiglass screen upon which the breast is placed. On opposite sides of the table are brackets holding vertical iron rods 28 cm. long and 2 cm. in diameter, one on each side (Fig. 1 and 2). These iron rods serve as guides for a compression plate 10.5 mm. thick made of duraluminum. The compression plate holds a 1 mm. thick plexiglass window measuring 10.5×7.5 cm. The plexiglass window is perforated by 70 holes 7 mm. in diameter with an interval of 9 5 mm. between the centers of the holes. The holes are marked in a coordinate system by engraved letters and numbers. The engravings have been filled with red lead color. The compression plate can be locked at variable distances from the table by a locking mechanism. When the breast has been compressed between the plexiglass screen and the compression plate, the film can be withdrawn from its slit, while the breast remains compressed.

TECHNIQUES OF BIOPSY

In practice the device is used in the following manner: The patient is first examined by ordinary mammography. The device is then used in case a biopsy is desirable. The breast is placed on the plexiglass screen in such a way that when the breast is compressed by the compression plate, the tumor will be situated under the middle of the perforated plexiglass window of the compression plate. When the breast is compressed, the compression plate is locked and the film is exposed with the greatest possible distance between film and tube. The film is then withdrawn from its slit and developed, while the breast remains compressed. On the film the holes of the plexiglass window are easily seen and identified by the numbers and letters. By inserting a needle through that hole under which the tumor is situated, the tumor can be punctured and a needle biopsy performed. The direction of the needle should follow that of the roentgen-ray beam. The breast remains compressed until a satisfactory biopsy specimen is obtained. No anesthesia is necessary.

CONCLUSION

The described apparatus is inexpensive to construct. The technique has been used for about a year and has been found easy to perform. The method may also be used to indicate suspected areas for operative

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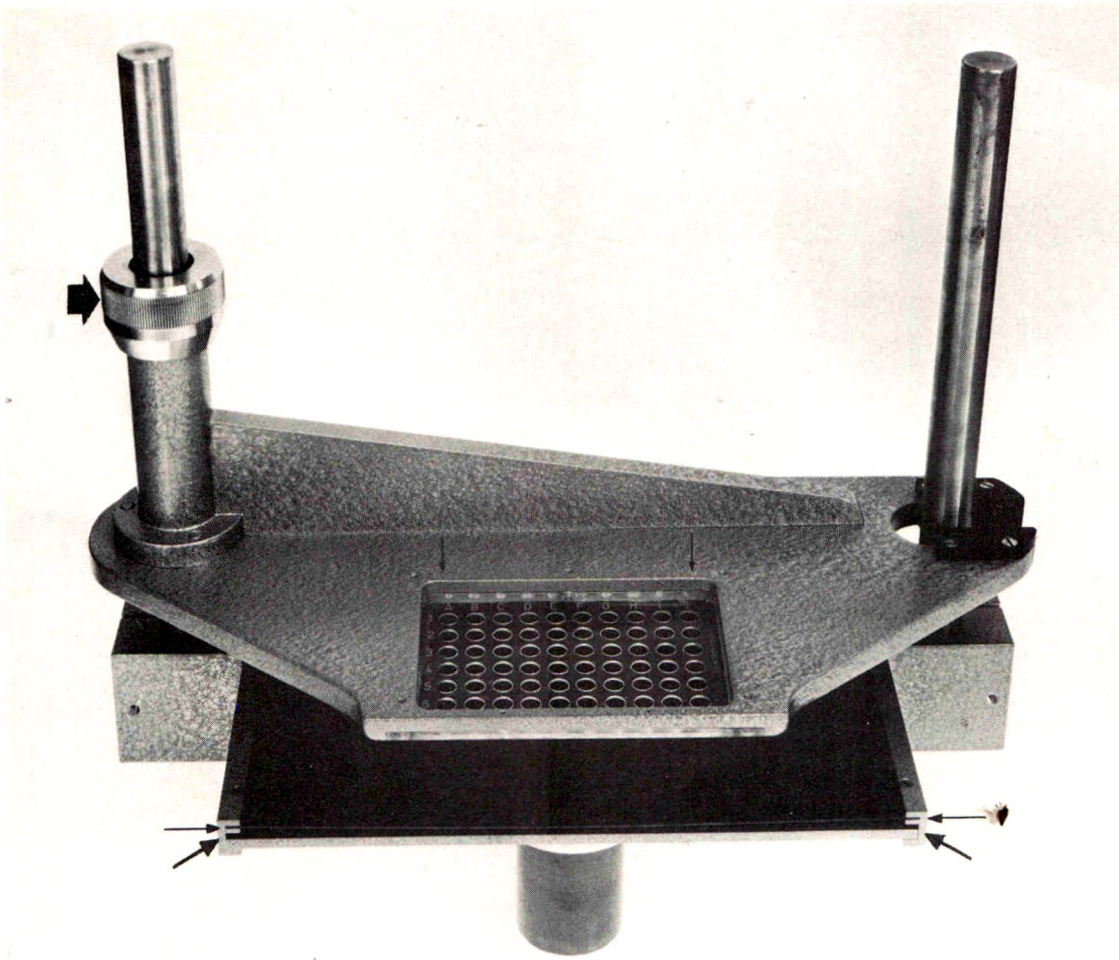


FIG. 1. Compression device. Frontal view. ↑ Perforated plexiglass window in compression plate. ↑ Plexiglass screen upon which the breast is placed 2 mm. above film slit (↑). ⬅ Locking mechanism.

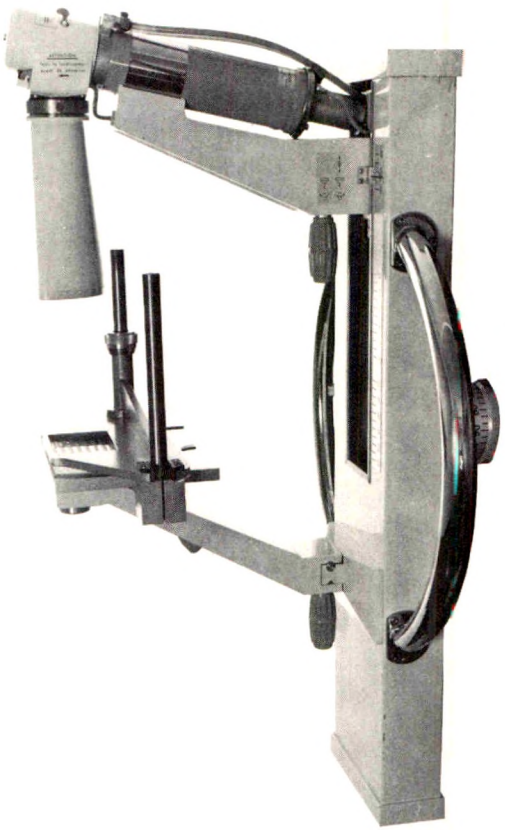


Fig. 2. Compression device mounted upon the Senograph.

biopsy by inserting long needles through the breast in 2 planes immediately before the operation. The surgeon is then able to easily find the crossing of the needles in the wound and perform the biopsy at that point.

SUMMARY

To make needle aspiration biopsy easier in nonpalpable tumors detected at mammography, a specially designed compression device has been constructed.

This device and its practical application are discussed.

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REFERENCES

1. Egan, R. L. Mammography. Charles C Thomas, Publisher, Springfield, Ill., 1964.
2. Franzén, S., and Zajicek, J. Aspiration biopsy in diagnosis of palpable lesions of breast. *Acta Radiol.* (Therapy), 1968, *7*, 241–262.
3. Gros, Ch. M. Les Maladies du Sein. Masson et Cie, Paris, 1963.
4. Ingleby, H., and Gershon-Cohen, I. Comparative Anatomy, Pathology and Roentgenology of the Breast. University of Pennsylvania Press, Philadelphia, 1960.
5. Wolfe, J. N. Xeroradiography of the Breast. Charles C Thomas, Publisher, Springfield, Ill., 1972.

ANALYSIS OF 462 BREAST CARCINOMAS*

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THE breast carcinomas that have been observed by xeroradiography of the breast at Hutzel Hospital, Detroit, were looked at as a group with, among others, the following objectives in mind: accuracy of diagnosis; frequency of occult lesions and their characterization; type and frequency of primary and secondary signs of carcinoma. Attention is called to the heretofore not sufficiently emphasized size and location of breast carcinomas and it is emphasized that a significant number occur deep within the breast. A correlation is made with certain environmental changes in the breast which are associated with carcinoma and characterize the patient most likely to have a breast cancer.

The material is drawn from a referral-type practice and it contained chiefly symptomatic women, but some nonsymptomatic ones as well. A profile of that practice can be seen in the radiographic reports of 1,000 consecutive patients (Table I).

Table I

RADIOGRAPHIC REPORT*

	No.	Per Cent
Standard normal/benign	821	82.1 (1 had ca.)
Significant benign lesion (no biopsy desired)	121	12.1
Suspicious for carcinoma	44	4.4 (2 had ca.)
Carcinoma	14	1.4 (all had ca.)
Note the low incidence of carcinoma:		1.7 per cent

* Four impressions are used at Hutzel Hospital. Suspicious has the connotation of a significant lesion, most likely benign, with a small chance of being malignant. Ideally they are biopsied. If not, they are followed. Carcinoma represents a very strong opinion.

Table II

ACCURACY OF DIAGNOSIS

(462 cases)

	No.	Per Cent
True positive	440	95.3
False negative	22	4.7

ACCURACY OF DIAGNOSIS

Every patient is not routinely followed for accuracy of radiographic diagnosis. There are several reasons, the most important of which is that the patients are drawn from a great geographic area involving multiple hospitals and many physicians not on the Hutzel Hospital staff. All cases operated upon at Hutzel Hospital are followed very closely and the results within the Hospital conform to the total shown in Table II.

SIGNIFICANT GENERAL OBSERVATIONS

There are a number of interesting and very important general observations about the entire group. Two of the most important concern the high incidence of occult carcinomas (30.1 per cent) and the great number of cases associated with calcifications (50.9 per cent) (Table III).

PRIMARY SIGNS OF CARCINOMA (440 Cases)

The signs of carcinoma for purposes of interpretation can be divided into primary and secondary. The primary signs concern the presence of either a mass and/or calcifications (Table IV).

SECONDARY SIGNS OF CARCINOMA (440 Cases)

Three of these signs having to do with retractive phenomena and skin thickening are associated with advanced disease and represent an extension of the carcinoma. A

* Presented at the Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P.Q., Canada, September 25–28, 1973.
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Table III

SIGNIFICANT GENERAL OBSERVATIONS

	No.	Per Cent
Occult carcinomas*	139	30.1
Occurred in remaining breast	68	14.7
Multiple foci in one breast	44	10
Bilateral simultaneous (no routine contralateral breast biopsy)	9	2
Associated with calcifications	244	50.9
Located deep within breast	26	5.9

* The term occult is to mean either there were no physical abnormalities, or if there were, they were unrelated to the carcinoma.

relatively low incidence of "signs of advanced carcinoma" is seen.

Unilateral ductal prominence, I don't believe, has received sufficient attention as a secondary sign of carcinoma. One can see that it occurs in 18 per cent of the cases. It is usually a segmental collection of prominent ducts which are more obvious on one side than in the similar area of the opposite breast. Unilateral ductal prominence was most frequent but also most subtle. Prominent duct pattern is caused by periductal collagenosis. This is a hyperplastic phenomenon involving connecting tissue deposited circumferentially around ducts. The ducts may or may not be normal, but are often normal both as to the size of the lumen and the epithelium (Fig. 1, *A* and *B*; Table v).

TUMOR SIZE

The measurement of the size of the cancer was made from the xeroradiograph of the breast. Two projections were considered in the measurement and that which was largest was recorded. As expected a greater percentage of small cancers are nonpalpable (59 per cent of those 1 cm. or less). One notices that there are 18 per cent of the carcinomas 2.6 cm. and over in the occult group. These represent carcinomas manifested by a calcific pattern alone with no masses. Typically these can be very large, up to 8 cm., and remain nonpalpable. Fortunately, the large size in these instances does not indicate necessarily advanced disease; in fact, very often the lymph nodes will be free of metastatic disease. Failure of palpability is due undoubtedly to the lack of a desmoplastic reaction (Table vi).

Table IV

PRIMARY SIGNS OF CARCINOMA

(440 cases)

	No.	Per Cent
MASS		
Spiculated	185	42
Nodular	105	24
Irregular density	57	13
Diffuse density	14	3
Circumscribed	9	2
Total	370	84
CALCIFICATIONS		
Only sign	70	16
Associated with mass	154	35
Total	224	51

AGE DISTRIBUTION

The ages of the patients with carcinoma of the breast were recorded as a group and then they were regarded as to whether or not the tumor represented an occult carcinoma or a nonoccult one.

It is interesting and important that carcinomas in young women are more likely to be occult as compared to all ages. This is probably a different opinion than that which has been held before. In women 31

Table V

SECONDARY SIGNS OF CARCINOMA

(440 cases)

	No.	Per Cent
Unilateral ductal prominence	79	18.0
Skin retraction	30	6.8
Nipple retraction	15	3.4
Skin thickening	26	5.9
Total	150	34.1

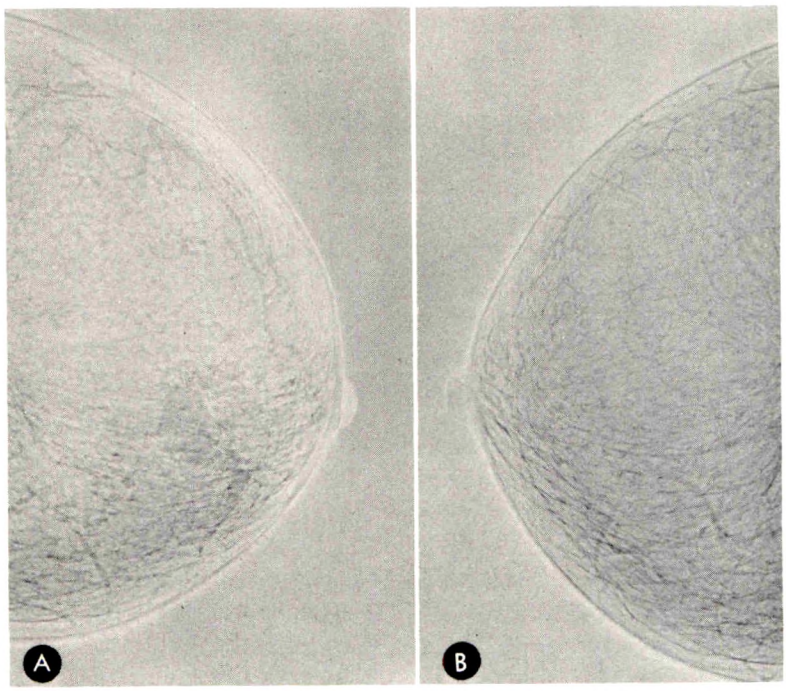


Fig. 1. (*A* and *B*) The unilateral duct prominence in the right breast would be sufficient for an impression of carcinoma even though the mass were not present. Note the asymmetry.

to 40, 43.2 per cent of 37 cancers were occult *versus* 30.1 per cent occult carcinomas in the entire group. Conversely, carcinomas in older women are more likely to be palpable than in their counterpart, the younger group. Only 18.7 per cent of 64 cases 71 years or older were occult *versus* 30.1 per cent for the entire group (Tables VII and VIII).

OCCULT CARCINOMAS

The most important consideration of radiographic examination of the breast is the discovery of a carcinoma which cannot be palpated either by the patient or her physician and typically these are smaller carcinomas with fewer metastases to regional lymph nodes. The over-all accuracy of mammography is important; however, relatively, the number of occult carcinomas is far more important (Table IX).

As can be seen in Table IX, one of the most important factors in the diagnosis of occult carcinomas is the occurrence of calcifications alone without a mass being present. This occurred in 37.4 per cent of

Table VI

TUMOR SIZE

Occult (139 cases)		Total (440 cases)			Nonoccult (301 cases)	
No.	Per Cent	Cm.	No.	Per Cent	No.	Per Cent
13	100.0	−0.5	13	2.9	0	0
73	54.8	0.6–1.0	133	30.2	60	45.1
31	21.4	1.1–1.5	145	32.9	114	78.6
8	16.0	1.6–2.0	50	11.3	42	84.0
2	6.1	2.1–2.5	33	7.5	31	93.9
12	18.2	2.6–	60	15.0	54	81.8

the occult lesions *versus* only 6.1 per cent in carcinomas which were palpable. These cancers can be very large and still remain nonpalpable. For example, there were 19 carcinomas in the entire group which measured 4 cm. or more in size. Of these, 7 were occult, and they were all manifested radiographically by a calcific pattern alone without evidence of a mass. Of the 12, 4 cm. or more in size which were palpable, only 2 were identified by the calcific pattern alone.

CARCINOMAS IN THE REMAINING BREAST

There was a significant number of carcinomas in the remaining breast (68 cases or 14.7 per cent of 462). This is an illustration of how frequently carcinoma occurs in the remaining breast, when one considers that that type of patient constitutes only about 8 per cent of the total mammographic examinations at Hutzel Hospital, and that in the total number of mammographic examinations there is an incidence of carcinoma in only about 1.7 per cent. These figures indicate that the number of 68 is completely out of proportion to the amount of patients examined.

LOCATION OF CARCINOMA

There was very little difference between whether the right or the left breast was involved, 50.6 per cent being in the right and 49.9 per cent in the left.

The location within the breast is essentially what one would expect with the knowledge of other studies. One notes that the large majority occurs in the upper axillary quadrant. This might be predicted because the upper axillary quadrant is the site of the greatest volume of active breast tissue for the longest period of time (Table x).

Of some importance is the fact that in 26 cases, or 5.9 per cent, the tumor was considered to be located "deep" within the breast; "deep" means very near the chest wall. This is important because as one develops techniques for examination of the breast, it is essential that the area near the chest wall be routinely imaged.

TABLE VII

AGE DISTRIBUTION

(440 cases)

Years	No.	Per Cent
−30	5	1.1
31–40	37	8.4
41–50	123	27.9
51–60	130	29.5
61–70	81	18.4
71–	64	14.5

TABLE VIII

AGE DISTRIBUTION RELATED TO OCCULT—NONOCCULT

Occult (139 cases)		Years	Nonoccult (301 cases)	
No.	Per Cent		No.	Per Cent
1	20.0	−30	4	80.0
16	43.2	31–40	21	56.8
49	39.8	41–50	74	60.2
36	27.6	51–60	94	72.4
25	30.9	61–70	56	69.1
12	18.7	71–	52	81.3

TABLE IX

BASIS FOR DIAGNOSIS OF OCCULT CARCINOMAS

	Total (440 cases)	
	No.	Per Cent
Mass without calcifications	216	49.1
Mass with calcifications	154	35.0
Calcifications alone	70	15.9
Occult (139 cases)		
Mass without calcifications	35	25.2
Mass with calcifications	52	37.4
Calcifications alone	52	37.4
Nonoccult (301 cases)		
Mass without calcifications	181	60.1
Mass with calcifications	102	33.8
Calcifications alone	18	6.1

TABLE X

LOCATION OF CARCINOMA

	No.	Per Cent
Upper axillary quadrant	259	58.9
Upper mammary quadrant	44	10.0
Lower axillary quadrant	45	10.2
Lower mammary quadrant	48	10.9
Subareolar	44	10.0

GENERAL APPEARANCE OF BREAST WITH CANCER

One of the most interesting aspects of the study was an assessment of the general appearance of the breast which has a cancer within it. From this information it was tried to derive a prototype which one could regard with a greater than average suspicion as indicative for carcinoma of the breast.

There were 3 considerations: breast size; coexistent mammary dysplasia; and coexistent prominent duct pattern or collagenosis (Table XI).

For purposes of comparison we examined the breasts of 456 women who did not have a carcinoma insofar as is known and observations were made on them.

It was noted that the small breast is more likely to be affected with cancer than either a medium or large sized breast. As a general rule, coexistent mammary dysplasia is not a prominent feature of breasts that have a carcinoma. There is a strong association between a prominent duct pattern and carcinoma.

Examples of these environmental changes associated with carcinoma can be seen in Figures 2, *A* and *B*, and 3, *A–C*.

Two additional points may be mentioned. One concerns carcinomas that were missed. There were 22 of these; this number is rather small and does not lend itself to a strong conclusion. All that one can say is that the small breast with excessive mammary dysplasia and moderate to severe prominent duct pattern is the type in which a carcinoma is most likely to escape detection by radiographic examination (small, dense breast). The other point is—and this is contrary to the general opinion—that there is not a strong dependence upon the age of the patient and misses may occur in any age group. The more important factor is that the breast is small and dense (Table XII).

DISCUSSION

This study demonstrates a rather high degree of accuracy attainable with xeroradiography. The examinations made at Hutzel Hospital are almost always of excellent quality; they are carried out by technologists who are dedicated to only that study. They have attained a high degree of skill with their specialization and interest in the work. It is believed that such skill can be obtained also by others, who are willing to work hard for a good technique and careful interpretation.

Images, even better than ours, insofar as accuracy of diagnosis and demonstration of occult carcinomas are concerned, have

TABLE XI

GENERAL APPEARANCE OF BREAST WITH CANCER

With Carcinoma (440 cases)			Without Carcinoma (456 cases)	
No.	Per Cent		No.	Per Cent
		BREAST SIZE		
193	43.9	Small	122	26.7
157	35.7	Medium	236	51.7
90	20.4	Large	98	21.5
		COEXISTENT MAMMARY DYSPLASIA		
217	61.6	None	171	37.4
70	13.6	Slight	127	27.8
83	18.9	Moderate	116	25.4
26	5.9	Severe	42	9.2
		PROMINENT DUCT PATTERN (Collagenosis)		
14	3.2	None	213	46.7
57	12.9	Slight	99	21.7
213	48.4	Moderate	96	21.5
156	35.5	Severe	48	10.5

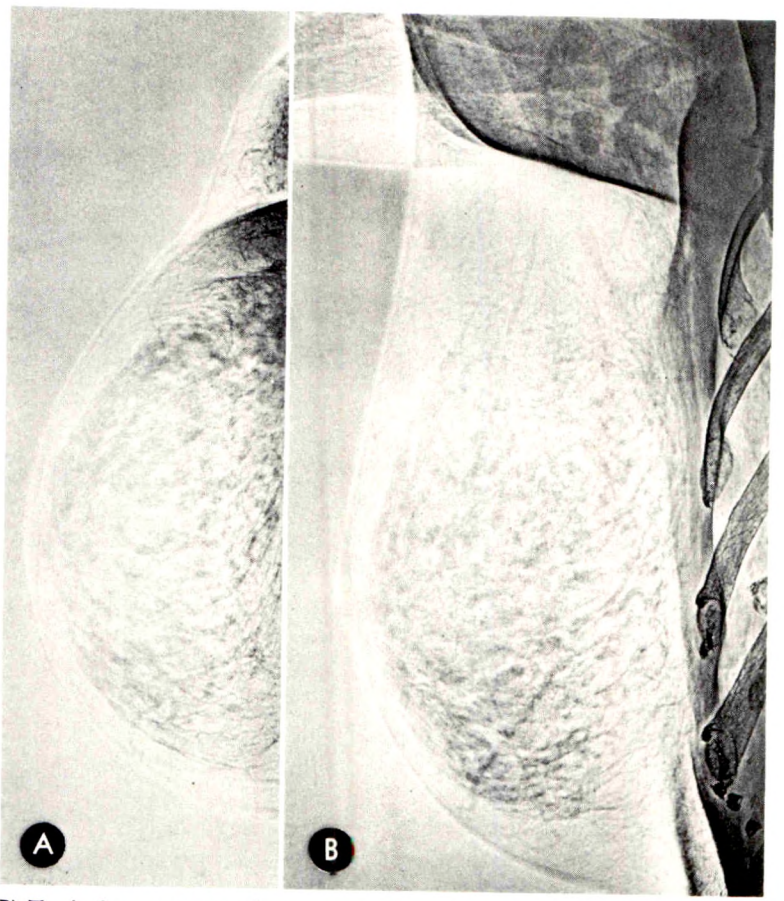


Fig. 2. (*A* and *B*) Typical appearance of severe prominent duct pattern. Note that it is a somewhat beaded pattern having some linearity to it. The multiple lymph nodes in the axilla represent lymph nodes involved with metastatic carcinoma, primary in the lung.

been obtained by Gloria Fraenkel[1] at Permanente Hospital in Los Angeles. In her series the follow-up was very closely done and included only one particular group of patients.

The ability to demonstrate small carcinomas in young women should be taken into consideration before excluding them from routine screening studies, although screening studies may be less frequent than those carried out for older women. It has been said that screening studies are of little or no value in the young population; this series would tend to make one disagree with that statement.

It is difficult to get accurate studies as to true positive and true negative rates in most practices, especially in one like that at Hutzel Hospital in Detroit. Accuracy studies are further complicated by the fact that it is difficult to draw the line when a carcinoma is missed. If the patient returns in a year and a half with a cancer of the breast and she has had previous studies, does that indicate an error in diagnosis on first study?

Also, it is difficult to get information especially today relative to the extent of disease. The modalities of operations for breast carcinoma are increasing and even within a single hospital multiple types of operations may be done depending upon the preference of the surgeon and the extent of disease. In addition, the way the operative specimen is studied by the pathologist for tumor size, multiplicity of

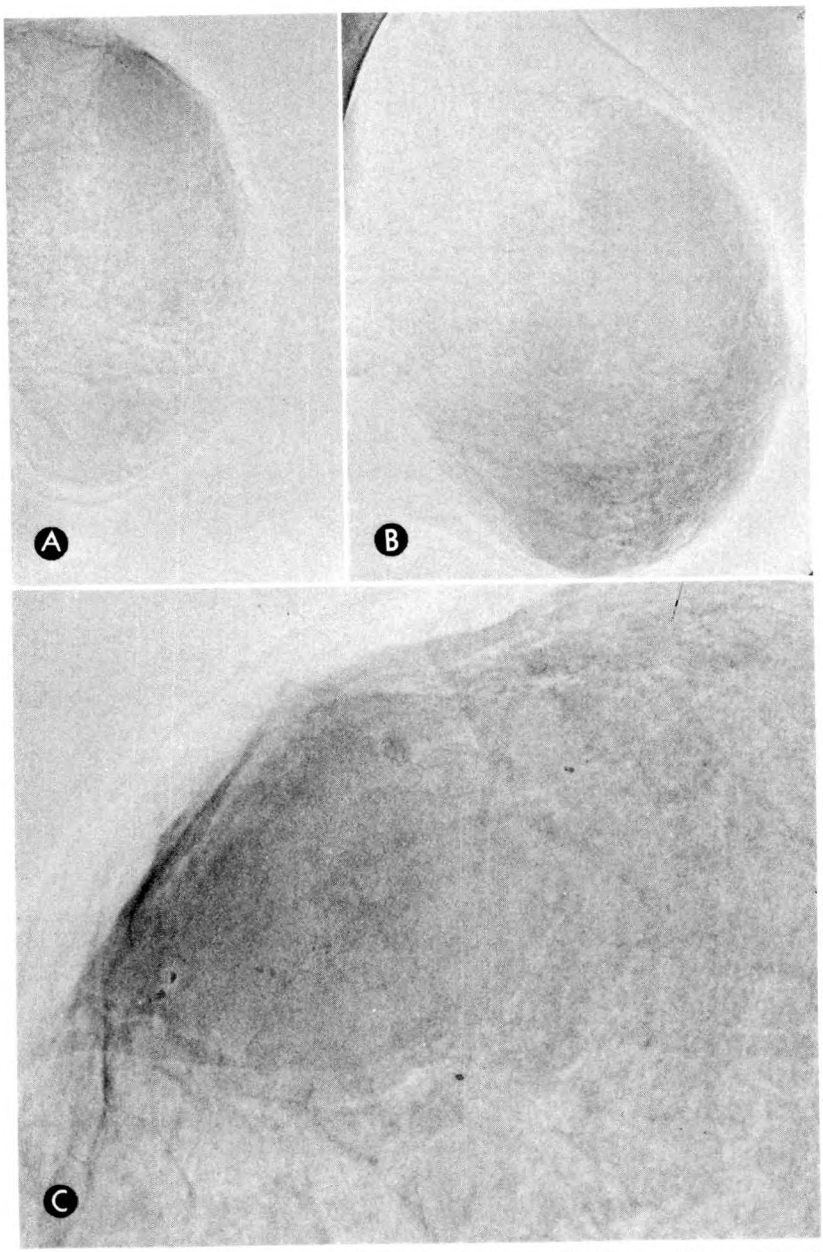


Fig. 3. (*A* and *B*) Extensive involvement of the breast with fibrocystic disease. Most of the density is due to connective tissue hyperplasia. (*C*) In the photographic enlargement the evidence of a carcinoma which was a lobular *in situ* one, is by virtue of the few calcifications visible.

tumors and metastatic disease to the axilla, varies greatly from institution to institution.

It would seem sufficient for evaluation of the efficiency of an examination if the following are recorded:

1. Tumor size as measured from the image;
2. Patient's age;
3. Presence or absence of calcifications; and
4. Presence or absence of demonstrable signs usually associated with advanced disease.

Frequent association of carcinoma of the breast with prominent duct patterns should

TABLE XII

MISSED CARCINOMAS

(22 cases)

AGE		
Years	No.	Per Cent
−30	1	4.5
31–40	2	9.1
41–50	7	31.8
51–60	6	27.2
61–70	4	18.2
71–	2	9.1

lead to further investigation. If a large number of patients with only this morphologic change within the breast and no evidence of carcinoma could be followed closely, it would be helpful to determine whether in this group there is an increased incidence of cancer of the breast, enabling one to have an additional high risk category of women for developing breast cancer.

CONCLUSION

The following statements can be made regarding a summary of this analysis of 462 women with breast cancer:

1. Small cancers are likely to be missed on physical examination.
2. Carcinomas manifested by calcific patterns routinely escape physical examination detection.
3. Mammography is very effective in discovering small carcinomas (1.0 cm. or less).
4. Carcinoma is almost always accompanied by a prominent duct pattern.
5. Carcinoma in the remaining breast lends itself to routine screening (30 per cent of cases occult).
6. Carcinoma in the remaining breast has a very high incidence (14.7 per cent of 462 cases, but constituting only 9 per cent of *all* mammographies performed, or an expected rate in all types of 1.7 per cent per 100 examinations).
7. Small breasted women aged 41 to 60, with a moderate to severe prominent duct pattern, are most likely to have breast cancer.
8. Errors are associated with small, dense breasts.
9. Occult carcinomas are more likely to be found in "young" women.
10. Carcinomas are more likely to be palpable in elderly women.

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REFERENCES

1. Fraenkel, G. Personal communication.
2. Martin, J. E. Xeromammography—improved diagnostic method: review of 250 biopsied cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 90–96.
3. Wolfe, J. N. Mammography: report on its use in women with breasts abnormal and normal on physical examination. *Radiology*, 1964, *83*, 244–254.
4. Wolfe, J. N. Xerography of breast. *Radiology*, 1968, *91*, 231–240.
5. Wolfe, J. N. Prominent duct pattern as an indicator of cancer risk. *Oncology*, 1969, *23*, 149–158.
6. Wolfe, J. N. Xeroradiography of the Breast. Charles C Thomas, Publisher, Springfield, Ill., 1972.

A NEW BIOPSY NEEDLE AND ITS CLINICAL USE*

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THE following design and operational features are desirable for a soft tissue biopsy needle:

1. It should be capable of providing an adequate specimen. For most types of tissue, a core of at least 1 mm. in diameter is necessary for paraffin block preparation.

2. In order to reduce injuries, the biopsy needle should be of the smallest possible diameter and should have very thin-walled tubings.

3. It should be simple in design and construction, and capable of being used with ease.

4. It should have the ability to sever the specimen and to retrieve it with minimal crushing.

5. It should permit aspiration during the operation in order to determine whether the needle tip is in solid tissue, a blood vessel, an abscess, a gas or fluid-containing cavity.

6. It should be disposable. After repeated use, the parts become dull or deformed.

7. It should provide visual confirmation of a specimen prior to withdrawal of the entire needle assembly.

8. It should permit multiple specimens to be obtained with each pass. It is reasonable to assume that multiple bites are safer and more representative than a single large bite.

9. It should permit hemostatic or additional anesthetic solution to be injected along the tract or at the biopsy site at any time, and should permit the instilling of contrast material.

10. It should be potentially applicable to various soft tissue sites or organs.

THE BIOPSY NEEDLE

DESIGN AND CONSTRUCTION

The needle assembly (Fig. 1) may be described as follows:

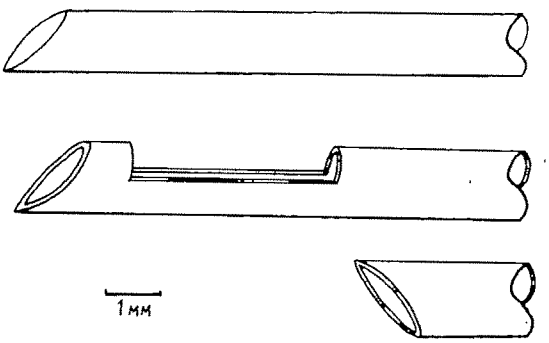


FIG. 1. Three parts of the biopsy needle.

1. It has a central pointed stainless steel stylet with a diameter of 1.02 mm. (0.040 inch).

2. It uses an inner stainless steel extra thin-walled spinal type of needle with a 1.27 mm. (0.050 inch) outside diameter (standard No. 18) and a 1.07 mm. (0.042 inch) inside diameter. A rectangular-shaped notch measuring 4 mm. long is ground near the beveled point.

3. It employs an outer pointed extra thin-walled stainless steel needle with an outside diameter of 1.47 mm. (0.058 inch, slightly smaller than standard No. 17) and an inside diameter of 1.32 mm. (0.052 inch).

When the unit is assembled for operation, the notch of the inner needle is exposed beyond the tip of the outer needle, with which it is aligned as shown in Figure 2.

Three lengths of the needle are available:* 7.6 cm. (3 inches), 12.7 cm. (5 inches), and 17.8 cm. (7 inches).

OPERATION

These technical steps are generally followed:

1. The puncture site is selected and marked. Fluoroscopy and pre-biopsy roent-

* Distributed by Unique Industries, P.O. Box 11413, Memphis, Tennessee 38111.

* From the Department of Radiology, Baptist Memorial Hospital, Memphis, Tennessee.

FIG. 8. (*A*) A huge retroperitoneal tumor producing anterior displacement of the inferior vena cava in a 31 year old woman. (*B*) Percutaneous biopsy needle position. Undifferentiated carcinoma was diagnosed by needle biopsy.

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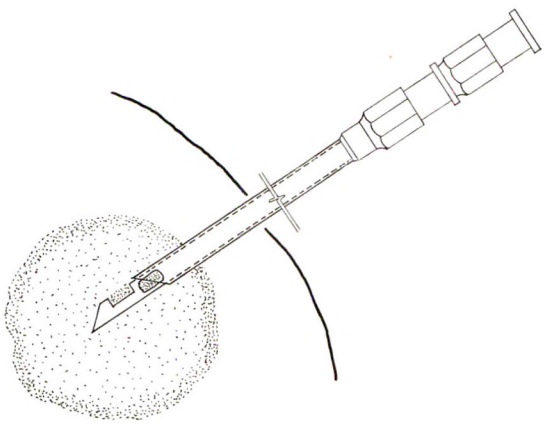


FIG. 6. The inner needle is then advanced completely.

perficial sites may be obtained by gradual withdrawal of the outer needle and by repeating the cutting operations (Fig. 5; 6; and 7).

7. Suction is maintained while the assembly is withdrawn so as to reduce the possibility of tumor cells or bacteria contaminating the tract.

Hemostatic or vasoconstrictive solution may be injected through either needle if brisk bleeding is observed through the outer needle, although the author has not had the occasion to use it in over 200 biopsies.

In the event that a cavity containing fluid or pus is encountered, and if it appears clinically indicated, radiographic contrast material may be introduced to outline its extent and shape, and to check for any filling defects or communication.

COMPARATIVE FEATURES

Several soft tissue biopsy needles that have been devised through the years appear partially deficient of the previously mentioned 10 features.[2–5,7,9–12,15,16] The design and operation of this needle satisfy substantially the criteria set forth.

HANDLING OF THE SPECIMEN

The major part of the specimen is sent in the capped syringe for histologic paraffin block preparation. In cases of suspected neoplasm, especially primary pulmonary carcinoma, Papanicolaou smear and stain of a small tissue fragment or of a drop of the centrifuged fluid often yield the diagnosis before the cell block preparation is ready. Appropriate bacteriologic examinations may be performed on a portion of the fluid.

CLINICAL APPLICATION

In the vast majority of instances, the tissue cores were sufficient for histologic cell block preparation.

A total of 224 percutaneous biopsies of the following lesions and organs have been performed: neoplastic and non-neoplastic pulmonary lesions; liver; hepatic tumors;

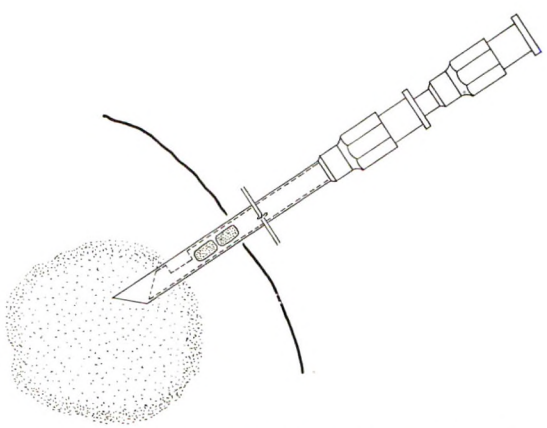
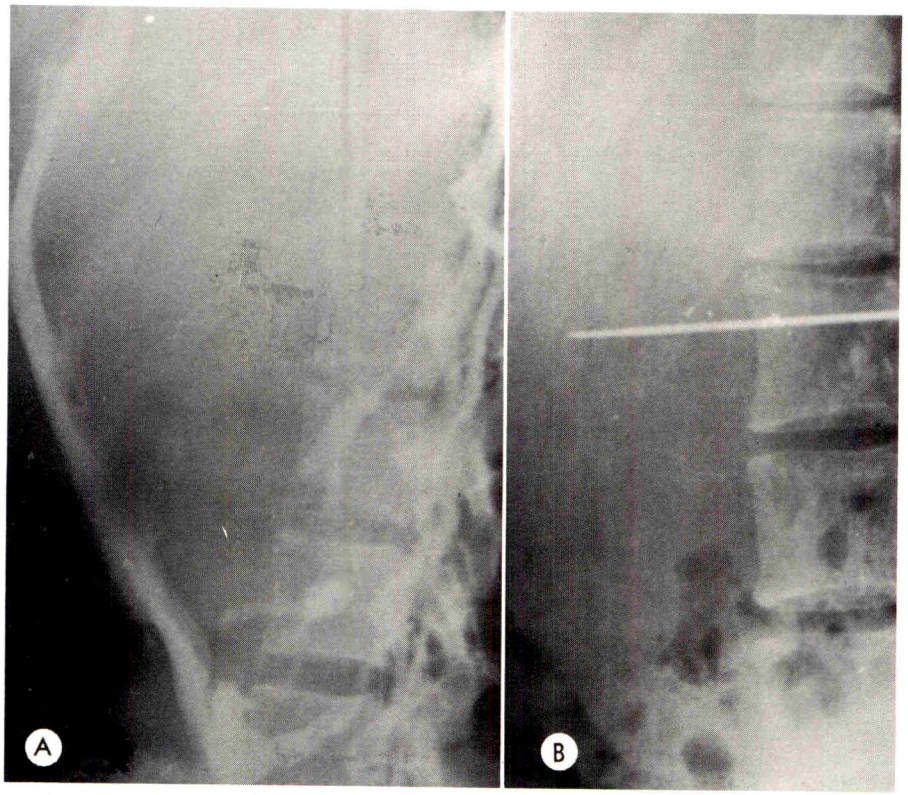
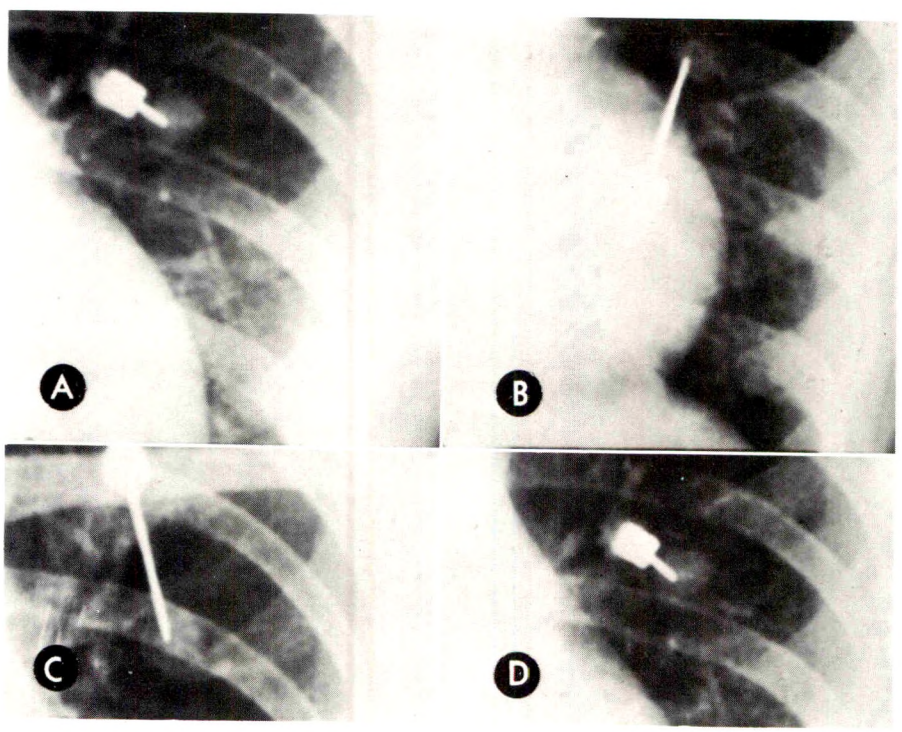


FIG. 7. As suction is applied to the withdrawing inner needle, another specimen is obtained.

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FIG. 9. (*A–D*) A solitary 1 cm. pulmonary nodule in the same patient shown in Figure 8. Angled fluoroscopy was used to ascertain the position of the biopsy needle tip without patient movement. Cell block revealed undifferentiated carcinoma.


A
B


A
B
C
D

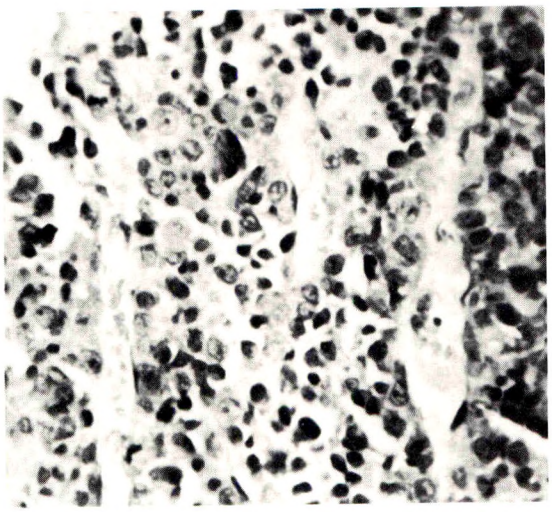


FIG. 10. Primary pulmonary carcinoma. Percutaneous needle biopsy. (H and E 400×.)

rib lesions; primary carcinomas of the breast; lytic lesions of the spine, pelvis, hips, and shoulder girdles; tumors of the extremities; renal cystic tumors; thyroid; palpable subcuteous masses and nodes; prostate; retroperitoneal tumors (Fig. 8, *A* and *B*); pancreatic carcinoma; pararectal masses; and adrenal tumor.

Accurate placement of the biopsy needle is the prime prerequisite, particularly for internal lesions. Figure 9, *A–D*, shows one of the techniques.

Among the 143 pulmonary biopsies, diagnoses of 102 neoplastic processes and 25 non-neoplastic processes were established—an accuracy of 89 per cent. A 15 per cent pneumothorax rate (16 small and 6 large) is comparable to or somewhat less than those of other reports.[1,4,6,8,13,14,16] Nine patients had mild hemoptysis of no consequence. Of the 81 nonpulmonary biopsies, 71 (88 per cent) were diagnostically accurate; and there was no clinically detectable complication. For many of these patients, surgery was spared. More detailed discussion and analysis will be communicated in subsequent reports.

By virtue of its small caliber and effectiveness, the needle presumably can be used for operative biopsy, *e.g.*, pancreatic, hepatic, and retroperitoneal masses. Other potential percutaneous biopsy sites under investigation are muscle, kidney, cervical masses, and lytic spinal bodies.

Adequacy of the specimen is illustrated in Figures 10 through 12.

SUMMARY

A new aspirating and severing soft tissue biopsy needle that is small, disposable, relatively simple (3 components), and

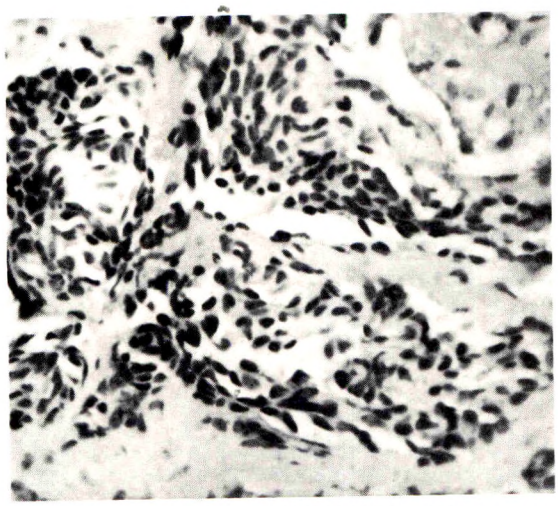


FIG. 11. Breast carcinoma. Percutaneous needle biopsy. (H and E 400×.)

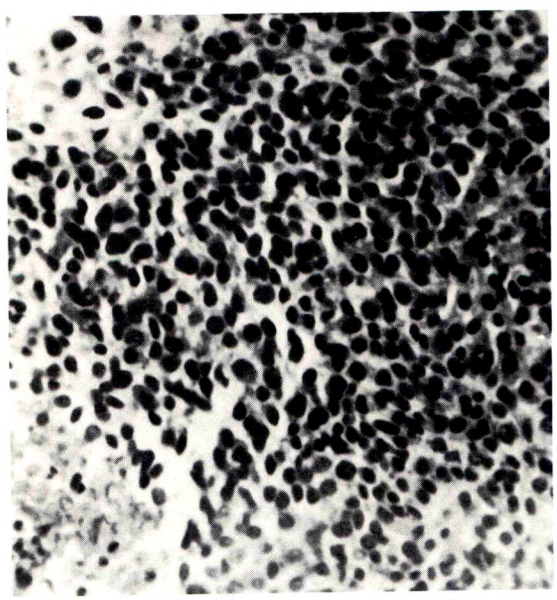


FIG. 12. Reticulum cell sarcoma, upper humerus. Percutaneous needle biopsy. (H and E 400×.)

very thin-walled has been used percutaneously for many lesions of the chest, abdomen, pelvis, extremities, subcutaneous masses, and lytic spinal bodies.

In the vast majority of instances, the tissue cores were sufficient for regular histologic cell block preparation. Among the 143 pulmonary biopsies, the diagnostic accuracy was 89 per cent.

There were 16 cases of post-biopsy small pneumothorax, 6 cases of large pneumothorax, and 9 incidences of mild hemoptysis of no consequence.

Of the 81 nonpulmonary biopsies, 71 (88 per cent) were diagnostically accurate; and there were no complications.

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REFERENCES

1. Adamson, J. S., and Bates, J. H. Percutaneous needle biopsy of lung. *A.M.A. Arch. Int. Med.*, 1967, *119*, 164–169.
2. Carpenter, R. L., and Lowell, J. R. Pleural biopsy and thoracentesis by new instrument. *Dis. Chest*, 1961, *40*, 182–186.
3. Cope, C. New pleural biopsy needle. *J.A.M.A.*, 1958, *176*, 1107–1108.
4. Dahlgren, S., and Nordenström, B. Transthoracic Needle Biopsy. Year Book Medical Publishers, Inc., Chicago, 1966.
5. De Bakey, M. E. New type of biopsy needle. *Surg., Gynec. & Obst.*, 1963, *116*, 755.
6. Fontana, R. S., Miller, W. E., Beaubout, J. W., Payne, W. S., and Harrison, E. G. Transthoracic needle aspiration of discrete pulmonary lesion. *M. Clin. North America*, 1970, *54*, 961–971.
7. Franseen, C. C. Aspiration biopsy with description of new type of needle. *New England J. Med.*, 1951, *224*, 1054–1058.
8. Krumholz, R. A., Manfredi, F., and Weg, J. G. Needle biopsy of lung. *Ann. Int. Med.*, 1966, *65*, 293–307.
9. McClure, J. N. New effective biopsy needle. *Surgery*, 1963, *54*, 747–748.
10. Menghini, G. Two-operator needle biopsy of liver. *Mod. Med.*, 1959, 71–72.
11. Parker, R. H., and Pearson, C. M. Simplified synovial biopsy needle. *Arth. & Rheumat.*, 1963, *6*, 172–176.
12. Silverman, J. A. New biopsy needle. *Am. J. Surg.*, 1938, *40*, 671–672.
13. Smith, W. G. Needle biopsy of lung. *Thorax*, 1964, *19*, 68–78.
14. Stevens, G. M., Weigen, J. F., and Lillington, G. A. Needle aspiration biopsy of localized pulmonary lesions with amplified fluoroscopic guidance. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 561–571.
15. Turner, A. F., and Sargent, E. N. Percutaneous pulmonary needle biopsy: improved needle for simple direct method of diagnosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 846–850.
16. Vitums, V. C. Percutaneous needle biopsy of lung with new disposable needle. *Chest*, 1972, *62*, 717–719.

THERMAL ENHANCEMENT OF THE RADIOSENSITIVITY USING CULTURED NORMAL AND NEOPLASTIC CELLS*

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FOR over a century it has repeatedly been claimed that hyperthermia may cause tumor regression, or even cure. The literature contains numerous reports of the arrest or total disappearance of malignant tumors after prolonged fevers. More recently, Cavaliere *et al.*[2] have reported a number of cures of human tumors treated with local application of heat. Overgaard and Overgaard[10] have provided an extensive literature review of clinical and experimental evidence indicating that tumor cells may be selectively killed by high temperature.

Cultures of normal tissue are relatively insensitive to heat, while malignant cells in culture are highly sensitive to thermal treatment. This difference in heat sensitivity was clearly demonstrated by Chen and Heidelberger[3] in experiments on transformed cells showing that the higher thermal sensitivity appears to be an integral part of the characteristics of the malignant cell. Levine and Robbins[9] have similarly shown a differential thermal sensitivity of normal and malignant cells in culture. They suggested that the differential thermal sensitivity of normal and malignant cells might be a result of varying growth states of the cell populations; *i.e.*, the contact inhibited state *vs.* the non-contact inhibited state. More recently, Westra and Dewey[14] and Palzer and Heidelberger[12] have shown that the thermal sensitivity markedly varied within the division cycle of Chinese hamster cells and HeLa cells. Furthermore, the thermal sensitivity could be significantly modified by treating cells with various metabolic inhibitors.

The present study was undertaken to elucidate cellular mechanisms involved in the thermal enhancement of radiosensitivity of human tumor cells, using *in vitro* cultures of both malignant and normal cells.

MATERIAL AND METHOD

Experiments were carried out with human and mouse cell lines, derived from both malignant and normal tissues. All the cultures were grown and maintained on Eagle's minimum essential medium supplemented with 10 per cent fetal calf serum.

Both plateau and log phase cultures of HeLa cells were used to simulate some of the behavior of *in vivo* solid tumors. 3T3 mouse fibroblasts, whose progression through the cell cycle is highly sensitive to cell population density, were used as experimental models of normal tissue. The details of cell growth at the different phases have been described.[7,12] Briefly summarized, the plateau phase of HeLa and 3T3 cell growth was obtained by inoculating 5×10^5 and 2.0×10^5 cells per plate (60 mm. Falcon Petri dish) and incubating them for 5 days at 37°C. in a humidified 5 per cent CO_2 air atmosphere, respectively. At this confluent stage, the maximum cell densities for 3T3 cells and HeLa cells are usually 2.5×10^5 cells/cm.^{2} and 6×10^4 cells/cm.^{2}, respectively (Fig. 1). For cultures of HeLa or 3T3 cells in exponential growth, the procedure was similar except that the initial inoculum was lowered by a factor of 10. In order to obtain quantitative dose survival curves of both log and plateau phase cultures, the colony-forming ability

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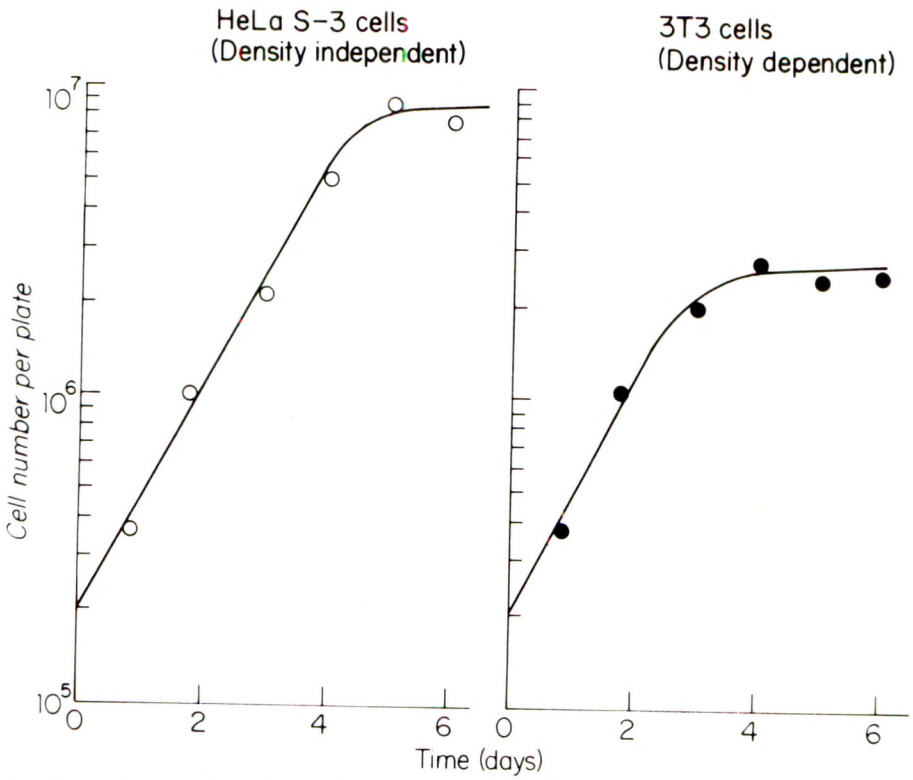


FIG. 1. Growth curves of HeLa S-3 cells and 3T3 mouse fibroblasts. Equal number of cells (2×10^5 cells per plate) were inoculated and the number of cells per plate enumerated. The culture medium was not changed after plating.

of plated single cells was determined throughout the study, as described in detail elsewhere.[5] Irradiation procedures were carried out at a room temperature using a Picker machine at 280 kvp., 20 ma., a half value layer of 0.5 mm. Cu, and a dose rate of 162 rad/min.

RESULTS AND DISCUSSION

Figure 2 shows a thermal sensitivity of HeLa cells and 3T3 fibroblasts in an exponential growth phase. Both cell lines were exposed to 42°C. It is quite evident that the neoplastic cell line, HeLa cells, exhibited significantly higher thermal sensitivity as compared to the density dependent cell line, 3T3 cells. The differential thermal sensitivity of 2 cell lines seems less pronounced, when the cell lines are exposed to hyperthermia (42°C.) in a confluent phase of their growth (Fig. 3). The less differential thermal sensitivity of HeLa cells

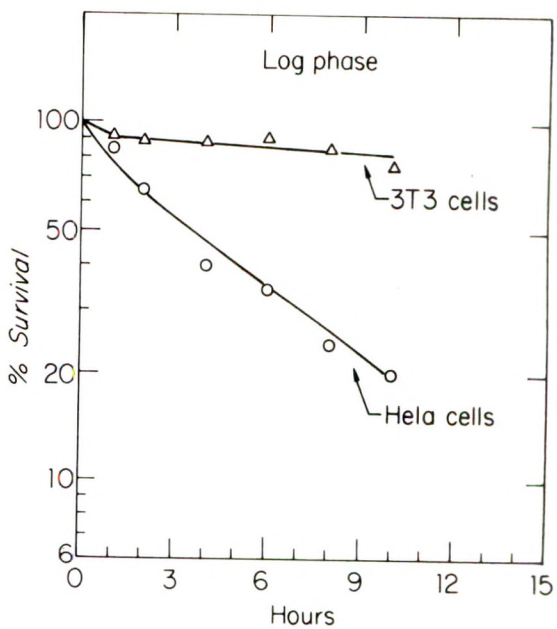


FIG. 2. Per cent survival of HeLa S-3 cells and 3T3 cells in the log phase as a function of incubation time at 42°C.

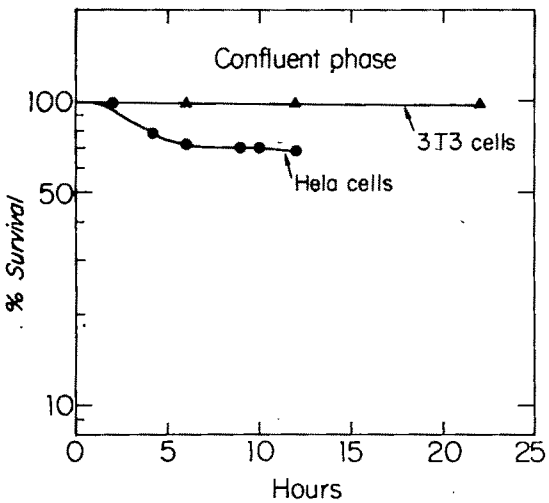


FIG. 3. Per cent survival of HeLa cells and 3T3 cells in the confluent phase as a function of incubation time at 42°C.

and 3T3 cells in the confluent stage may be explained on the basis of a population distribution within the division cycle of the cells. It has been shown by others[12] that cells in the pre-DNA synthetic phase are less heat sensitive than those in the late S phase. Cells in a confluent phase are thought to be mostly in a pre-DNA synthetic phase.

Before proceeding with a dose survival study with irradiation and heat treatment, an experiment was carried out to determine a temporal relationship of the thermal enhancement of radiosensitivity in HeLa cell culture. Figure 4 shows that a 2 hour exposure of cells to 41.5°C. significantly reduces the cell survival either immediately before or after irradiation. Figure 5 shows the survival curves of irradiated HeLa cells exposed to varying degrees of hyperthermia. As the temperature was increased, cells exhibited more sensitivity to irradiation; on the other hand, when the irradiated 3T3 cells was exposed to 42°C. for 3 hours, they showed a slight enhancement of the radiosensitivity (Fig. 6).

The mechanism of the thermal enhancement of radiosensitivity is not clearly understood. Ben-Hur *et al.*[1] showed that at least recovery from sublethal damage of

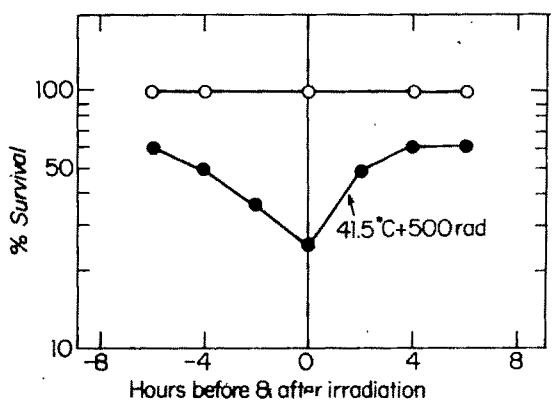


FIG. 4. Per cent survival of HeLa cells treated to 41.5°C. for 2 hour pulse exposure before or after a single dose of 500 rads. The open circles represent the normalized survival at 500 rads alone. The points at the zero or 4 hour time scale represent a 2 hour pulse exposure of cells to 41.5°C. before or after irradiation at the indicated time.

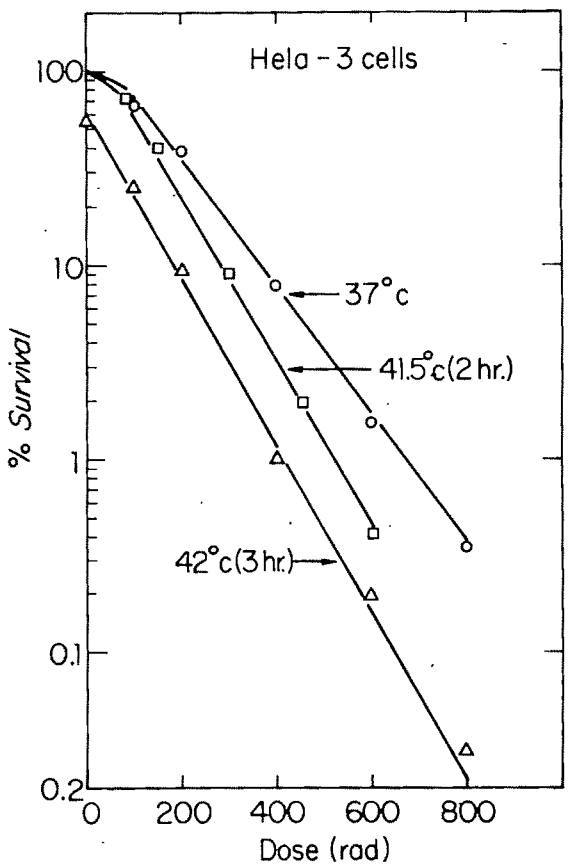


FIG. 5. Survival curves of irradiated HeLa cells exposed to varying temperatures. Log phase cells were irradiated.

radiation was impaired when the radiation was given at a low dose rate. It is difficult from our present data to suggest that the enhancement of the thermal radiosensitivity is due to a reduction in the sublethal repair process or to an increased intrinsic radiosensitivity of cells. A 2 dose split experiment might yield useful information. It has also been shown that radiosensitivity varies not only with cells at different growth phases, *i.e.*, exponential and plateau phases, but also with cells at different stages within the division cycle. It is also noteworthy that thermal sensitivity markedly varies within the division cycle. Experiments with synchronously dividing culture would be helpful in elucidating cellular mechanisms of thermal enhancement of radiosensitivity.

Combined use of hyperthermia with radiation therapy was studied by Crile,[4] who showed an enhanced effect as measured by tumor regression following local hyperthermia and irradiation. More recent studies by us and others of x-irradiation and heat using animal tumor systems have shown that a significant enhancement of the radiosensitivity of the tumor was obtained.[6,11] In our animal study with mice bearing a Ridgway osteogenic sarcoma, 96 per cent (71/74) animals were cured following a combined treatment of irradiation and hyperthermia, as compared to 44 per cent (8/18) with irradiation and 0 per cent (0/10) with heat alone. Furthermore, the cure rate following combined therapy appeared independent of tumor size. *In vitro* work, currently in progress, suggests that hypoxic tumor cells, usually the most radioresistant cells, are rendered more sensitive than oxygenated cells after a short exposure to hyperthermia.[8] This finding and also a recent work by Robinson *et al.*[13] would be of considerable clinical significance since one would be able to overcome a highly radioresistant hypoxic human tumor cell by a combined treatment of heat and irradiation. The demonstration of a differential thermal enhancement of the radiosensitivity of normal and malignant cells could lead to improved local control of irradiated tumors. This would be of particular value in the management of those human tumors that have been resistant to conventional fractionated irradiation regimens, and which kill by local growth.

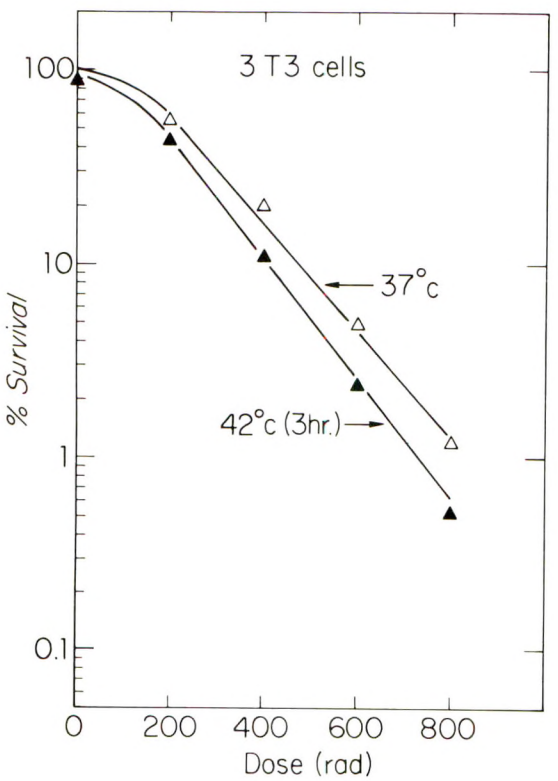


Fig. 6. Survival curves of 3T3 cells exposed to 42°C. for 3 hours immediately after irradiation. Log phase cells were irradiated.

SUMMARY

Cultures of normal tissue are relatively insensitive to heat, while malignant cells in culture are highly sensitive to thermal treatment.

This differential thermal sensitivity is most pronounced when the cells of normal and neoplastic tissues are exposed to heat in their exponential growth phase.

Dose survival curves of HeLa and 3T3 cells with and without postirradiation thermal treatment showed that the neoplastic HeLa line exhibited significant enhancement of radiosensitivity when the

irradiated cells were exposed to elevated temperatures (42°C. for 3 hours). Similar heat treatment of irradiated 3T3 fibroblasts did not appreciably enhance the radiation response.

The potential significance of these findings for clinical radiotherapy is discussed.

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REFERENCES

1. BEN-HUR, E., BRONK, V., and ELKIND, M. M. Thermally enhanced radiosensitivity of cultured Chinese hamster cells. *Nature*, 1972, *238*, 209–210.
2. CAVALIERE, R., CIOCATTO, E. C., GIOVANELLA, B. C., HEIDELBERGER, C., JOHNSON, R. O., MARGOTTINI, M., MONDOVI, B., MORICCA, G., and ROSSI-FANELLI, A. Selective heat sensitivity of cancer cells. *Cancer*, 1967, *20*, 1351–1381.
3. CHEN, T. T., and HEIDELBERGER, C. Quantitative studies on malignant transformation of mouse prostate cells by carinogenic hydrocarbons in vitro. *Internat. J. Cancer*, 1969, *4*, 166–178.
4. CRILE, G., JR. Effects of heat and radiation on cancers implanted on feet of mice. *Cancer Res.*, 1963, *23*, 372–380.
5. DJORDJEVIC, B., and KIM, J. H. Modification of radiation response in synchronized HeLa cells by metabolic inhibitors: effects of inhibitors of DNA and protein synthesis. *Rad. Res.*, 1969, *37*, 435–450.
6. HAHN, E., ALIERE, A., and KIM, J. H. Effect of heat in combination with radiation therapy on Ridgway osteogenic sarcoma in vivo. Radiol. Soc. North America, (abst.), 1973.
7. KIM, J. H., KIM, S H., PEREZ, A. G., and FRIED, J. Radiosensitivity of confluent density: inhibited cells. *Radiology*, 1973, *106*, 447–449.
8. KIM, S. H., KIM, J. H., and HAHN, E. Thermal enhancement of radiosensitivity of hypoxic tumor cells. To be published.
9. LEVINE, E. M., and ROBBINS, E. B. Differential temperature sensitivity of normal and cancer cells in culture. *J. Cell. & Comp. Physiol.*, 1970, *76*, 373–380.
10. OVERGAARD, K., and OVERGAARD, J. Investigations on possibility of thermic tumor therapy I. *Europ. J. Cancer*, 1972, *8*, 65–78.
11. OVERGAARD, K., and OVERGAARD, J. Investigations on possibility of thermic tumor therapy II. Action of combined heat-roentgen treatment on transplanted mouse mammary carcinoma. *Europ. J. Cancer*, 1972, *8*, 573–575.
12. PALZER, R. J., and HEIDELBERGER, C. Studies on quantitative biology of hyperthermic killing of HeLa cells. *Cancer Res.*, 1973, *33*, 415–427.
13. ROBINSON, J. E., WIZENBERG, M. J., and MCCREEDY, W. A. Radiation and hyperthermia: mouse bone marrow studies. Presented at the 15th Annual Meeting of the American Society of Therapeutic Radiologists, October, 1973, (abst.).
14. WESTRA, A., and DEWEY, W. C. Variation in sensitivity to heat shock during cell cycle of Chinese hamster cells in vitro. *Internat. J. Rad. Biol.*, 1971, *19*, 467–477.

Vol. 121, No. 4

COMPUTER GENERATED ISODOSE CURVES FOR HIGH ENERGY X-RAY MACHINES*

EXPERIMENTAL VERIFICATION AND TIME-SAVING STRATEGIES

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THE dose distribution in a radiation therapy treatment plan is calculated at the Hospital of the University of Pennsylvania using the method developed by Cunningham.[2] The technique consists of evaluating separately the primary and scattered dose components at each point of dose calculation, inside or outside the irradiated volume. This method requires knowledge of the tissue-air ratio (TAR) for zero field size and the scatter-air ratio at various depths and portal sizes. We have obtained appropriate data for the 2 MV Van de Graaff generator and 6 MV linear accelerator in our department from measurements of central axis depth dose in a water phantom for various portal sizes. In addition, information on the beam profile in air, inside and outside the field, is required to calculate dose distributions.

BEAM PROFILES

The radiation intensity drops very rapidly at the edge of the beam for high energy x-ray machines. For focal-spot sizes encountered with these sources, the edge of the beam is defined primarily, we believe, by the attenuation of the x rays in the collimator. Figure 1 shows the geometric relationship between t, the thickness of the collimator material through which the beam passes for the 2 MV machine, and x, the distance from the central ray.

When $[x/(\mathrm{SSD}+d)]^2 \ll 1$, it can be shown that

$$t \cong \mathrm{SCD}(x - x_0)/x. \qquad (1)$$

For a 30×30 cm. field at an SSD of 100 cm., the error in t is 1 per cent.

The geometric penumbra, G, is defined as

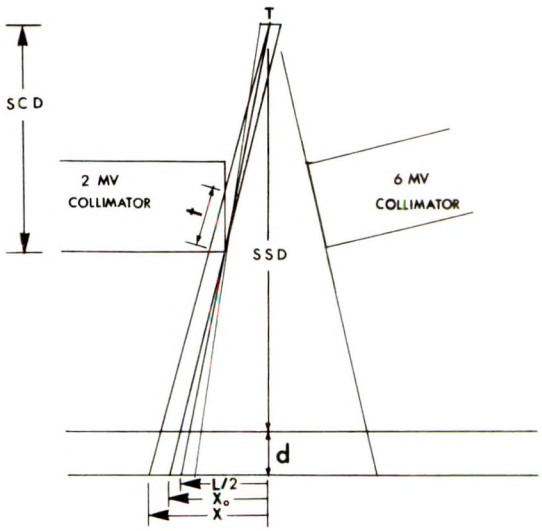


FIG. 1. Geometry used to calculate the attenuation of the beam through the collimator jaw edge. To adjust the portal size on the 2 MV Van de Graaff machine, the collimator jaws are moved linearly. The portal size on the 6 MV linear accelerator is adjusted by moving the collimator jaws on an arc so that the beam edge penetrates the total thickness of collimator material.

$$G = T(\mathrm{SSD} + d - \mathrm{SCD})/\mathrm{SCD}, \qquad (2)$$

where T is the effective target size.

Outside the field, $x > x_0$, the intensity of the primary beam in air, $P(x)$, is assumed to be represented by an exponential function:

$$P(x) = P(x_0) \exp(-Bt), \qquad (3)$$

where the attenuation constant, B, must be determined from the measured beam profile, and $P(x_0)$ is the relative primary intensity of the x-ray beam at the very edge of the field.

Within the field, $x < x_0$, the primary beam intensity, $P(x)$, can be represented

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TABLE I

PARAMETERS DESCRIBING THE BEAM PROFILE IN AIR FOR THE 2 MV AND 6 MV X-RAY MACHINES AT THE HOSPITAL OF THE UNIVERSITY OF PENNSYLVANIA

Therapy Unit	T	$x<x_0$	$x>x_0$
2 MV	2.5 mm.	$A=0.009$ cm.$^{-2}$	$B=0.49$ cm.$^{-1}$
6 MV	0.5 mm.	$A=0.000$ cm.$^{-2}$	$B=0.65$ cm.$^{-1}$ For $x>1.15x_0$ $B=0.066$ cm.$^{-1}$ For $x<1.15x_0$

by a Gaussian function:

$$P(x) = P(0) \exp(-Ax^2), \qquad (4)$$

where $P(0)$ is the intensity along the central ray and A is determined from the measured variation of the primary beam intensity within the field.

Table 1 shows the value of the effective target size, T, and the constants A and B for the two units that were determined by curve fitting measured beam profiles in air for various field sizes. Data were obtained for the beam profiles in air by recording the radiation-induced change in current of a silicon diode[1]* operated in the reverse bias mode as it transversed the beam. The effective target size obtained from beam profile measurements may not correspond to the actual target size, since it is a mathematically fitted parameter. The variation in the beam intensity within the field is very small for both units. To describe mathematically the radiation intensity at the edge of the field for the 6 MV unit required two values for B. The first value is used for points falling within 15 per cent of the width of the field. Note that the experimentally determined value, B, is significantly higher for the 6 MV unit. This occurs because the radial design of the collimator allows the very edge of the beam to pass through the full thickness of collimator material. This results in a reduction in the penumbra due to edge transmission. Figure 2 shows calculated and measured beam profiles in air.

Because of the sharp gradient at the field edge, the spatial resolution of the detector is of great importance. Figure 3 compares the measured beam profile of the 6 MV unit using a 0.6 cm.[3] Farmer ionization chamber with a lucite cap of equilibrium thickness and a silicon diode without equilibrium cap. The spatial resolution of the Farmer chamber with the equilibrium cap is not sufficient to describe the edge of the beam accurately.

The lateral dose distribution at any depth in a water phantom can be calculated taking into account the primary beam profile. Figure 4 demonstrates excellent agreement between calculated and measured dose distribution at a 10 cm. depth in a water phantom for the 2 therapy units. Comparison of Figure 3 and Figure 4 shows that, because of scattered radiation, the edges of the field are not as sharp in the water phantom as in air.

The excellent agreement between measured and calculated profiles for the water phantom indicates that the method of calculating the dose to a point by separating the primary and scattered components is accurate. To follow the rapid change in dose at the edge of the field, however, it was necessary to choose a grid for calculation whose lateral point spacing was very small (0.125 cm.).

CALCULATION OF ISODOSE CURVES

For computer calculation of isodose curves, the depth interval for dose calculation is less critical than the lateral grid spacing since the relatively slow rate of change of dose with depth allows accurate interpolation with a depth grid spacing as large as 1 cm.

To investigate the effect of varying the lateral grid spacing, isodose curves were calculated using successively coarser lateral grids.

Figure 5 shows calculated isodose curves

* Micro Semiconductor Corporation, Model MC-170. Dimensions approximately 0.05 × 0.48 × 0.66 mm.

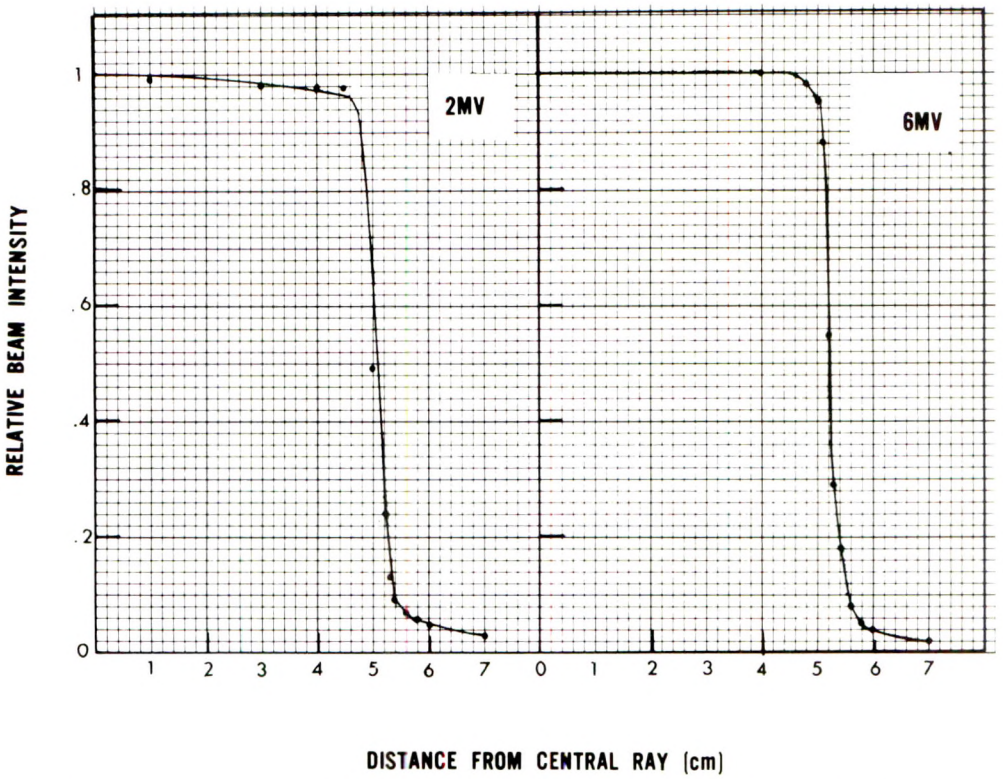


FIG. 2. Profile of relative beam intensity in air at 100 cm. target-detector distance for 2 MV and 6 MV x-ray beams. Solid lines are calculated profiles using the parameters given in Table 1. Dots represent measured values.

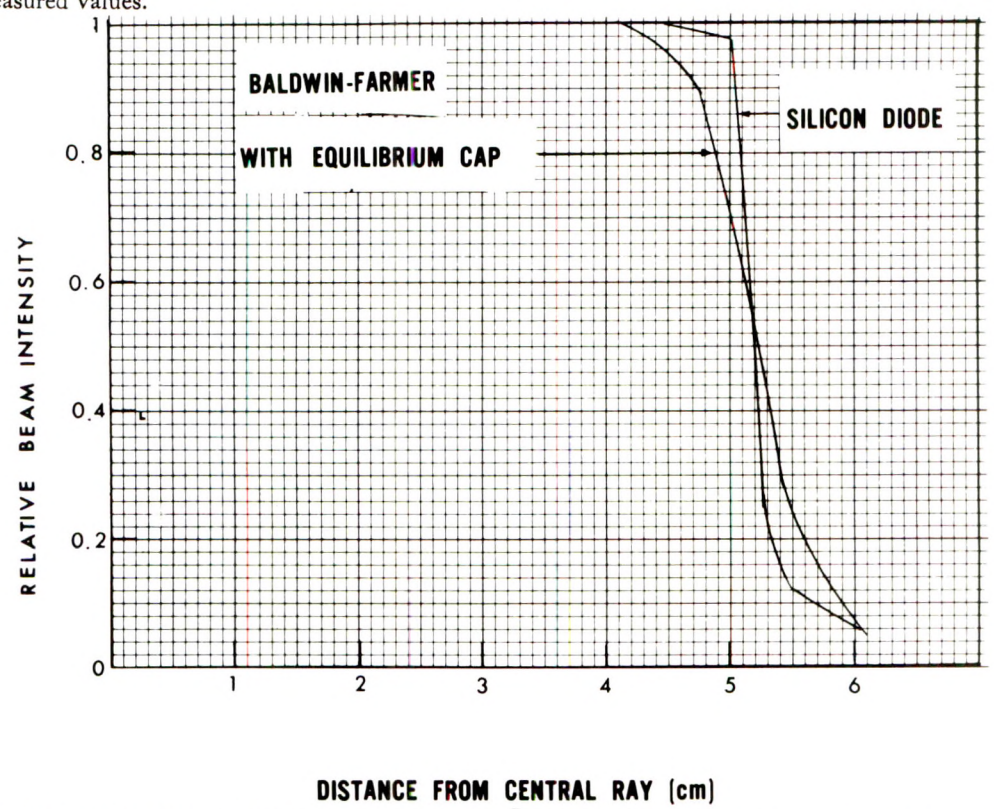


FIG. 3. Measured profile of 6 MV beam in air using 2 detectors. A small detector must be used to measure accurately the rapid change in beam intensity near the field edge for a high energy x-ray machine.

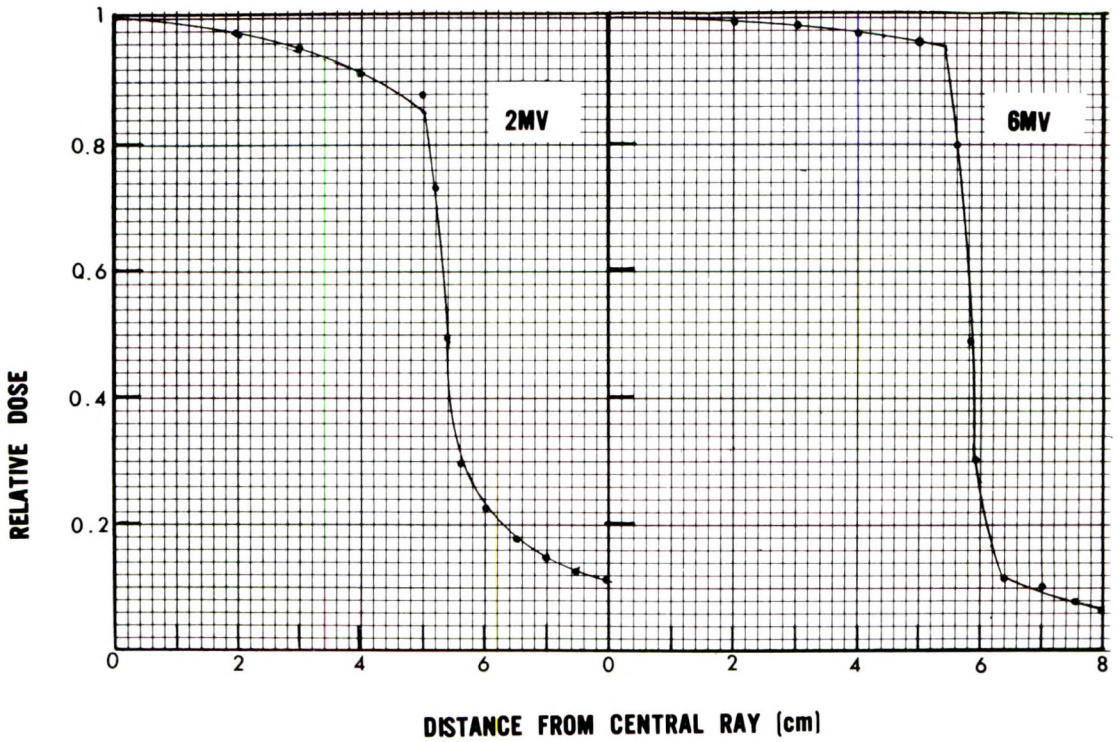


FIG. 4. Comparison of calculated and measured dose across 2 MV and 6 MV fields at a depth of 10 cm. for 5×5 cm. fields at 100 cm. SSD. Solid lines calculated; dots measured.

for the 2 MV beam for a 5×5 cm. field at 100 cm. SAD, using 4 different calculation intervals: 0.125 cm., 0.25 cm., 0.5 cm., and 1 cm. There is no significant change between the curves calculated with 0.125 cm. and 0.25 cm. intervals. The use of a 0.5 cm. interval, however, clearly distorts the pattern. For this reason, a 0.25 cm. lateral grid interval was chosen for all subsequent calculations. Similar calculations of isodose curves for the 6 MV beam (Fig. 6) agree with this conclusion.

Calculations of the dose distribution for half of a 20×20 cm. field for the 2 MV unit were performed.* As the lateral grid interval was varied, the central processing unit (CPU) times required for the calculations were noted. The results are shown in Table II. As expected, the CPU time increased dramatically as the calculation interval decreased. The results show that calculation with the 0.25 cm. interval necessary for accurate delineation of the edge of the field would be time-consuming and expensive.

* The calculations were done on the PDP-10 computer in the Medical School of the University of Pennsylvania using time-sharing mode. The graphic display was obtained on a Tektronix Graphic Computer Terminal Model 4002A, with Hard Copy Unit Model 4601, located in the Radiotherapy Section of the Radiology Department.

REDUCTION OF COMPUTING TIME

The scatter dose component at Q_1 (Fig. 7) at depth d is obtained by summing the scatter contributions from annular areas in 10° sectors along R and then summing over 360°. The number of calculations

TABLE II

PROCESSING TIMES TO GENERATE 2 MV ISODOSE PATTERNS FOR ½ OF 20 × 20 CM. FIELD

Calculation interval (cm.)	1	0.5	0.25	0.125
CPU Time (sec.)	14	39	119	445

CPU = Central processing unit.

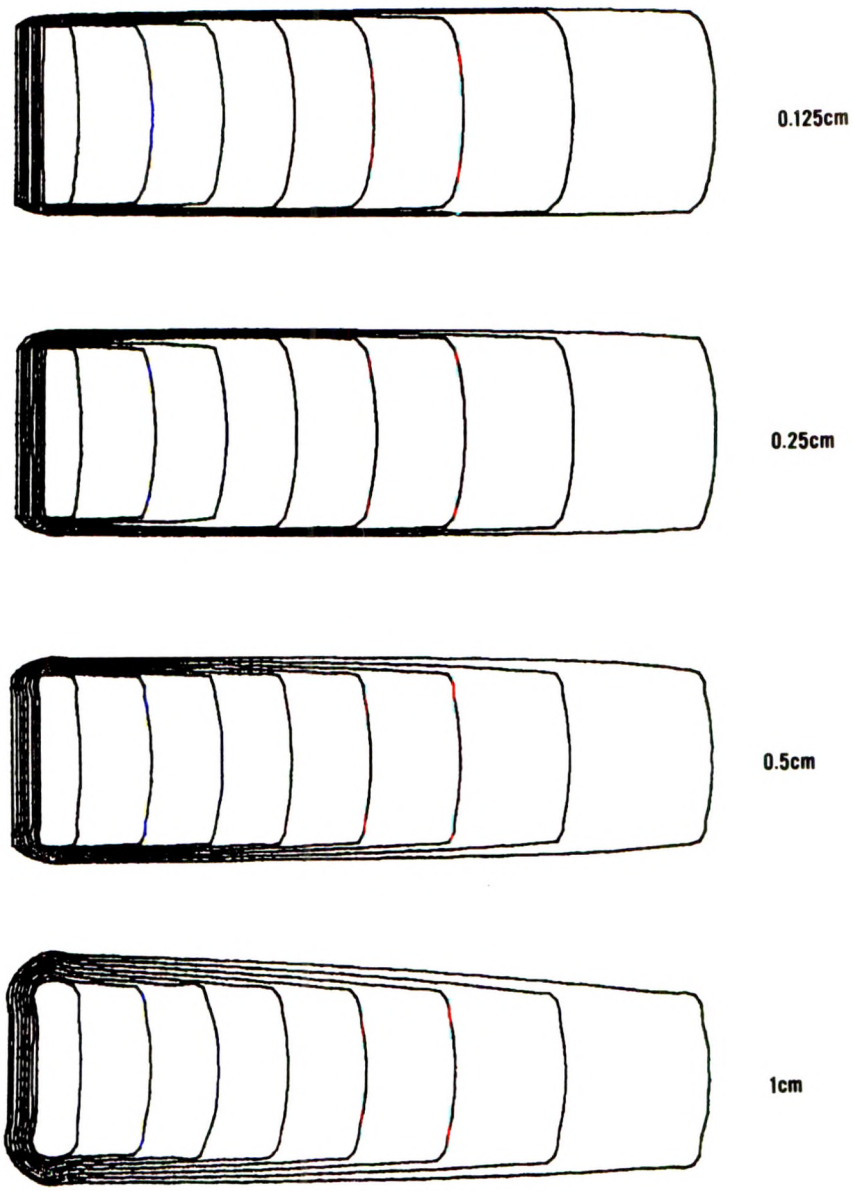


Fig. 5. Isodose patterns obtained on a 2 MV beam 5×5 cm. field and 100 cm. SAD, when the distance between points of lateral dose calculation is changed from 0.125 cm. to 1 cm. The depth increment does not affect the shape of the isodose curve and was 1 cm. for all calculations. The 0.25 cm. lateral grid results in a satisfactory description of the dose distribution.

necessary to obtain the dose distribution is very large for large fields. One way to reduce this number is to increase the radial width, W, of the annular area.

After a minor change in Cunningham's BEAM computer program, the dose distribution for a 5×5 cm. field was calculated for the 6 MV unit using a 0.25 cm. lateral grid for dose point calculations and annular widths of 1, 0.5, and 0.25 cm. The isodose curves obtained are shown in Figure 8. The figure demonstrates that the 1 cm. wide annular sector introduces artifacts in the isodose curves. There is little difference between the curves calculated with 0.25 or 0.5 cm. annular widths. The calcu-

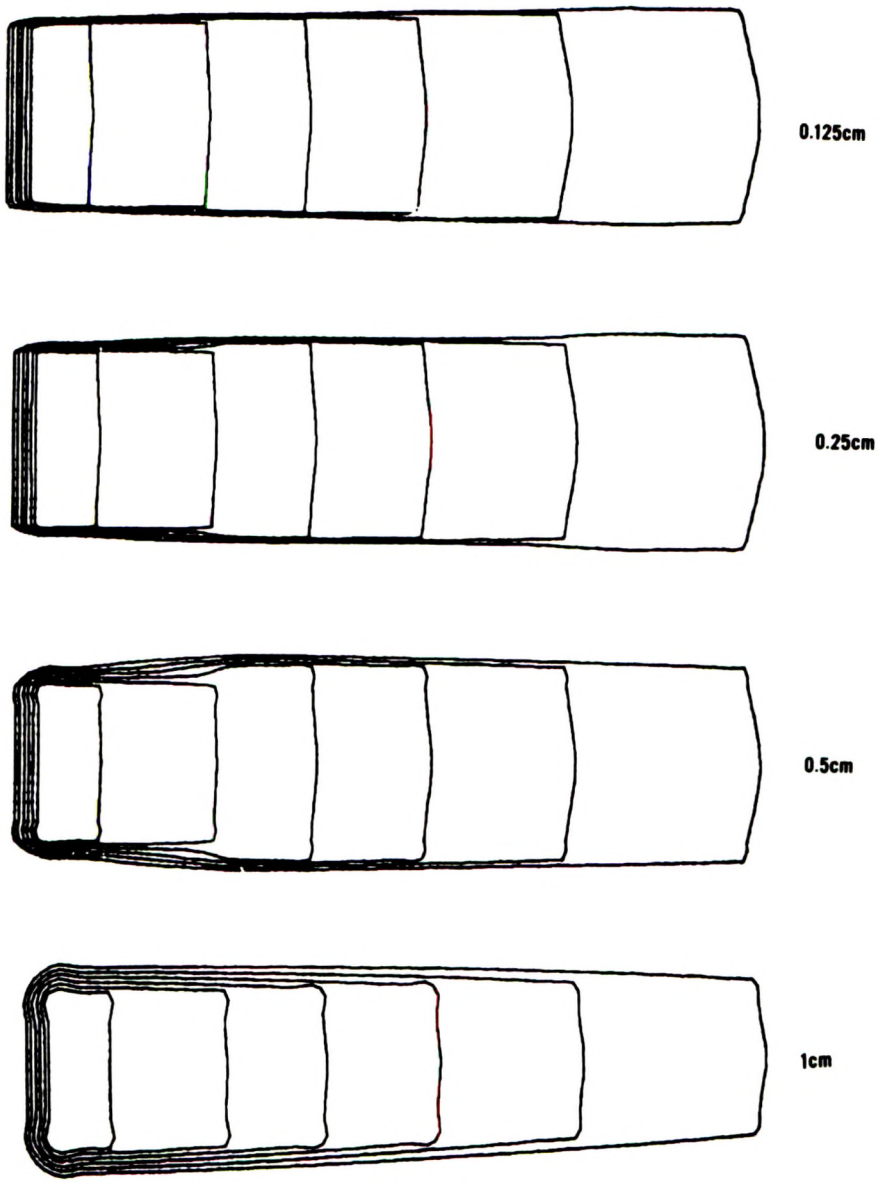


Fig. 6. As Figure 5 for a 6 MV beam.

lation time for the 0.5 cm. wide scatter sector was approximately half the time required for the 0.25 cm. wide sector.

Further reduction in computing time can be expected by calculating the dose on a relatively large lateral grid within the field, where the dose varies slowly, and on a finer grid near the field edge, where the dose is changing rapidly. To achieve this objective the computer program was modified to calculate doses on a 1 cm. lateral grid within the field and to within 1 cm. of the beam edge. The separation of the points of dose calculation was then changed to 0.25 cm. for points nearer the edge. In existing computer plotting programs all dose points must be spaced equidistantly. To accomplish this, points within the field between the 1 cm. dose calculation points were "filled in" at 0.25 cm. intervals using linear

interpolation. Figure 9 shows the calculated isodose curve for a 5×5 cm. field of the 6 MV unit calculated by this method. Comparison of the curves shown in Figure 8, B and C with Figure 9 shows that they are nearly identical. The times required to produce the three isodose curves, however, are dramatically different.

The CPU times necessary to calculate half-field isodose patterns were compared for four field sizes using the three calculation methods. The results are shown in Table III. As expected, the differences in computer time are particularly dramatic for larger fields.

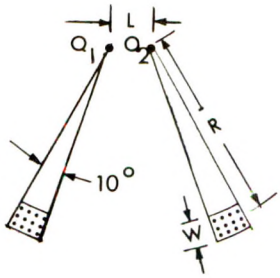


FIG. 7. Figure illustrating the method of calculating the effect of scattered radiation to the dose at two points. L is the distance between points of dose calculation. W is the width of the area from which scatter at distance R from the point of dose calculation Q_2 is calculated.

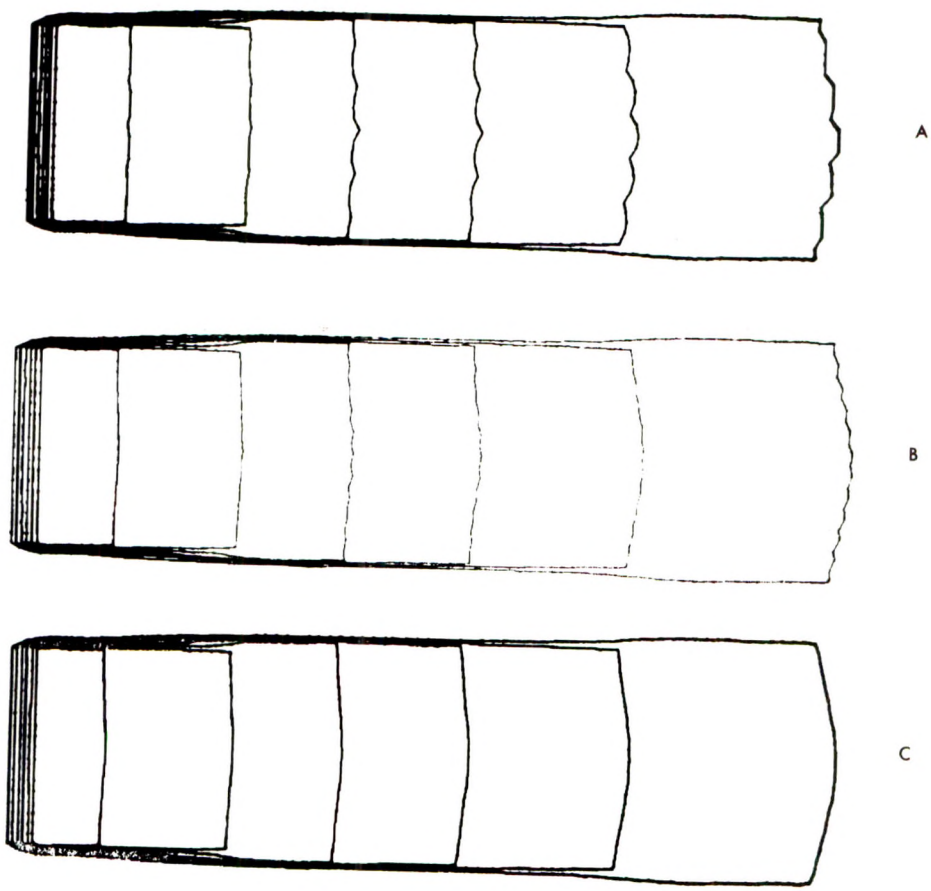


FIG. 8. Isodose curves calculated by the following methods of incrementing points of dose calculation. Lateral grid interval 0.25 cm. Scatter sector width: A, 1 cm.; B, 0.5 cm.; C, 0.25 cm.

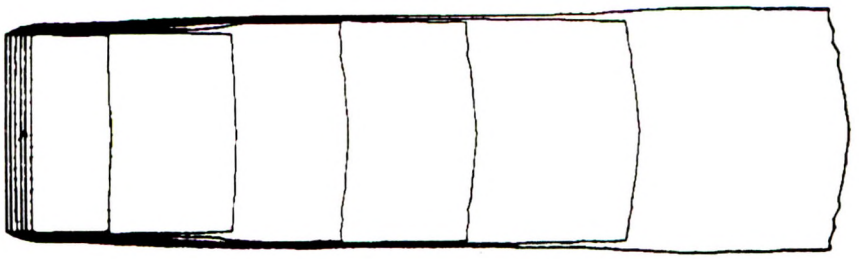


FIG. 9. Dose calculated with a 1 cm. lateral grid of points for dose calculation within the portal field and with a 0.25 cm. grid when the point of dose calculation is within 1 cm. of the field edge. Scatter sector width, 0.5 cm.

SUMMARY

Calculation of isodose patterns for high energy therapy units using Cunningham's BEAM program requires accurate information on the beam profile in air. This may be obtained by scanning across the x-ray beam using a very small silicon diode detector. Data obtained on a particular unit may not be applicable to any other unit.

Computer calculations of isodose curves for high energy x-ray machines must use a 0.25 cm. or smaller lateral grid of dose calculation points to ensure accurate reproduction of the sharp fall-off of the dose at the beam edge. Computer calculation time to obtain the isodose curves using this size lateral grid is prohibitively long. The computing time can be appreciably reduced by: (1) changing the width of the annular sectors used in calculating the contribution of scattered radiation to the dose to 0.5 cm.; and (2) calculating the dose at 0.25 cm. lateral intervals within 1 cm. of the beam edge, but changing the spacing of the grid to 1 cm. inside the field. These programming strategies do not produce any significant change in the isodose curves but lead to a significant reduction in computer time.

TABLE III

COMPARISON OF CPU TIMES IN SECONDS TO GENERATE HALF-FIELD ISODOSE PATTERNS FOR THE 2 MV VAN DE GRAAFF MACHINE

Field Size (cm.)	5×5	10×10	15×15	20×20
Calculation Method				
A	34	61	92	119
B	17	33	46	65
C	17	20	31	30

Method A: Dose calculated on 0.25 cm. grid, scatter sector thickness 0.25 cm.
Method B: Dose calculated on 0.25 cm. grid; scatter sector width 0.50 cm.
Method C: Dose calculated on 1 cm. grid within field; near the field edge (within 1 cm.) the dose is calculated at 0.25 cm. intervals, scatter sector width 0.50 cm.
CPU = Central processing unit.

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REFERENCES

1. BAILY, N. A., and KRAMER, G. Lithium-drifted silicon p-i-n junction as an x-ray and gamma-ray dosimeter. *Radiat. Res.*, 1964, *22*, 53–80.
2. CUNNINGHAM, J. R., Scatter-air ratios. *Phys. Med. & Biol.*, 1972, *17*, 42–51.

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DESIGN OF X-RAY TARGETS FOR HIGH ENERGY LINEAR ACCELERATORS IN RADIOTHERAPY*

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IN a recent paper Rawlinson and Johns[6] showed that a 25 MeV linear accelerator (Varian Clinac-35) produced an inferior beam of photons to a 25 MeV betatron (Allis Chalmers). In fact, from a depth dose point of view, the linac's beam was equivalent to the beam produced by the betatron when it was operated at 16 MeV. The reasons for this were attributed to differences in the design of the target and the flattening filter in the 2 machines. The betatron employs a thin tungsten target and an aluminum flattening filter, while the linac uses a thick tungsten target and a tungsten flattening filter.

The purpose of this paper is to present the experimental results of our further investigation of this problem and to suggest some methods for improving the x-ray beams from linacs used in radiotherapy. In the present experiments the 25 MeV electron beam from the linear accelerator was used to produce x-rays in targets of different atomic numbers. The x-ray beams were flattened with flattening filters made of various materials. Using these targets and filters, depth doses and x-ray yields were measured both along the central axis of the beam and at different angles to the central axis.

We have found that by using a *thick aluminum* target and an *aluminum flattening filter* in the linear accelerator we can obtain the same depth dose distribution as from the Allis Chalmers betatron operating at the same energy. Furthermore, the x-ray yield in the forward direction is the same within a few per cent, whether we use a thick aluminum or thick lead target. It thus appears that thick targets in linear accelerators should be made of low atomic number materials. For such targets a more penetrating beam is produced and the yield in the forward direction is as good as from a high atomic number material.

EXPERIMENTAL APPARATUS AND PROCEDURES

Experiments reported in this paper have been carried out at the Ontario Cancer Institute in Toronto using the electron beam from a Varian Clinac-35 linear accelerator. The schematic diagram of the experimental apparatus is shown in Figure 1. As normally used in radiotherapy, the electron beam is bent first through 57°, next focused by 2 quadrupole lenses, and then bent 90° to hit a tungsten target where it produces an x-ray beam which emerges at port No. 3. This port was not used in our experiments because of the difficulties in removing the linac head (collimators, transmission ion chamber, etc.) to obtain an unobstructed electron beam. Instead, in our studies we turned off the 90° bending magnet allowing the electron beam to emerge at port No. 2 through a thin beryllium window. The extracted electron beam was then made to produce x-rays in our own targets which were secured to the linac in the position shown in the diagram. With the 90° bending magnet turned off, the electron beam was focused onto the target into a circle of ~3 mm diameter. The experimental apparatus will be discussed in more detail in the following sections.

TARGETS

The targets were cylinders of 5 cm diameter positioned in air 3 cm from the beryllium exit window. Their thicknesses

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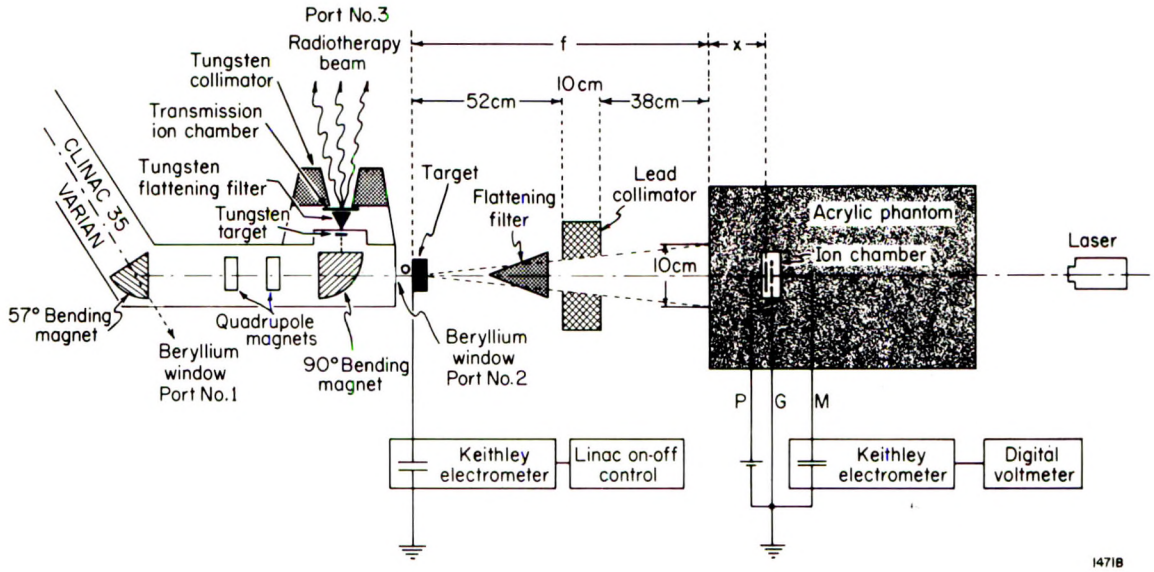


FIG. 1. Schematic diagram of the complete experimental apparatus.

were chosen to be equal to the mean range of 25 MeV electrons in the target material. This ensured a complete stopping of the incident electrons with minimum attenuation of the x-ray beam produced in the target. Using range values tabulated by Berger and Seltzer[1] the target thicknesses were 0.9 cm and 5.2 cm for lead and aluminum, respectively.

FLATTENING FILTERS

The flattening filters were made of lead and aluminum. They were conically shaped with a base diameter of 12 cm and a height of 3.4 cm and 25 cm for the lead and aluminum filters, respectively. The filters produced a beam which was uniform to within ±10 per cent over a field of diameter 40 cm at a phantom depth of 10 cm and a source to chamber distance of 100 cm. The filter was positioned on the beam axis and arranged to move along the axis on a V block (not shown in the Figure) thus enabling us to introduce more or less of the filter material into the x-ray beam. The beam flatness could be quickly checked using our matrix dosemeter[4] placed behind the flattening filter and the position of the filter then moved slightly to give the optimum beam.

COLLIMATORS

The collimator used for depth dose measurements along the central axis was made of lead with a tapered hole at the center designed to give a 10 cm diameter circular field at 100 cm from the target. For the depth dose measurements at an angle to the central axis, the collimator was designed to give a 3 cm diameter circular field at 125 cm from the target. For these latter measurements the trolley carrying the various components was rotated about point O shown in Figure 1.

PHANTOM

The phantom was made of acrylic slabs with a density of 1.18 g/cm^3 and an area 12×12 inches. Into one of these slabs a cavity was milled to house the special ion chamber. To change the depth in the phantom, slabs were placed in front of the chamber and the phantom moved away from the target so as to keep the source to surface distance (SSD) equal to 100 cm.

DETECTOR

A cylindrical polystyrene end window ion chamber was built with a sensitive volume of 2 cm diameter and 1.2 mm depth. The polarizing electrode, P, was made of

50 microns aluminized mylar and connected to a 400 volts power supply. A thin coat of aquadag covering a circle (2 cm diameter) on the bottom surface of the chamber served as the measuring electrode, M. The charge collected here due to radiation was measured by a Keithley amplifier whose output was fed into a digital voltmeter, as shown in Figure 1. The guard ring, G, which was electrically insulated from the measuring electrode, and grounded, was a 5 mm wide aquadag ring painted around the measuring electrode leaving a gap of about 0.5 mm. The chamber leakage current was less than 10^{-12} A, while the charge collected on the measuring electrode after a typical exposure of 1 second was on the order of 10^{-9} C which shows that the chamber leakage currents were not a serious problem. The collection efficiency of the chamber was measured to be better than 99 per cent under the conditions used in the experiments.

MONITOR

The linac was arranged so that it was turned off after a given exposure. To do this the target was electrically insulated from ground and the charge delivered to it during the exposure was measured by a Keithley electrometer, whose output voltage (0–3 volts) was compared with a preset voltage by an operational amplifier. The positive output of the amplifier was used to trigger a relay which turned the linac beam off, when the charge collected on the target reached the preset value. Exposures delivered this way were reproducible to better than 1 per cent. At 80 pulses per second, a pulse width of 1.8 μsec and a pulse height of 40 mA, the charge collected on the target was about 6×10^{-6} C/sec.

ALIGNMENT OF THE COMPONENTS

The flattening filter, collimator and phantom were mounted on a trolley which could be placed in a fixed position with respect to the linac. One of the experimental problems that was encountered in our measurements was the alignment of the apparatus. It is virtually impossible to obtain good beam flatness data without making sure, ahead of time, that the electron beam axis coincides with the axis of the apparatus; *i.e.*, with the central axes of the flattening filter, the collimator and phantom. The procedure was as follows: (1) 2 pieces of radiation sensitive paper,* one 2 cm and the other 10 cm from the beryllium window, were exposed to the electron beam. The discolored spots produced by the radiation on the papers defined the electron beam axis. (2) A laser, located about 2 m from the window, was oriented so that its light beam was directed onto the beryllium window through the centers of the 2 spots on the radiation sensitive paper. In this way, the laser beam was made to coincide with the electron beam axis and further alignment procedure for the rest of the components was easy.

DEPTH DOSE MEASUREMENTS

Depth doses were measured in the acrylic phantom from the surface to a depth of ~25 cm. After each exposure the charge collected on the ion chamber measuring electrode was determined. Readings were obtained with both positive and negative polarities on the chamber ($\pm$400 V) and the results averaged. Since published depth doses are generally obtained using water phantoms, a correction was made to transform our acrylic depth dose measurements into doses in water. The transformation involves both an electron density correction and an inverse square law correction. If x is the depth in the acrylic phantom as shown in Figure 1 and $D_a(x)$ is the dose measured in acrylic at x, one obtains the dose in water D_w at a depth in water $x' = \{\rho_a/\rho_w\}x$ using the following expression:

$$D_w(x') = D_a(x)\left\{\frac{f+x}{f+x'}\right\}^2, \qquad (1)$$

* Available from Vickers Ltd., Radiation and Nuclear Engineering Division, South Merston Works, P.O.B. 8, Swindon, Wilts, U.K.

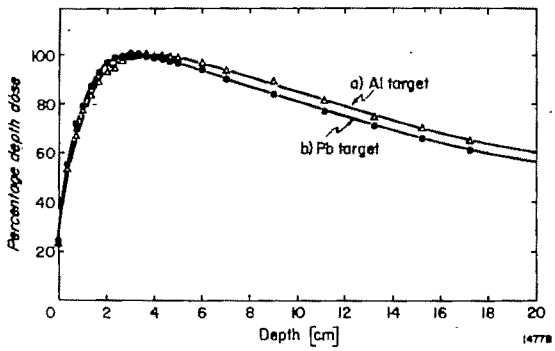


FIG. 2. Central axis percentage depth dose curves in water of unflattened x-ray beams produced by 25 MeV electrons impinging on (a) aluminum thick target, and (b) lead thick target. Circular field size with 10 cm diameter at a target to surface distance of 100 cm.

where f is the source to phantom surface distance and $\{\rho_a/\rho_w\}$ is the ratio of the electron densities in acrylic and water. For these 2 materials this is also close to the ratio of actual densities.

EXPERIMENTAL RESULTS

THICK X-RAY TARGETS

In this section we discuss the differences in x-ray beams produced by various targets, using *no* flattening filter. Figure 2 shows depth dose curves obtained from aluminum and lead targets at a source to phantom distance of 100 cm and a field size of 10 cm diameter at the phantom surface. A comparison between the 2 curves in Figure 2 shows that the aluminum target gives a more penetrating beam than the lead target, its surface dose is slightly lower and the depth of the maximum dose is larger (4 cm *vs.* 3 cm). A measurement of the depth dose curve of x-rays produced by a copper target (not shown in Figure 2) revealed that it lay between the curves for lead and aluminum. While the curves of Figure 2 are of no practical value, because the x-ray beam is unflattened, they do show that the beam from the aluminum target is more penetrating than the beam produced in lead.

The central axis x-ray yield of different targets was measured as a function of the target atomic number Z. The results are shown in Figure 3 where, for a given charge delivered to the target, the actual ion chamber current is plotted *versus* depth. It is evident that the aluminum target gives the highest yield, although the differences between the peak currents from aluminum, copper and lead targets are small. This result is surprising since one usually assumes that the yield increases with Z.

The findings summarized in Figures 2 and 3 suggest that low Z material should be used as the target in radiotherapy linacs. The beams so produced are more energetic than those from high Z targets and the useful x-ray outputs are just as high.

FLATTENING FILTERS

All high energy x-ray machines used in radiotherapy employ flattening filters to produce a uniform depth dose distribution over the maximum field size used. In this section are presented the results of an investigation into the characteristics of the flattened x-ray beams as a function of the target and filter material.

Depth doses were measured for the flattened beams with a circular field size of 10 cm diameter at the target to surface distance of 100 cm using the experimental arrangement shown in Figure 1. We found, as predicted by Rawlinson and Johns,[6] that a high Z flattening filter material

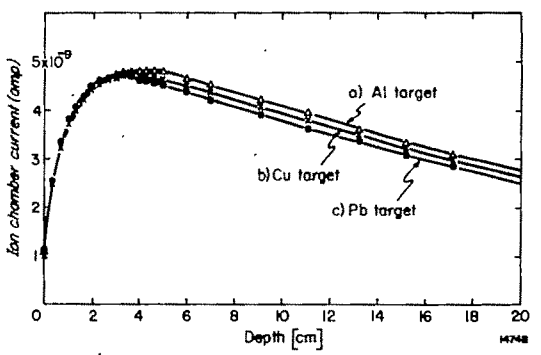


FIG. 3. Ion chamber current as a function of the depth in water for unflattened x-ray beams produced by 25 MeV electrons impinging on (a) aluminum thick target, (b) copper thick target, and (c) lead thick target. Field size 10 cm diameter at a target surface distance of 100 cm.

(lead in our experiments) softens a beam, while a low Z flattening filter material (aluminum in our experiments) hardens it. The most penetrating x-ray beam was obtained with an aluminum target/aluminum flattening filter combination, while a lead target/lead flattening filter combination rendered the least penetrating beam. The depth dose curves for these 2 extreme cases are shown in Figure 4. The lead target/lead flattening filter combination gives a depth dose curve which is almost identical to the one produced by the Clinac-35 beam which is used in radiotherapy. This is not unexpected since the Clinac-35 employs a tungsten target/tungsten flattening filter combination in the radiotherapy x-ray mode and the atomic numbers of tungsten and lead are similar.

In Figure 5, our experimental central axis depth dose curve obtained with the aluminum target/aluminum flattening filter combination is compared with the central axis depth dose curve from the Allis Chalmers 25 MeV betatron. The 2 curves are almost identical. This shows that from a depth dose point of view a considerable improvement of the linac x-ray beams is achieved with a thick, low Z target/low Z flattening filter combination as compared to the high Z target/high Z flattening filter combination generally used.

A summary of our central axis depth

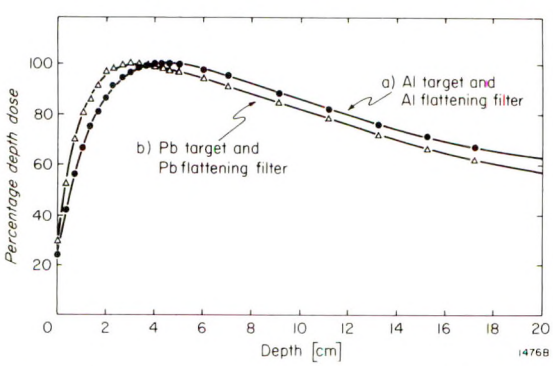


Fig. 4. Central axis depth dose curves of flattened x-ray beams in water produced by 25 MeV electrons impinging on a thick target: (a) aluminum target and aluminum flattening filter; and (b) lead target and lead flattening filter. Field size 10 cm diameter at a target to surface distance of 100 cm.

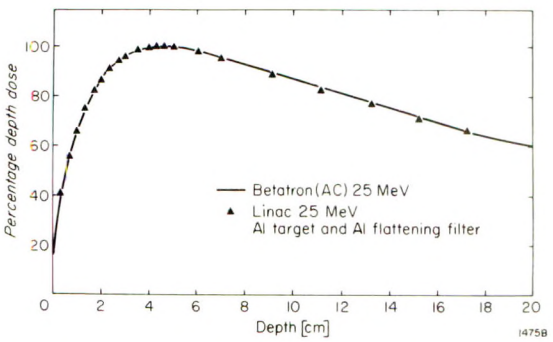


Fig. 5. Percentage depth dose curve from an Allis Chalmers 25 MeV betatron (solid line) compared to 25 MeV linac data obtained using an aluminum target and an aluminum flattening filter (▲).

dose measurements is given in Table 1. The depth dose data measured for x-ray beams produced by lead and aluminum targets and flattened with lead and aluminum filters are shown in Columns 2, 3, 6 and 7. For comparison purposes we also show the depth dose data of the unflattened beams produced by the 2 targets (Columns 1 and 5, also shown in Figure 2), as well as the depth dose data for the Varian Clinac-35 and the Allis Chalmers 25 MeV betatron (Columns 4 and 8, respectively). Most of the conclusions that can be drawn from this Table were already mentioned, so that now only a brief summary will be given.

(1) Aluminum targets give a more penetrating beam than lead targets (compare Columns 1 and 5).

(2) Lead flattening filters soften the beam, although the effect is not very pronounced (compare Columns 1 with 3 and 5 with 6).

(3) Aluminum flattening filters harden the beam (compare Columns 1 with 2 and 5 with 7).

(4) The Varian Clinac-35 radiotherapy beam is almost identical to the beam produced by a lead target/lead flattening filter combination (compare Columns 3 and 4).

(5) The Allis Chalmers betatron beam is almost identical to the beam produced by an aluminum target/aluminum flattening filter combination (compare Columns 7 and 8).

TABLE I*

Flattening Filter / (cm) Depth in Water	Lead Target			Linac	Aluminum Target			Betatron
	None (1)	Al (2)	Pb (3)	Radio-therapy Beam (4)	None (5)	Pb (6)	Al (7)	Radio-therapy Beam (8)
0	24.3	26.4	30.4	—	23.5	26.7	23.4	—
0.1	33.0	29.5	36.0	20.5	32.0	32.5	27.0	18.8
0.25	50.5	35.5	45.0	38.5	42.5	41.5	35.5	30.0
0.50	62.5	49.8	56.1	59.0	60.3	56.0	46.0	45.3
0.75	74.5	58.1	72.3	72.4	71.2	67.3	57.5	55.5
1.0	80.7	66.8	78.7	80.1	77.2	76.5	66.5	64.9
1.5	89.5	79.0	89.2	90.3	85.9	86.1	78.3	77.3
2.0	95.7	87.8	95.7	95.5	91.1	91.6	86.1	86.0
2.5	98.7	94.3	98.9	98.7	95.3	95.3	92.5	91.9
3.0	99.8	97.7	100.0	99.9	97.8	97.8	96.1	95.6
3.5	100.0	99.3	99.8	100.0	99.2	99.4	98.0	98.1
4.0	99.2	100.0	99.3	99.7	100.0	100.0	99.1	99.5
4.5	97.8	99.4	98.6	99.0	99.7	99.2	99.8	100.0
5.0	96.7	98.4	97.3	97.6	98.7	98.0	100.0	99.8
6.0	93.7	96.1	93.8	94.5	96.4	95.4	98.2	98.2
7.0	90.6	93.3	90.8	91.4	93.6	92.4	95.5	95.9
8.0	87.3	90.7	87.9	88.4	90.6	89.2	92.4	92.9
9.0	84.1	87.8	84.7	85.2	87.4	85.9	89.6	90.2
10.0	81.2	84.8	81.4	81.9	84.0	82.7	86.8	87.4
11.0	78.1	81.8	78.5	78.8	80.8	79.7	83.7	84.6
12.0	75.2	78.8	75.5	75.9	77.7	76.7	80.8	81.6
13.0	72.2	76.0	72.6	72.9	74.7	73.4	77.7	78.5
14.0	69.4	73.2	69.6	70.1	71.9	70.5	74.8	75.4
15.0	66.6	70.3	66.7	67.2	68.9	67.7	72.1	72.5
16.0	64.1	67.7	64.2	64.5	66.3	65.0	68.8	69.6
17.0	61.6	65.5	61.8	61.8	63.9	62.4	66.7	66.7
18.0	59.2	63.5	59.6	59.2	61.6	60.0	64.6	64.3
19.0	56.8	61.5	57.2	56.7	59.2	57.8	62.1	62.1
20.0	54.5	59.5	54.8	54.5	57.0	55.7	59.9	60.0
21.0	52.4	57.4	52.6	52.5	55.0	53.6	57.6	57.7
22.0	50.1	55.4	50.6	50.3	52.8	51.1	55.7	55.9
23.0	47.8	53.5	48.3	48.2	50.7	49.7	53.6	53.7

* Depth dose data obtained from an electron beam of 25 MeV using various combinations of filters and targets. The linac data are for a Varian machine with a tungsten target and filter, while the betatron data are for an Allis Chalmers machine using a thin tungsten target and aluminum filter. Field size 10 cm circle at 100 cm. When flattening filters were used, the field was flattened to ±10 per cent over 90 per cent of the field.

ANGULAR DISTRIBUTION OF X-RAYS

We have already shown experimentally that the central axis x-ray yield is essentially independent of the target atomic number Z. On the other hand, the theories of x-ray production[2,3,5] predict a yield that increases with Z. These 2 apparently contradictory statements can perhaps be reconciled, if one keeps in mind that the theories are concerned with the *total* yield integrated over all angles, while our measurements so far presented are for the forward direction only. In order to gain a better understanding of this problem we have investigated the properties of the x-ray beam at various angles from the direction defined by the electron beam. For these measurements a small circular beam of 3 cm in diameter at a fixed target to surface distance of 125 cm was used. In addition, the diameter of the cylindrical target was increased to 10 cm. Depth dose

distributions were measured for both the aluminum and lead targets at various angles θ up to 60° from the central axis. In Table II we show the relative maximum doses normalized to 100 per cent for the beam produced at 0° by the aluminum target and obtained from the depth dose data at various angles θ. It is evident that as the angle θ increases, the value of the dose decreases rapidly. Furthermore, at large angles much more radiation is emitted by the high Z material, and even at 10° almost twice as much radiation is emitted from lead as from aluminum.

Table II does not give a complete picture, since not only does the number of photons decrease with angle but also the photon spectrum changes. This can be seen from Figure 6 which shows percentage depth dose curves for angles $\theta=0$, 10, 20 and 30° or from Table III, where we show the depths of the dose maxima in centimeters for various angles θ. For both the aluminum and lead targets, the larger the angle the smaller is the depth of maximum dose, indicating a lower effective beam energy. Again, as in Figure 2, the central axis beam ($\theta=0$) produced by the aluminum target is more penetrating than that produced by the lead target. At $\theta\sim10°$, however, both targets produce almost equally penetrating beams, and at angles larger than 10° the lead target gives a more penetrating beam than the aluminum target. It should be emphasized however that in radiotherapy one is interested in angles less than 10°, in which case the aluminum target produces a more penetrating beam than the lead target.

Table II*

Target \ Angle θ (deg.)	0	5	10	20	30	45
Aluminum	100	44.5	16.8	6.5	2.4	0.7
Lead	95	54.6	31.7	15.1	7.5	3.7

* Dose maxima as a function of angle for targets of aluminum and lead. The values are normalized to 100 per cent for aluminum at 0°.

We have used the depth dose data obtained at different angles to determine approximately the total energy emitted into a given solid angle. To do this the depth dose curves were extrapolated to infinity and the integral dose under each curve was calculated. The photon energy fluence per incident electron at a target to surface distance of 125 cm was then determined as a function of angle θ. This is shown by the dashed curves in Figure 7. The absolute fluence values shown in the figure are of limited accuracy because of the many approximations used in the calculation. Nevertheless, one can draw some conclusions from the relative shapes of the 2 fluence curves shown in the figure. In the forward direction, the fluences for aluminum and lead are essentially the same, but as the angle is increased beyond 5° the fluence from aluminum falls much more rapidly than that from lead. This may be explained as follows: in a high Z material larger angular deviations of the electron are produced than in a low Z material. The deviated electrons then produce x-rays, so more radiation is emitted at large angles from a lead than from an aluminum target. Furthermore, the electrons in lead suffer fewer collisions in being deviated to a large angle θ than those in aluminum, so they have greater energy and thus produce more energetic x-rays.

The total x-ray energy emitted by the targets can be calculated by integrating the fluence over the surface of a sphere of a radius equal to the source to phantom distance (125 cm). This integral is plotted in Figure 7 (solid line curves) as a function of angle θ from $\theta=0$ to $\theta=90°$. It is evident that the photon yield in the forward half sphere of the lead target ($\sim$20 per cent) is more than a factor of 2 larger than that of the aluminum target ($\sim$9 per cent). Thus, the total x-ray yields do depend on the atomic number Z of the target: the higher the Z the larger the x-ray yield. Over the range of angles of interest in

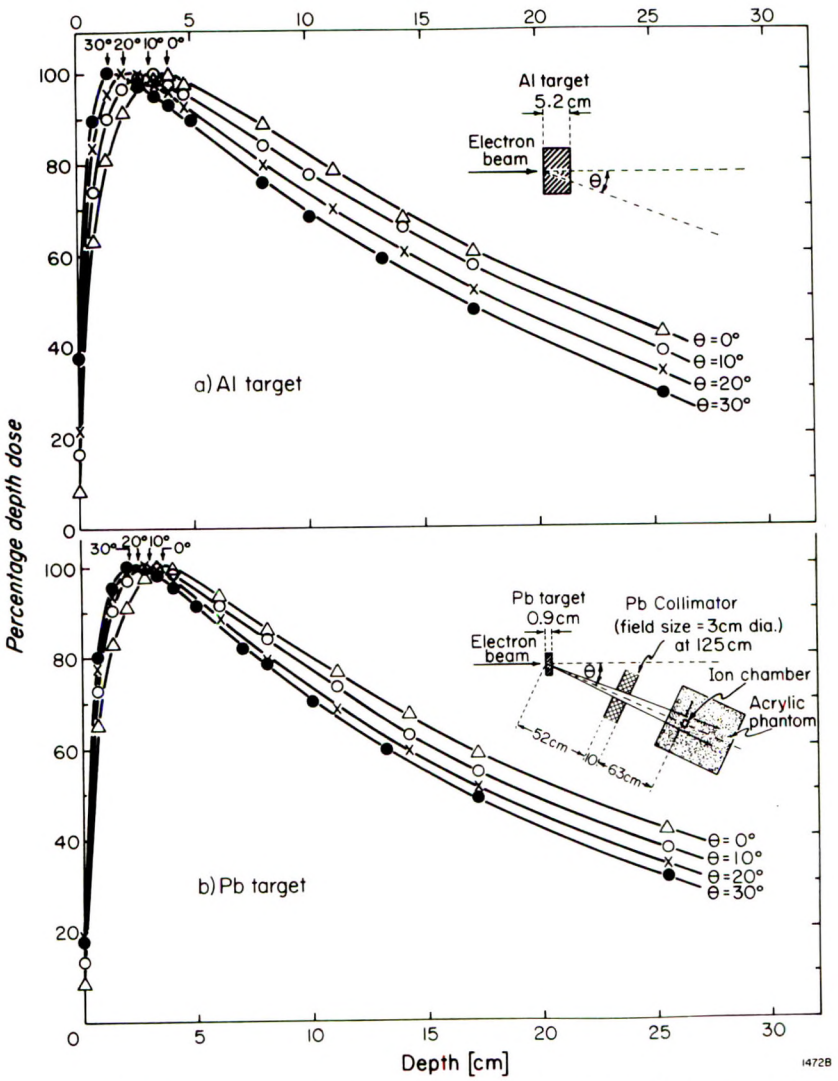


FIG. 6. Percentage depth dose curves of unflattened beams produced by (a) aluminum target and (b) lead target, for various angles from the central axis. Field size 3 cm diameter at a target to surface distance of 125 cm.

radiotherapy, however, the x-ray yields of aluminum and lead are not much different.

FIELD FLATNESS

We have shown in previous sections that an aluminum target produces a more energetic x-ray beam than a lead target, indicating that aluminum targets should be used in radiotherapy linear accelerators. But we have also shown that the x-rays from the aluminum target are more forward peaked than those from a lead target,

TABLE III*

Target \ Angle θ (deg.)	0	5	10	20	30	45
Aluminum	4.1	3.8	3.1	2.2	1.7	1.3
Lead	3.5	3.4	3.0	2.6	2.3	2.0

* Depths of dose maxima (in cm) as a function of angle for targets of aluminum and lead.

which suggests that in practice it might be difficult to flatten the beam produced by an aluminum target over an extended field. That this is not the case can be seen from Figure 8, which shows the results of our field flatness measurements obtained by scanning a Baldwin-Farmer ion chamber across the beam at a target to detector distance of 100 cm and a depth in the acrylic phantom of 10 cm. The dashed curves of Figure 8 show the unflattened and flattened profiles produced by the lead target, while the solid curves show the corresponding profiles produced by the aluminum target. In both cases, the filters have been designed to flatten the beam over a circular field size of at least 40 cm diameter. As can be seen from the figure, in order to flatten a beam of a given field size, the unflattened beam from the aluminum target has to be attenuated more than the one from the lead target. However, this difference is small and results in an effective output from the flattened aluminum beam which is only 20 per cent less than that from the flattened lead beam.

The variation of beam flatness with depth in the phantom was studied by comparing flatness measurements obtained at various

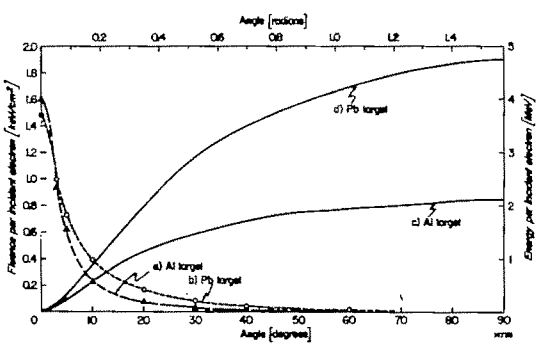


Fig. 7. The dashed curves represent the energy fluence (dE/dA), per incident 25 MeV electron as a function of the angle θ from the central axis (a) for an aluminum target and (b) for a lead target. The solid curves represent the integral of the fluence, $\int_0^{2\pi}\int_0^{\theta}$ (dE/dA) $R^2 \sin\theta d\theta d\phi$, over the surface of a sphere with radius, R, equal to the target to phantom surface distance (125 cm). This integral is equal to the x-ray energy emitted into a solid angle, Ω, defined by the angle θ.

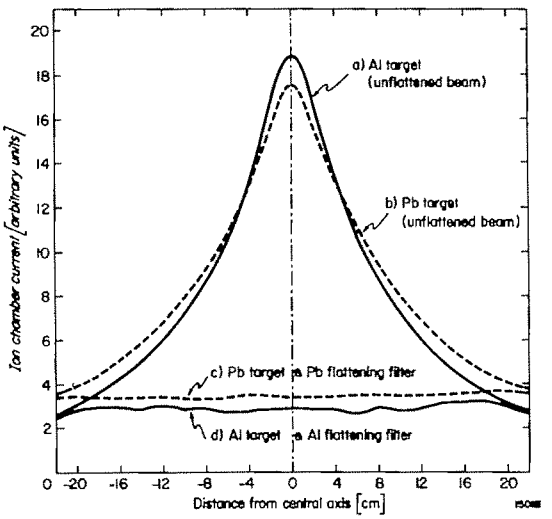


Fig. 8. A comparison between unflattened and flattened x-ray beams produced by 25 MeV electrons impinging on an aluminum target (solid line curves) and on a lead target (dashed curves).

depths (3 cm, 10 cm, 20 cm). One would anticipate a larger variation of flatness with depth in the case of the aluminum target/aluminum filter combination as compared to the lead target/lead filter combination. Although this was found to be true, the measured variation in both cases was small. For aluminum, assuming a perfect beam flatness at 10 cm depth, the uniformity across the beam at depths of 3 cm and 20 cm would be within ± 3 per cent.

OTHER CONSIDERATIONS

A number of other factors must be taken into account before we can consider the practical use of thick aluminum targets and filters in a radiotherapy linear accelerator. (1) The target must not be an intense source of neutrons; aluminum is satisfactory from this point of view. (2) The target should not become intensely radioactive; again aluminum creates no difficulty since most of the activity is short lived. (3) The cooling of an aluminum target is not a consideration. (4) An argument could be made that a thick target of low Z material would give a larger effective source of x rays than a thick target of high Z material. This

could conceivably increase the size of the penumbra. Experiments designed to test this idea showed, however, that there was no difference. (5) The main problem involves space, since large thickness of target and filter is required and their replacement in a finished linac would require a complete redesign of the head. In a linac under design, however, these considerations should be taken into account.

CONCLUSIONS AND SUMMARY

The 25 MeV electron beam was extracted from a Varian Clinac-35 linear accelerator and made to produce x-rays in thick targets of different materials. The x-ray beams were flattened by filters of various materials.

We have found that an aluminum thick target gives a more penetrating beam in the forward direction than does a lead or tungsten target.

The x-ray yield in the forward direction from 0–5° is essentially the same for both aluminum and lead targets. At angles larger than 10°, a lead target shows a higher x-ray yield and a more penetrating beam than an aluminum target.

The flattening filter material is important. A more penetrating beam is produced if the flattening filter is made of aluminum rather than tungsten or lead.

With an aluminum target and an aluminum flattening filter, we obtain the same depth dose distribution from our linear accelerator as we do from our betatron unit operating at the same energy. In the betatron unit, the radiation is produced in a thin target of tungsten and filtered by aluminum.

For the situations that arise in radiotherapy we have shown that the beam from an aluminum target/aluminum flattening filter combination can be flattened just as easily as the beam from a lead target/lead flattening filter combination.

We conclude, therefore, that contrary to conventional practice, low atomic number materials should be used for the targets and flattening filters of high energy radiotherapy linear accelerators.

We plan to extend our investigations of target and filter design to lower electron energies (10–20 MeV).

It is hoped that this paper will inspire manufacturers of new linacs to design the head to include aluminum targets and filters so that the optimum characteristics of the beam can be realized.

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REFERENCES

1. Berger, M. J., and Seltzer, S. M. Tables of Energy Losses and Ranges of Electrons and Positrons. Nuclear Science Series, Report Number 39, National Academy of Sciences, National Research Council, Publication 1133, Washington, 1964, pp. 205–268.
2. Berger, M. J., and Seltzer, S. M. Bremsstrahlung and photoneutrons from thick tungsten and tantalum targets. *Phys. Rev.*, 1970, *2*, 621–631.
3. Heitler, W. The Quantum Theory of Radiation. Third edition. Oxford University Press, London, 1954.
4. Johns, H. E., Rawlinson, J. A., and Taylor, W. B. Matrix dosemeter to study the uniformity of high energy x-ray beams. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *120*, 192–201.
5. Koch, H. W., and Motz, J. W. Bremsstrahlung cross-section formulae and related data. *Rev. Mod. Phys.*, 1959, *31*, 920–955.
6. Rawlinson, J. A., and Johns, H. E. Percentage depth dose for high energy x-ray beams in radiotherapy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 919–922.

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THREE-DIMENSIONAL IMAGING OF ABDOMINAL ORGANS WITH ULTRASOUND*

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THE usefulness of ultrasound in the assessment of internal abdominal anatomy is now well established.[11] Since ultrasound is sensitive to differences in acoustic impedance, it permits visualization of tissues with similar radiologic densities. Because it is limited to a two-dimensional image, however, it is necessary for the ultrasonographer to take multiple scanning sections and, from these sections, to reconstruct in his mind the three-dimensional nature of the patient. Suggested methods of producing a three-dimensional ultrasonic image include: varying the cathode-ray tube display to introduce perspective and stereoscopic viewing of different cathode-ray tube presentations;[3,12,13,21] use of a mobile fiber-optics display unit coupled with stereoscopic photography;[15] mounting of serial sections on transparent background and reconstruction of the sections in original order;[2] presentation of serial sections on a motion picture film or by holographic reconstruction;[18,23] and computerized reconstruction and display of serial sections.[14,17,20] These various methods have been reviewed.[7,24]

We have evolved a computerized reconstruction of two-dimensional ultrasonic B-scans obtained from currently available commercial B-scanners. Relatively simple computer manipulations permit multiple display modes of the ultrasonic information.

METHOD

The ultrasonic scanning apparatus consists of a modified Hewlett Packard 7214A Diagnostic Sounder, an Escoline B-scanner with a 2.25-MHz 10 cm. focused transducer and a modified Tektronix 564 storage oscilloscope. Any of the commercially available B-scan units would be satisfactory. The scanning procedure consists of recording a series of two-dimensional scans in the conventional manner.

The following coordinate system is used for all operations. The Z-axis traverses the body in a caudo-cranial direction and passes through the lumbar spine (*i.e.*, approximately through the center of the body); the cranial direction is considered positive and the caudal direction negative. The X-axis is perpendicular to the Z-axis and passes horizontally through the body 10 cm. above the iliac crests; the right side of the patient is considered negative and the left side positive. The Y-axis is perpendicular to the X- and Z-axes and passes through their intersections (*i.e.*, it passes vertically through the body 10 cm. above the iliac crests); the dorsal surface of the body is negative and the ventral surface positive.

Transverse scanning sections are made perpendicular to the Z-axis and are identified by their linear displacement in centimeters along that axis. Scans may be placed at any level, but we have found 2–3 cm. intervals to be the most useful. A zero-level transverse scan must be made in order to correctly orient the longitudinal sections. An appropriately labeled Polaroid photograph is made of each transverse scan.

Longitudinal scans are made in the craniocaudal direction and are oriented radially about the Z-axis with the negative X-axis considered 0° and the positive Y-axis 90° (*i.e.*, clockwise when viewed from

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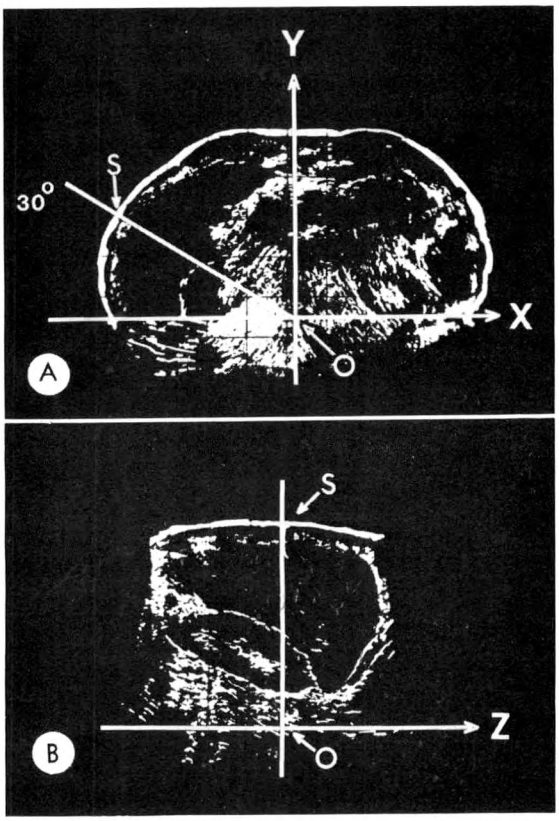


Fig. 1. Examples of ultrasonic sections showing coordinate axes. (*A*) Transverse section at level zero: the 30° line shows the orientation of the 30° longitudinal section. (*B*) Longitudinal section at 30°: the Z-axis is marked by measuring the distance S—O on the transverse section.

the patient's feet). We usually make longitudinal sections at 10° intervals. The location of the zero transverse section is marked upon each longitudinal section by raising the transducer off the skin as it crosses this level (10 cm. above iliac crests). A Polaroid photograph, labeled with the degree of rotation, is made of each longitudinal section. The location of the Z-axis is marked on each longitudinal print by measuring the distance from the skin to the spine in the appropriate orientation on the zero transverse section and then marking off this distance at the zero level on the longitudinal section (Fig. 1, *A* and *B*).

The outline of abdominal organs on the Polaroid prints is digitized with a D-Mac Pencil follower interfaced with the computer. The selection of material to be displayed is made at the time of digitization. The computer stores and displays every point which is traced by the pencil follower. If the physician wishes only information concerning the liver, he digitizes only the contour of the liver; if he desires a projection of the body surface and a tumor, he digitizes the skin and the tumor outline. Once the prints have been digitized, the information can be transferred to computer cards or tape and kept as part of the patient's record.

The computer used is an IBM 1800, which has 32 K, 16 bits per word with a disk-storage capacity of 1.500 K word.*

Several modes of display are available. The data points to be displayed are first entered on the computer's bulk storage, either by use of the pencil follower or by feeding in cards or tape containing previously digitized data. The viewing position is then specified as a degree of rotation about the X- and Y-axes. For example, X=0°, Y=0° will project the image as if it were being viewed from the patient's feet; X=90°, Y=0° will view the image in frontal projection; and X=0°, Y=90° yields a right lateral projection. After the viewing orientation has been specified, the

* The computer used is an IBM 1800. The storage requirements for the computer program described including FORTRAN routine system and data tables are approximately 11 K. The program is a synthesis of several standard techniques[15] and further alterations in the display modes may be introduced rather easily. The overlay feature of the MPX monitor system is used extensively. The system is programmed entirely in ANSI BASIC FORTRAN IV, but some inhouse-developed subroutine servicing the D-Mac pencil follower and the display system are used. To speed up the picture dynamics, nearly all calculations are performed in one-word integers and a scaling program is used to preprocess the data to be displayed to secure the picture quality and to avoid overflow during the processing of the picture data. The programs for the system described should have an acceptable performance and be adaptable to the following minimum computer environments:

CPU: add time<4 μseconds; multiply time<20 μseconds.
Core memory: 16 K words, 16 bits per word. Cycle time<2.5 μseconds per word.
Bulk storage: 100 K words. Mean position time<0.5 seconds.
Graphical display unit: video conversion system/storage scope.
Digitizing system: with a conversion precision of I 0.5 mm.
The monitor system of the configuration should have overlay capabilities and allow on-line communication with a running program.

Most mini-computers, *i.e.* Digital, Hewlett Packard, Nova, can be configurated to fulfill these requirements. A listing and some notes concerning the program may be obtained from the author if desired.

composite image is projected on the computer monitor screen with a perspective correction (*i.e.*, points further from the viewer are projected in smaller scale). If the viewing orientation is unsatisfactory, the viewpoint can be automatically shifted about the X- or Y-axes in 5° increments. Once initiated this process continues automatically and gives the feeling of slowly walking around the image. When a suitable orientation is found, the image is frozen and a Polaroid photograph taken.

If desired, a posterior plane can be introduced into the image at any specified distance from the origin of the coordinate system.† Only points lying anterior to this plane will be projected.

EXPERIMENTAL TRIALS

To illustrate some of the display modes of the present system, we constructed the outline of a familiar object—a Viking war helmet—and digitized 11 transverse and 4 longitudinal sections. Figure 2 shows this image projected from 2 viewpoints. In Figure 3, a posterior plane has been introduced into the image.

A normal abdominal scan is shown in Figure 4, *A* and *B*). In this particular case only the skin surface, the liver, and the spine were digitized; however, this choice was arbitrary and any identifiable outline could have been selected.

The three-dimensional projection of a patient with a 8×3 cm. perinephric abscess is shown in Figure 5. In this case the skin surface, abscess, and both kidneys are displayed.

A pancreatic tumor measuring 3 cm. is evident in Figure 6. In this case it was not possible to outline the tail of the pancreas due to an overlying gas-filled stomach.

Visualization of a perspective image in three dimensions, although requiring some concentration and practice, is possible for most observers. With some experience it has become easy for us to properly orient various structures in their correct mutual relationship. The greatest problem associated with such images is multistability (the spontaneous and involuntary shift of viewpoint).[1] When the perspective image is inadequate for a three-dimensional effect, a simple modification of the program can permit a horizontal shift of the viewpoint by 8 cm. Photographs taken of the image before and after this shift will form a true stereo pair which can be viewed in any stereo viewer.

The computer program may be easily modified to suit individual needs. In addition to the stereo shift, other options include: rotation about all three axes; provision for using oblique scanning sections as input data; and selective elimination of data points. Thus it would be possible to introduce an anterior as well as a posterior plane into the image, allowing visualization of a "slice" of the body in any orientation. Robinson[20] and Milan[16] have described

† This section of the program was developed by Andreas Munk-Madsen, B.Sc. and Jorgen Thomsen, B.Sc., University of Aarhus, Institute of Data Processing and Statistics.

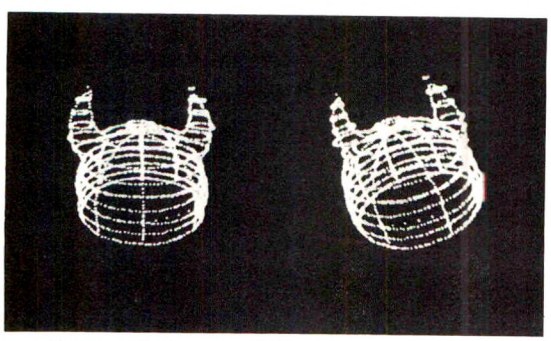


Fig. 2. Viking war helmet in 2 projections.

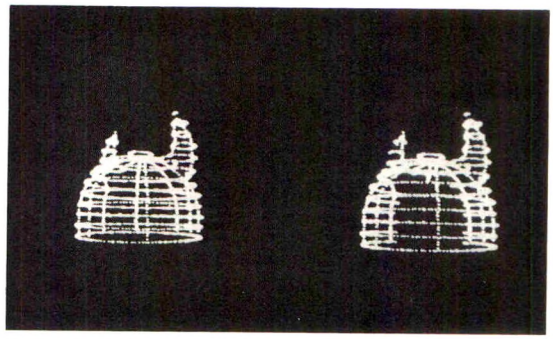


Fig. 3. Viking war helmet. The left figure is the complete helmet. The right figure is the same projection but with a posterior plane introduced.

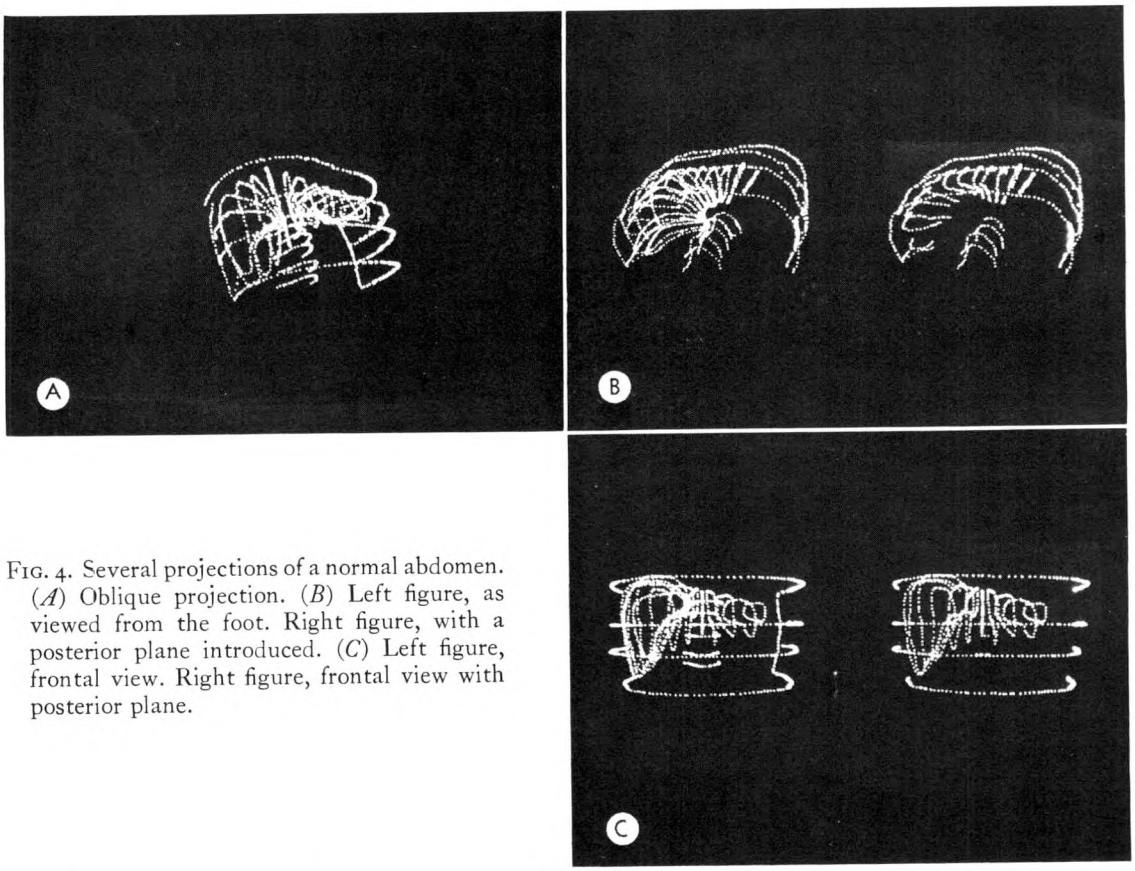


Fig. 4. Several projections of a normal abdomen. (*A*) Oblique projection. (*B*) Left figure, as viewed from the foot. Right figure, with a posterior plane introduced. (*C*) Left figure, frontal view. Right figure, frontal view with posterior plane.

similar systems. It would be easy to superimpose additional contours, such as a proposed radiotherapy treatment port or isodose curve, into the image.

DISCUSSION

In developing a computerized three-dimensional display of ultrasonic scans, we have tried to construct a system which could be easily integrated with currently available scanning equipment. The only additional hardware needed for this system is a mini-computer and digitizing apparatus. The scanning technique is like routine abdominal B-scanning, except that accurate orientation and labelling of the sections is required. Digitizing of the scans requires 15–30 minutes, depending on the

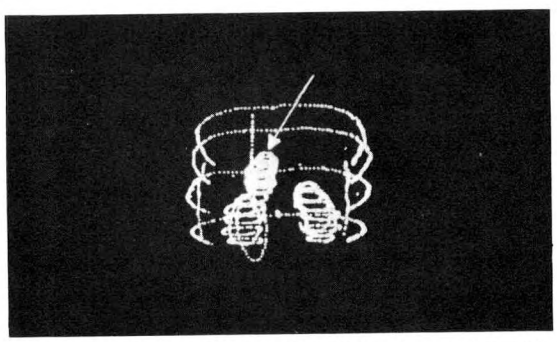


Fig. 5. Patient with a perinephric abscess (arrow) above the right kidney. Frontal projection.

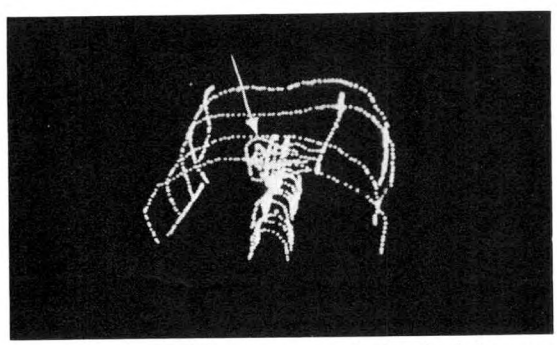


Fig. 6. Patient with a tumor in the head of the pancreas (arrow). The tail of the pancreas is not completely outlined.

number of sections and experience of the operator. Once digitized, the data can be stored on cards and displayed at the physician's convenience. The required number of scanning sections varies from patient to patient, but we have found transverse sections at 2–3 cm. intervals and 6 to 10 longitudinal sections to be adequate in most cases.

How much the three-dimensional image contributes to patient diagnosis and care is not known at this time. Due to the highly subjective nature of this kind of evaluation, it will probably require considerable experience by several different investigators before any answer to this question is possible.

Radiotherapy treatment planning is one area where such information can be definitely useful. Ultrasonic identification of organ and tumor contours has been reported from many different institutions.[6,8,19,22,25]

The use of computers in radiotherapy treatment planning has greatly facilitated the accuracy and speed of dosimetry calculations. However, to obtain the maximum benefits from the computer, it is necessary to have a highly accurate input of anatomic information. Brascho[4,5] and Eule *et al.*[10] have reported the use of digitalized ultrasonic B-scans as an input to treatment planning programs.

The system we have described is readily adaptable to a computerized treatment planning program. The patient undergoes a single abdominal scanning which, when digitized, produces an accurate map of his body contour, abdominal organs, and tumor. This image is displayed in the three-dimensional mode and rotated until a desired projection is obtained. Then anterior and posterior planes are introduced to give a cross section of the body in the selected orientation. The digital data from this cross section are fed into a treatment planning program either through a direct interface or *via* punch cards or tape. The generated isodose curves can then be superimposed on the image for a visual conformation of their adequacy. Because each section can be accurately localized on the patient's body by means of the standard coordinate system, more exact placement of treatment ports is possible. Progress of treatment can be followed by repeat scanning.

We hope by this report to stimulate an interest in ultrasonic three-dimensional imaging and, by such interest, to more rapidly define its ultimate place in clinical medicine.

SUMMARY

The ultrasound technique of imaging abdominal organs is presently limited to a two-dimensional representation.

The method for three-dimensional display of ultrasonic data presented here is based on computer processing of digitized data from multiple two-dimensional scans. The computer displays the total information in a perspective which can be visualized in 3 dimensions by most observers and simple modifications of the program will enable true stereoscopic images to be produced. Rotation and editing of the image is possible.

Requirements in hardware and personnel time are modest and should be within the means of most large hospitals.

The system should be of immediate value in radiotherapy planning, but its ultimate place in clinical diagnosis and care requires more extensive and widespread evaluation.

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REFERENCES

1. Attneane, F. Multistability in perception. *Scient. Am.*, 1971, *225*, 63–71.
2. Baum, G., and Greenward, I. Orbital localization by three-dimensional ultrasonography. *New York J. Med.*, 1961, *61*, 4149–4157.
3. Berkley, C. Three-dimensional representations on cathode ray tubes. *Proc. I.R.E.*, 1948, *36*, 1530–1535.

4. Brascho, D. J. Clinical applications of diagnostic ultrasound in abdominal malignancy. *South. M. J.*, 1972, *65*, 1331–1339.
5. Brascho, D. J. Computerized radiation treatment planning with ultrasound. Presented at Second World Congress on Ultrasonics in Medicine, Rotterdam, June 4–8, 1973.
6. Brown, R. E., Bogardus, C. R., and Sartin, M. Ultrasound, computers and radiation therapy planning. Presented at Second World Congress on Ultrasonics in Medicine, Rotterdam, June 4–8, 1973.
7. Brown, T. G. Visualization of soft tissues in two and three dimensions: limitations and development. *Ultrasonics*, 1967, *5*, 118–124.
8. Cohen, W. N., and Hass, A. C. Application of B-scan ultrasound in planning of radiation therapy treatment ports. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *111*, 184–188.
9. Danielsson, P. E. Three-dimensional electronic display. *B.I.T.*, 1970, *10*, 156–167.
10. Eule, J., Jr., Bockenstedt, F., and Salzman, E. Diagnostic ultrasound scanning: valuable aid in radiation therapy planning. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 139–145.
11. Holm, H. H. Ultrasonic scanning in diagnosis of space-occupying lesions of upper abdomen. *Brit. J. Radiol.*, 1971, *44*, 24–36.
12. Howry, D. H., Pasakony, G., Cushman, C. R., and Holmes, J. H. Three-dimensional and stereoscopic observation of body structure by ultrasound. *J. Appl. Physiol.*, 1956, *9*, 304–306.
13. Iams, H. A., Burtner, R. L., and Chandler, C. H. Stereoscopic viewing of cathode ray tube presentations. *R.C.A. Review*, 1948, *9*, 149–158.
14. Levinthal, C., and Ware, R. Three-dimensional reconstruction from serial sections. *Nature*, 1972, *236*, 207–210.
15. McDicken, W. N., Lindsay, N., and Robertson, D. A. R. Three-dimensional images using a fibre optic ultrasound scanner. *Brit. J. Radiol.*, 1972, *45*, 70–71.
16. Milan, J. Computer based system for three-dimensional scanning. Presented at the Second World Congress on Ultrasonics in Medicine, Rotterdam, June 4–8, 1973.
17. Rasmussen, S. N., Nielsen, S. S., and Stigsby, B. Three-dimensional visualization of liver by ultrasonic scanning. Presented at the 17th meeting of the American Institute of Ultrasound in Medicine, Philadelphia, Oct. 31–Nov. 2, 1972.
18. Redman, J. D., Walton, W. P., Fleming, J. E., and Hall, A. M. Holographic display of data from ultrasonic scanning. *Ultrasonics*, 1969, *7*, 26–29.
19. Reiner, K., and Heckrodt, R. A. Die Verwendung des Ultraschall-B-Bildverfahrens bei der Bestrahlungsplanung. *Strahlentherapie*, 1970, *140*, 639–646.
20. Robinson, D. E. Display of three-dimensional ultrasonic data for medical diagnosis. *J. Ac. Soc. Amer.*, 1972, *52*, 673–687.
21. Schmitt, O. H. Cathode ray presentation of three-dimensional data. *J. App. Physics*, 1947, *18*, 819–827.
22. Smith, E. H., and Holm, H. H. Ultrasonic scanning in radiotherapy treatment planning. *Radiology*, 1970, *96*, 433–435.
23. Szilard, J. Improved 3D display system. Presented at the Second World Congress on Ultrasonics in Medicine, Rotterdam, June 4–8, 1973.
24. Wells, P. N. T. Physical principles of ultrasonic diagnosis. Academic Press, Inc., New York, 1969, pp. 186–188.
25. Weyrauch, U., and Schoknecht, G. Eine Anwendung von Ultraschall zur Bestrahlungsplanung. *Strahlentherapie*, 1970, *139*, 541–547.

Vol. 121, No. 4

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Publication Committee: Luther W. Brady, Chairman, Philadelphia, Pa.; Kenneth L. Krabbenhoft; Alan F. Schroeder.

Public Relations Committee: Luis A. Vallecillo, San Turce, Puerto Rico, Chairman; Edward J. Beattie; Gerald Stephen Brown.

Janeway Lecture Committee: Franz J. Buschke, Chairman, '75; Luther W. Brady, '77; Alfred S. Ketcham, '79.

Representatives on the American Board of Radiology: Justin J. Stein, Los Angeles, Calif.; Ralph M. Scott, Louisville, Ky.; E. Richard King, Richmond, Va.

Liaison Representative on the National Council on Radiation Protection and Measurements: Morris J. Wizenberg, Baltimore, Md.

Representative on the Board of Chancellors of the American College of Radiology: Juan A. del Regato, Colorado Springs, Colo.

Fifty-seventh Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

EDITORIAL

FIFTY-SIXTH ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE Fifty-sixth Annual Meeting of the American Radium Society was held at the Maui Surf Hotel in Maui, Hawaii, April 21–25, 1974. The meeting, which was under the over-all direction of the President of the Society, Dr. Jerome M. Vaeth, was marked by a significant and important scientific program and by major decisions as to the over-all direction of the Society itself.

At the Business Meetings of the Society held on April 23, 1974 and April 24, 1974, the membership unanimously reaffirmed the multidiscipline character of the Society. Specific recommendations were made to broaden the membership of the Society to include all specialties of oncology. Also, the Society reaffirmed the fact that the AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY, AND NUCLEAR MEDICINE was the official journal of the Society, and that specific changes would be implemented on January 1, 1975, requiring subscription only to the 3 issues containing the publication of papers presented at the meetings of the Society.

Detailed discussion resulted in changes for membership in the Society eliminating the 3 year waiting period following completion of training and the necessity for submission of a thesis for membership. This was done in hopes of eliminating artificial barriers to membership, and also to stimulate younger members.

The final major decision taken by the Society was the unanimous approval that no more than 1 meeting out of 4 would be held outside the continental limits of the United States. This latter decision has required a review of the meeting sites for future years. With this in mind, the meeting site for 1975 will be San Juan, Puerto Rico, for 1976, Vancouver, British Columbia, for 1977, Las Vegas, Nevada, and for 1978, New Orleans, Louisiana.

The Scientific Session began on Monday, April 22, 1974. In the President's Address, Dr. Jerome M. Vaeth re-emphasized the role of radiation therapy in the development of the cancer center and, specifically, elaborated on the development of the West Coast Cancer Foundation and its impact on cancer care in this northern California area.

A Historic Symposium was chaired by Dr. Emanuel R. N. Grigg and dealt not only with the historic prospective of cancer care with specific reference to radiation therapy, but also with the development of new techniques in terms of diagnosis and treatment in cancer. Members of the symposium panel included Luther W. Brady, M.D., Klaas Breur, M.D., Robert L. Comis, M.D., Frederick W. George, M.D., Harold E. Johns, Ph.D., Malcolm H. McGavran, M.D., Theodore R. Miller, M.D., Milford D. Schulz, M.D., and Justin J. Stein, M.D.

The Resident Award Paper was presented by Dr. A. Lee Dellon and was titled "Thymus-dependent Lymphocyte Levels During Radiation Therapy for Bronchogenic and Esophageal Carcinoma: Correlation with Clinical Course in Responders and Nonresponders." Dr. Dellon dealt with the relationship between T-cell levels in patients with carcinoma of the lung and carcinoma of the esophagus correlating these levels with clinical tumor volume, tumor type, and the prognostic value that

might be associated with these levels. In his work, he was able to demonstrate that a return to a normal T-cell level was highly significant—a positive prognostic sign in squamous cell carcinomas of the lung. In adenocarcinomas of the lung, the findings were similar, but the evidence was not statistically significant. As the stage of the tumor increases, the T-cell level is depressed. Also, T-cell level can be correlated with the potential for surgical cure. Dr. Dellon suggested that sequential monitoring of T-cell levels could give significant information with regard to the development of metastasis. When the metastases develop, T-cell levels are dramatically decreased. Similar results were found in adenocarcinomas of the lung. As T-cell levels are diminished, the question of metastasis becomes a greater probability, but as T-cell levels increase, the potential for cure becomes more probable. During radiation therapy, T-cell level monitoring can be very helpful in predicting response. If T-cell levels diminish before the completion of radiation therapy, most patients develop metastasis within 2 months. Dr. Dellon suggested similar results in carcinomas of the esophagus. This represented a significant and important contribution in the prediction of prognosis in lung and esophageal cancer.

During the First Scientific Session, a symposium was held on renal carcinoma. Dr. George P. Annes and Dr. James L. Bennington of San Francisco dealt with the diagnostic radiographic features and the pathologic features relative to prognosis. Dr. Sameer Rafla discussed tumors of the upper urothelium in terms of natural history and the role of radiation therapy. He presented 41 cases of malignant tumors of the renal pelvis, 27 being transitional cell carcinomas, and 14 squamous cell carcinomas. The transitional cell carcinomas spread primarily to the ureter followed closely by the renal bed and bladder. Squamous cell carcinomas spread primarily through the renal bed followed by the draining lymph nodes. The results of treatment and factors influencing them were discussed indicating that transitional cell carcinomas faired better in terms of 5 year survivals (25 per cent) than squamous cell carcinomas (15 per cent). The 10 year results were 7 and 20 per cent respectively. Dr. Rafla pointed out that these tumors are responsive to radiation therapy, provided adequate dosage is delivered to the relevant volume within the proper time period.

Mr. Bowen Keller presented the rationale and technique for preoperative irradiation in renal cancer. The suggestion was made that the combination of radiation therapy and surgery was more effective than nephrectomy alone in the treatment of renal cancer. Data on 300 patients were presented with 155 being treated preoperatively. These data were from the National Renal Study and detailed analysis of radiation treatment plans with the frequency of their use was presented.

The Second Scientific Session was held Tuesday, April 23, 1974. Pathologic features of soft tissue sarcomas were presented by Dr. Robert A. Jeffrey, Jr. of San Francisco, California. Dr. Carlos H. Fernandez discussed the coordinated treatment plan for childhood rhabdomyosarcoma, presenting 103 children treated at the M. D. Anderson Hospital in the interval from 1950 to 1972. Eighty patients were evaluable at 3 years with an over-all 74 per cent of 23 patients receiving combined treatment being free of disease at 3 years compared with 42 per cent of 57 patients when other therapy techniques were used. This improvement was most evident when the primary site was the head and neck. The coordinated treatment programs suggested were radiation therapy delivering 5,500 to 6,000 rads tumor dose in 5 to 6 weeks, in conjunction with chemotherapy utilizing weekly vincristine, intermittent courses of actinomycin, and daily or high dose schedules of cyclophosphamide.

Dr. J. G. Fortner and his associates from New York presented a multidisciplined approach to the management of sarcomas

of the thigh. They pointed out that soft tissue sarcomas affect the thigh more commonly than any other site, making a modern multidisciplined approach including limb preservation possible in an increasing proportion of patients. Chemotherapy perfusion of the isolated extremity combined with muscle group resection was the program suggested. The chemotherapy program included adriamycin and methyl CCNU along with radiation therapy. Data were presented demonstrating that with wide soft part treatment, 5 year results of 50 per cent were obtained with 10 year results of 49 per cent. These compared with the results obtained by amputation of 42 per cent at 5 years and 32 per cent at 10 years. Only 10 per cent of the patients subjected to soft part resection in the series failed to achieve local control if previously untreated.

Dr. Robert D. Lindberg and associates presented the role of surgery and postoperative radiation therapy in the treatment of soft tissue sarcomas in adults. In the interval from 1954 through 1970, 97 patients were treated by simple excision and postoperative megavoltage radiation therapy. The local recurrence rate of soft tissue sarcomas in patients treated by this technique was significantly reduced.

Dr. Julian P. Smith and his associates presented a program for combined irradiation and chemotherapy for sarcomas of the pelvis. Thirty-eight women with recurrent, metastatic, or advanced sarcoma of the pelvis were treated with combined irradiation and chemotherapy. Fourteen of the patients remained alive and free of recurrence 8 months to 6 years after completion of the treatment. Six of these patients had metastatic sarcoma in the abdomen or the chest when treatment was started, 2 had recurrent sarcoma in the pelvis, and 6 had advanced sarcoma and were treated primarily with combined irradiation, chemotherapy and surgery. The combined treatment program offered significant and important contributions to the management of this problem.

Dr. William A. Caldwell presented on behalf of Dr. Burton Speiser and associates their study on malignant metastasizing fibrous histiocytoma with a presentation of 6 cases. These patients were studied clinically and pathologically at the University of Rochester, New York. Radiation doses of 2,000 to 5,500 rads were used for primary metastatic sites, following limited surgical procedures in 4 cases. Postirradiation specimens demonstrated changes consistent with maturation of the neoplastic histiocytes into more mature differentiated collagen formers. Specimens obtained in nontreated areas or from nontreated patients did not show these effects. The differentiation of the malignant elements toward benign elements in the irradiated areas suggests that radiation therapy has the potential for eradication of both the primary and metastatic lesions in malignant fibrous histiocytoma at therapeutically acceptable dose levels.

Dr. M. H. Shiu reported on the results of surgical and radiation treatment in liposarcomas of the extremities. Ninety-six patients with primary operable liposarcoma of the extremities were seen in the interval from 1949 to 1967 at the Memorial Sloan-Kettering Cancer Center. Soft tissue part resection was attempted in all cases and radiation therapy was employed in 43 of the 96 patients. This was used as an adjunctive therapy to surgical excision in 32 cases, and for distant metastasis in 11. The survival rate for soft tissue part resection at 5 years was 62 per cent *versus* 39 per cent for amputation, and at 10 years 51 per cent *versus* 30 per cent. Five and 10 year survival rates for all patients regardless of therapy were 53 per cent and 44 per cent with the local recurrence rate of 6 per cent. The author suggests that external irradiation is of definite value in the control of bulky tumors, locally recurrent cases, and for certain distant metastatic sites. Amputation might not be necessary in some instances.

In the second portion of the Second Scientific Session, a program on malignant

bone tumors was given with Dr. George P. Annes presenting the diagnostic radiographic considerations and Dr. Robert A. Jeffrey presenting the pathologic considerations.

Dr. H. O. Hustu and associates reviewed Ewing's sarcoma with information relative to local recurrence and distant metastases after concurrent irradiation and chemotherapy. Twenty-one children with clinically localized Ewing's sarcoma were treated in this manner. Of 14 patients treated over 2 years ago, 9 are alive without evidence of disease and 5 have died of the disease. Of the remaining 7 patients, all of whom were treated less than 2 years ago, 6 are alive without evidence of disease and 1 has died. The results indicate that irradiation given concurrently with chemotherapy reduces the incidence of local recurrences and distant metastases. This is associated with improved survival.

Dr. Thomas C. Pomeroy and Dr. Ralph E. Johnson presented the prognostic factors for survival in Ewing's sarcoma. Increased survival has resulted from the aggressive use of adjuvant chemotherapy. The analysis of 66 consecutive cases admitted to the National Cancer Institute indicates the significance of certain prognostic factors in future treatment planning. The site of the primary lesion and the age at diagnosis appear to be the most reliable indicators. Sex, constitutional symptomatology, histopathologic criteria, immunologic competency, and menarche in the female are less well-defined but nonetheless provocative variables of potential importance for the evolution of optimal therapeutic management of this disease.

Dr. Ralph E. Johnson and Dr. Thomas C. Pomeroy also presented the evaluation of therapeutic results and mechanisms of treatment failure in Ewing's sarcoma. The management for Ewing's sarcoma is based upon the premise that although metastases are not detectable, the disease should be recognized as systemic despite clinically "early" presentation. Based on this philosophy, a progressively modified therapeutic approach has been rewarded with improving survival rates for 66 patients treated at the National Cancer Institute. The results now approach a 50 per cent long-term survival for patients without clinical evidence of metastases at diagnosis and an impressive prolongation of survival for patients with obvious metastases on initial presentation.

Dr. Justin J. Stein presented a clinical report of 50 cases of osteogenic sarcoma treated at the UCLA Center for Health Sciences from 1955 to 1972. The methods of treatment and end results were discussed, indicating the value of combined treatment techniques.

The Third Scientific Session occurred on Wednesday, April 24, 1974. A symposium on malignant brain tumors was presented with Dr. George P. Annes giving the diagnostic radiographic features and Dr. Robert A. Jeffrey, Jr. the pathologic considerations.

Dr. Claude Y. Chong and associates presented radiation therapy for brain stem tumors. In the interval from 1960 to 1973, 96 patients were seen, with 6 demonstrating errors in diagnosis. Histologic diagnosis was obtained in 17 at the time of operation or at autopsy. Triad of signs were: cranial nerve deficits; pyramidal tract signs; and cerebellar signs. All patients received steroid treatment, 8 being treated with conventional voltage techniques and 82 by supervoltage techniques. The greatest incidence of death occurred during the first year with 10 of 52 patients surviving 5 years. The factors significant to prognosis were: histology; radiation dosage; field sizes for radiation therapy; and length of period with symptoms and signs. Age and sex had no influence on prognosis.

Dr. William L. DeGinder presented a program on the enhancement of therapeutic ratio in radiation therapy of brain neoplasms. He suggested that combined chemotherapy and radiation therapy strives for and succeeds in small gains in the treatment of brain malignancy. The over-all limiting factor, however, was the tolerance of normal tissues.

Dr. Gerald A. King and Dr. Robert H. Sagerman presented 26 patients with medulloblastoma treated postoperatively at the State University of New York, Upstate Medical Center. Fields encompassing the entire cerebral spinal axis were utilized, with currently acceptable programs of radiation therapy. The 5 year survival was 40 per cent. However, three of the long-term apparent disease-free survivors (6, 6, and 7 years) developed recurrence of disease. Data to explain the reason for failure were given.

The second portion of the Third Scientific Session was devoted to a discussion of salivary gland tumors. Dr. Robert A. Jeffrey, Jr. dealt with the pathology of salivary gland tumors.

Dr. Richard H. Jesse and associates presented data on carcinomas of the parotid gland. Until 1965, an aggressive program of surgery was followed at the M. D. Anderson Hospital, but after 1965 surgery has been combined with radiation therapy. Data with regard to 125 patients treated in the interval from 1944 to 1965 were presented. The ultimate fate of these patients depends upon the local control of the disease and this is emphasized by the fact that 72 per cent of the patients developing distant metastases also had local recurrences. Dr. Jesse reinforced the evidence that radiation therapy will increase the local control of tumor of the more aggressive histologic types, such as high-grade mucoepidermoid, adenoid cystic, malignant mixed, and anaplastic lesions. Indications for postoperative radiation therapy were: tumors of highly malignant character; invasion of bone, skin, or muscle; positive regional lymph nodes following resection of recurrent tumor; tumors of the deep lobe of the parotid; gross residual disease following surgical resection; and tumor adjacent to the facial nerve.

Dr. Kent J. Rossman presented the role of radiation therapy in carcinoma of the parotid. A retrospective study of 47 patients seen at Ohio State University Hospital showed a 72 per cent 5 year survival for surgery alone, 25 per cent 5 year survival for radiation therapy plus surgery, and 20 per cent 5 year survival for irradiation alone. Dr. Rossman indicated that survival was dependent upon the extent of the disease and tumor histology. The poorest survivors were found in those who had squamous cell carcinomas or undifferentiated lesions of the parotid. Of significant importance also was the fact that NSD doses greater than 1,800 rads were associated with 100 per cent control.

The Janeway Lecturer was Dr. Milford D. Schulz, Professor of Therapeutic Radiology at Harvard Medical School. His subject was "The Supervoltage Story." Dr. Schulz gave a broad, in-depth study of the development of supervoltage apparatus in the world. During his lecture, he presented early research work with laboratory notebooks and laboratory data as well as information relative to the newest machines.

The Fourth Scientific Session was held on Thursday, April 25, 1974. Dr. Carlos A. Perez and associates presented the effects of regional lymphadenectomy on tumor cure with experimental observations in normal and previously cured mice, using a transplanted murine lymphosarcoma. Previous experiments showed that lymphadenectomy or irradiation of the regional lymph nodes significantly decreased cure rates when these procedures were performed between 4 days prior to the subcutaneous transplantation of Gardner lymphosarcoma or 5 days following transplantation. The data presented by Dr. Perez further supported the concept that the integrity of the regional lymph nodes is paramount to the initiation of an effective immune response in this particular tumor.

Dr. William M. Wara and associates presented the data on immunosuppression following radiation therapy for carcinoma of the nasopharynx. Eleven patients have had prospective immunologic evaluation prior to and following definitive radiation therapy for nasopharyngeal carcinoma. Antibody mediated immunity was evaluated as well as thymic independent anti-

gens. Cell mediated immunity was also assayed along with serum IgA levels. Cellular immunity was severely depressed following irradiation, with an average total lymphocyte count of 698, depressed PHA stimulation, and decreased rosette formation at 1 and 6 months.

Professor Dr. K. Breur discussed the possibilities of clinical application of fast neutrons and the need for cooperative clinical trials. To determine the type of tumors in which local neutrons can be expected to be more advantageous, efforts should be made to use, apart from experimental laboratory studies, quantitative data on the differential effect in clinical models. He presented a study of pulmonary metastases to illustrate this point.

Dr. Stanley E. Chism and Dr. Henry Keys presented data on a time/dose analysis of control and complications in carcinoma of the cervix. Three hundred and fifty patients were treated by radiation therapy from 1963 to 1972. The data were evaluated in terms of Ellis's Nominal Standard Dose concept and also from the standpoint of the merits of lymphangiography, diagnostic selective lymphadenectomy, and extended field irradiation.

Dr. Alvah J. Nelson discussed the indications for adjunctive conservative extrafascial hysterectomy in selected cases of carcinoma of the uterine cervix. He pointed out that there had been an increased use of this modality of treatment since 1954 with 212 patients with either squamous cell or adenocarcinoma having had the treatment. In squamous cell carcinoma and adenocarcinoma, equivalent end results were achieved indicating that in certain situations this combined modality (extrafascial hysterectomy and radiation therapy) was preferable to radiation therapy alone. These indications were: bulky intercervical lesions; pregnancy; fibroids; and positive endometrial biopsy. The incidence of complications from the conservative surgery and radiation therapy were 14.7 per cent, whereas the complication rate was 36.3 per cent when radiation therapy and radical hysterectomy and lymphadenectomy were carried out. Dr. Nelson also suggested that adenocarcinoma is no longer considered a surgical problem, since the disease is sensitive to radiation therapy.

Dr. John T. Fazekas and associates presented a program of radiation therapy for Stage I and Stage II Hodgkin's Disease. One hundred and forty-seven patients were presented, seen at Walter Reed General Hospital between 1960 and 1970. The data indicate that as the volume of irradiation increased from involved sites to extended fields to the current policy of therapy, the 5 year disease-free survival increased from 55 per cent to over 95 per cent.

Dr. Douglas S. Wong and associates discussed their experience with extranodal lymphomas of the head and neck. One hundred and twenty-eight Stage I and Stage II extranodal non-Hodgkin's lymphomas of the head and neck were presented, having been treated definitively between 1944 and 1969.

Dr. D. W. Kinne and associates gave an analysis of 62 cases of primary gastric lymphoma. Of these cases, 9 were lymphosarcomas, 1 Hodgkin's disease, and 49 reticulum cell sarcomas. The 5 year survival without disease in those patients treated by biopsy and radiation therapy was 6 per cent; where surgical resection alone without postoperative radiation therapy or chemotherapy was used, the 5 year survival was 32 per cent; and in those patients treated by surgical resection plus postoperative adjunctive radiation therapy, the 5 year survival was 45 per cent. The data indicate that surgical resection of primary gastric lymphoma with postoperative radiation therapy is the treatment of choice for primary gastric lymphoma.

Dr. Jack J. Schneider and associates presented data on the control by radiation therapy alone of nonfixed clinically positive lymph nodes from squamous cell carcinoma of the oral cavity, oropharynx, supraglottic larynx and hypopharynx. One hundred and ninety-one patients were studied, treated

by external irradiation alone, and a dose response analysis was made relating size and multiplicity of lymph nodes.

Dr. J. H. Kim and Dr. B. Hilaris presented a clinical experience with the use of encapsulated iodine 125 sources in interstitial tumor therapy. The clinical experience in 91 patients with metastatic carcinoma of the neck lymph nodes implanted with I-125 (54 patients), Ir-192 (14 patients), and Rn-222 (23 patients) was presented. It is suggested that I-125 sources are adequate and effective and compare well to other radionuclides used. The over-all local tumor control rates in patients treated with I-125 and Rn-222 seeds were 76 per cent and 61 per cent. The local complication rates were 19 per cent and 30 per cent, respectively.

Dr. Eric P. Weber presented a progress report in the treatment of carcinoma of the breast by definitive radiation therapy. One hundred cases of regional, potentially curable, breast malignancy were treated at the Joint Center for Radiation Therapy from July 1, 1968 to June 30, 1973. They were treated with megavoltage radiation and supplementary Ir-192 implantation in the area of residual disease. At the point of evaluation, there were no local failures in those patients who had Stage I and Stage II disease, and there had been a high percentage of local control in those patients with clinical Stage III disease. The author concluded that the survival is similar to other treatment techniques and that this might be an adequate substitute for radical mastectomy.

The Fifty-sixth Annual Meeting of the American Radium Society was also marked by outstanding social events including a Welcoming Reception, a Luau, the Annual Banquet, and other entertaining social activities. Credit must be given to the Arrangements Committee under the chairmanship of Dr. Carl W. Boyer and to Dr. Jerome M. Vaeth for arranging an excellent scientific program. Special credit is due to Mr. Lee Kirkland for his devoted and continuing efforts in making the meeting such an outstanding success.

Luther W. Brady, M.D.

Chairman, Publication Committee of the American Radium Society

NATIONAL RESEARCH COUNCIL

COMMITTEE ON RADIOLOGY, DIVISION OF MEDICAL SCIENCES

Picker Foundation Awards, 1974–1975

ON behalf of the James Picker Foundation, the National Research Council (NRC) announces the awarding of 14 fellowships and grants, which are renewals for work already in progress. Six are being given for the first time, and 4 pilot grants are also awarded.

Those selected to participate in the program for the 1974–1975 academic year are:

JAMES PICKER FELLOWSHIPS IN ACADEMIC RADIOLOGY

Dr. Eric L. Effmann, in his first year as a James Picker Fellow, will investigate the developing lung by radiographic techniques with major emphasis on the study of vertebrate embryology and mastery of techniques in classical and experimental embryology. The work will be done under the sponsorship of Dr. Richard H. Greenspan, at Yale University School of Medicine.

Dr. Samuel J. Hessel's first 9 months of his fellowship program will be spent in the cardiac catheterization and angiography laboratories of Harvard Medical School and Peter Bent Brigham Hospital, working in the areas of myocardial and coronary physiology and angiography under the sponsorship of Dr. Herbert L. Abrams. The remaining 3 months will be spent at Children's Hospital Medical Center, Boston.

Dr. Anne G. Osborn, an academic trainee in diagnostic radiology at the National Institutes of Health, will conduct research, during this first year as a Picker Fellow, in self-instructional methods in diagnostic radiology using programmed instruction techniques and a learner-controlled, computer-assisted instruction system. Her work will be done at the University of Utah College of Medicine and Medical Center and the Division of Instructional Research, Development, and Evaluation at Brigham Young University, under the sponsorship of Dr. David G. Bragg.

Dr. Richard M. Sigel, as a first-year Picker Fellow, will conduct investigations of the microscopic and ultrastructural aspects of cerebrospinal fluid production and absorption at Cornell University Medical Center under the sponsorship of Dr. John A. Evans and the supervision of Dr. D. Gordon Potts.

Dr. Sam Silverman, a resident at the University of California School of Veterinary Medicine, Davis, has been awarded a Picker Fellowship to study the application of nuclear medical and special radiographic procedures to subhuman primates. The work will be done under the sponsorship of Dr. J. P. Morgan, veterinary medical director and chairman of the Department of Radiological Sciences at UCD.

Dr. James Winter, as a first-year Picker Fellow in Academic Radiology, will study transverse tomography using holographic image reconstruction. His research will be done at the University of California at Los Angeles under the sponsorship of Dr. Gabriel H. Wilson, and the supervision of Dr. Moses A. Greenfield. The goal of his proposed research is the implementation of transverse axial tomography using coherent optical methods for the required computation.

ADVANCED FELLOWSHIPS IN ACADEMIC RADIOLOGY

Dr. Jason C. Birnholz has been awarded his second-year Advanced Fellowship to continue his investigation of quantitative ultrasound with specific identification of intra-abdominal neoplasms by sonic imaging techniques. This work will be under the sponsorship of Dr. Juan M. Taveras at Massachusetts General Hospital, Harvard Medical School.

Dr. Conrade C. Jaffe was awarded a renewal of his fellowship, but, since he has been called to active duty by the U. S. Navy, the fellowship will be interrupted until Dr. Jaffe completes his service in the Navy.

Dr. Charles A. Mistretta, during his third year as an Advanced Fellow, will continue his investigation of new imaging techniques in diagnostic radiology under the sponsorship of Dr. John Juhl at the University of Wisconsin Medical School. His research during the past year has dealt primarily with use of storage tubes for catheter guidance in angiography, image enhancement using a multiple subtraction technique, and fluoroscopic dose reduction using pulsed x-rays and image storage.

RESEARCH FELLOWSHIP

Dr. Pejavar S. Rao, born in Alleppey, India, will spend his second year as a Research Fellow continuing his investigation of the detection and delineation of soft tissue tumors under the direction of Dr. Earle C. Gregg at Case Western Reserve University. He has a well-prepared and sound research plan for enhancing the density contrast of x- and gamma rays by counting Compton radiations at predetermined angles using the Case Western spiral scanner.

SCHOLAR AWARDS

Dr. Melvyn Korobkin's second Scholar Award will enable him to continue his research, "Urographic and Angiographic Studies of Renal Ischemia," at the University of California Medical Center in San Francisco.

Dr. James A. Nelson, with a second Scholar Award, will continue his investigation of the problems associated with clinical use of oral and intravenous contrast media for visualization of gallbladder and biliary ducts.

RESEARCH GRANTS

Dr. Michael A. Davis, during his second Research Grant, will continue his attempts to develop techniques for the labeling of autologous lymphocytes, granulocytes, and platelets with technetium and to evaluate the products as imaging agents for use in diagnostic nuclear medicine.

Dr. Norman A. Frigerio has been awarded a second-year Research Grant to investigate the localization of radiolabeled compounds in spontaneous and induced malignancies. This research involves 3 types of murine tumors: Krebs Ascites transplanted as a sarcoma; 3-methylcholanthrene-induced squamous cell carcinoma; and the C3H/Hej spontaneous mammary adenocarcinoma.

Dr. E. Stephen Garnett, with a second Research Grant, will continue to work at McMaster University, Hamilton, Ontario, to develop [F^{18}]-3, 4-dihydroxy-5-fluoro phenylalanine ([F^{18}]-fluoro-DOPA) as a radiopharmaceutical with a specific activity high enough to make it useful as a brain-scanning agent in man, which will delineate vascular and neoplastic lesions with equal facility.

Dr. Michael T. Gyepes of the Center for the Health Sciences in Los Angeles, California had the term of his first-year Research Grant deferred until this year. His research will involve evaluation of fetal circulation generally and in specific organ systems through angiographic techniques. Such evaluations will permit *in vivo* imaging of the fetal circulation with advanced recording techniques.

Dr. John G. McAffee, Professor and Chairman of the Department of Radiology at the State University of New York Upstate Medical Center, supported by a second-year Research Grant, is to continue his work in the development of a new radiopharmaceutical labeled with Tc^{99m}, which can be safely administered to humans intravenously and which will concentrate in the normal myocardium sufficiently well to demonstrate areas of infarction as filling defects.

Dr. Salvador Treves, born in Buenos Aires, Argentina, is now the head of the Division of Nuclear Medicine of the Chil-

dren's Hospital Medical Center in Boston, Massachusetts. With this second-year Research Grant, he plans to investigate radionuclide assessment of regional lung function in babies and small children using a radioactive gas (xenon 133), a gamma scintillation camera, and an on-line dedicated digital computer.

Dr. Jack Wittenberg, in the Department of Radiology at Boston University School of Medicine, with a second-year Research Grant, will study infusion cholangiography to determine the precise dose and rate of delivery of iodipamide (Idp) needed for optimal opacification of the biliary tract for both a normal animal and one with biliary obstruction.

Dr. Gerald L. Wolf, at the University of Nebraska Medical Center, has been awarded a second-year Research Grant to investigate a micro-electrode study of contrast-agent-induced arrhythmias. The overall goal of his project is to elucidate, at cellular level, the mechanisms of cardiac arrhythmias produced by contrast agents.

PILOT STUDY GRANTS

Dr. Harold A. Baltaxe of Cornell University Medical College has received a pilot grant to study intravenous hepatosplenography.

Dr. Patrick J. Conte's pilot grant will be used to investigate combined hyperbaric oxygen and P^{32} therapy for skeletal metastases from breast carcinoma. His work will be done at the Saint Barnabas Medical Center.

Dr. Donald L. King will study three-dimensional, computer-assembled ultrasonic imaging of the heart at Columbia University.

Dr. Frank F. Zboralske, Stanford University, will be investigating the electrical and motor properties of gastric sling fibers.

NATIONAL COUNCIL ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

THE National Council on Radiation Protection and Measurements has just published NCRP Report No. 41, *Specification of Gamma-Ray Brachytherapy Sources.*

Brachytherapy is a method defined as radiation therapy in which an encapsulated source or a group of such sources is utilized to deliver gamma or beta radiation at a distance up to a few centimeters either by surface, intracavitary, or interstitial application.

Various encapsulated radionuclides that emit gamma rays are used in brachytherapy in the same manner as, and frequently substitutes for, radium sources. Except for radionuclides that emit low energy gamma photons, the relative distribution of absorbed dose around such substitutes is frequently given with an accuracy sufficient for medical purposes, when the dose distribution is calculated from available radium dosage tables. Because radium dosage tables have been available and used for many years, many radiation therapists wish to retain them to determine the relative dose distribution produced by other radionuclides. However, calculation of absolute dosage requires an appropriate quantitative specification of the particular brachytherapy source. The new report carries the NCRP's recommendations for this specification.

Presently, encapsulated radionuclides are being specified in terms of the activity of the radionuclide, in terms of the equivalent mass of radium, or in terms of the exposure rate at a specified distance from the source. The new report examines each of these methods of specification and develops recommendations on the method of specification considered to be appropriate. The report resulted from the Council's belief that a single well defined method of specification for sealed sources will be advantageous for both the users and suppliers of such sources.

The present report was prepared by the Council's Scientific Committee 27 on Specification of Radium Substitutes. Serving on the Committee during the preparation of this report were: *Chairman*, Marvin M. D. Williams; *Members*, Ivan M. Grotenhuis, Antolin Raventos, Robert J. Shalek; *Consultants*, John G. Beach, L. Thomas Dillman, Robert O. Gorson, John Hale, Robert Loevinger, Herbert M. Parker, Walter S. Snyder, Nagalingam Suntharalingam, and Harold O. Wyckoff.

The officers of the NCRP are: Lauriston S. Taylor, *President;* E. Dale Trout, *Vice President;* and W. Roger Ney, *Executive Director.*

Copies of NCRP Report No. 41, "Specification of Gamma-Ray Brachytherapy Sources," can be purchased at the following prices: 1 through 99 copies, each copy $3.00; 100 through 999 copies, each copy $2.25; and 1,000 or more copies, each copy $1.50. Individuals and organizations already on the NCRP Publications Standing Order List will receive copies of the new report automatically and be invoiced for their order. Others may purchase copies of the new report, or place their name on the Standing Order List, by directing their order or request to: NCRP Publications, P.O. Box 30175, Washington, D.C. 20014.

I.A.E.A. CATALOGUE OF RADIATION DOSIMETRY DATA

THIS catalogue, published by the International Atomic Energy Agency (I.A.E.A.) in Vienna, contains a comprehensive list of practical radiotherapy physics information culled from Departments of Radiology and Radiobiology throughout the world. The Agency acts as a clearing center for these data as part of its general commitment to disseminate information on radioisotopes and other radiation sources in order to assist its Member States to gain the knowledge needed for their proper applications of ionizing radiations in medicine. Originating some 30 years ago in Great Britain as the Diagrams and Data Scheme of the Hospital Physicists' Association, this activity was taken over by the I.A.E.A. in 1963, and the current catalogue, last revised in 1970, contains some hundreds of individual items. These consist mainly of isodose charts for beams of x rays and of cobalt 60 and cesium 137 gamma rays, and of dose distributions around standard radium containers. Data such as the attenuation coefficients for photons in various materials over a wide range of energies are also included. Items of data are made available in the form of transparencies, as photostat copies, and in certain cases as microfilm, to all *bona fide* users in accordance with the Royal Society's Fair Copying Declaration, on a nonprofit-making basis.

The Agency recently invited me (D. E. A. Jones) to undertake the revision and updating of the 1970 catalogue and I have contracted with their Division of Life Sciences to collect and evaluate the data needed and to extend the scope of the catalogue. The additional information will, it is hoped, include depth dose charts for neutron beams and dose distributions for brachytherapy containers of artificial radioisotopes. Broad beam attenuation data are also needed for all the appropriate photon and neutron energies, in media of interest for the purposes of radiation protection in building construction.

This announcement will, it is hoped, stimulate the interest of those workers in the field of ionizing radiation in medicine to submit such data as they may have accumulated (or expect to obtain shortly) and which appears to be suitable for inclusion in the catalogue. Actual charts, tables or data in other forms should not be transmitted at this stage. The details should be addressed to D. E. A. Jones, 147 Cassiobury Drive, Watford, Hertfordshire, U. K. Every entry in the catalogue will make reference to the author or the institution of origin.

For those readers as yet unfamiliar with the I.A.E.A. Catalogue, the current issue will be sent gratis upon application to the International Atomic Energy Agency, Dosimetry Section, Kärntner Ring 11, P. O. Box 590, A-1011 Vienna, Austria.

NEWS ITEMS

ASSOCIATION OF UNIVERSITY RADIOLOGISTS

At the Annual Meeting of the Association of University Radiologists held in New York City, New York in May of 1974, the following officers were elected: *President*, Melvyn H. Schreiber, M.D., Galveston, Texas; *President-Elect*, Richard H. Greenspan, M.D., New Haven, Connecticut; and *Secretary-Treasurer*, Mark M. Mishkin, M.D., The Graduate Hospital, University of Pennsylvania, Philadelphia, Pennsylvania.

The 23rd Annual Meeting will be held in San Diego, California, May 4–6, 1975.

Information concerning that meeting may be obtained from Dr. Elliot Lasser, Department of Radiology, University of California, San Diego, California 92103.

SYMPOSIUM ON GENERAL DIAGNOSTIC RADIOLOGY

The Department of Diagnostic Radiology of the Lahey Clinic Foundation announces a "Symposium on General Diagnostic Radiology" to be held at the Copley Plaza Hotel, Boston, September 13–14, 1974.

The Course is credited by the AMA for 14 hours Category 1 credit.

For further information, please contact: Carl R. Larsen, M.D., Program Chairman, Department of Radiology, Lahey Clinic Foundation, 605 Commonwealth Avenue, Boston, Massachusetts 02215.

POSTGRADUATE COURSE ON THE GENITOURINARY TRACT

A Postgraduate Course on the Genitourinary Tract will be held at the Barbizon Plaza Hotel in New York City, October 7–9, 1974.

The Course is sponsored by the Department of Radiology, The New York Hospital-Cornell Medical Center.

For further information, please contact: John A. Evans, M.D., Department of Radiology, The New York Hospital-Cornell Medical Center, 1300 York Avenue, New York, New York 10021.

POSTGRADUATE COURSE IN RADIOLOGY OF BONE

Harvard Medical School

A Postgraduate Course in Radiology of Bone will be sponsored by Dr. Herbert L. Abrams and Dr. Harry T. Griffiths and the Harvard Medical School, October 14–18, 1974.

This Course will revise the radiology of bone disease in relationship to its fundamental pathology and clinical relevance. The Course is intended to complement other courses on the Radiology of the Skeletal System, by approaching bone disease from a clinical, orthopedic and pathologic point of view. The lectures have been drawn from those specialties as well as from radiology—both local and distant.

The course is directed at specialists in radiology, pathology and orthopedics.

Please send enquiries to: Associate Dean, Harvard Medical School, Department of Continuing Education, 25 Shattuck Street, Boston, Massachusetts 02115.

REFRESHER COURSE ON RADIOLOGY OF CHEST DISEASES

The University of Toronto, Faculty of Medicine, Division of Postgraduate Medical Education and the Department of Radiology will sponsor a Postgraduate Refresher Course on the Radiology of Chest Diseases at Mount Sinai Hospital, Toronto, Ontario, October 25 and 26, 1974.

The Course will include a series of symposia. Some of the topics will be as follows: I. Diffuse Lung Disease; II. Obstructive Lung Disease; III. Pulmonary Vascular Disease; and IV. The Chest and the Intensive Care Unit. There will also be a panel

on The Investigation of the Unknown Lesion.

The Guest Faculty members are to be Dr. Robert G. Fraser, Diagnostic Radiologist-in-Chief, Royal Victoria Hospital, Montreal and Dr. Richard Greenspan, Professor and Chairman, Department of Radiology, Yale University.

For further information, please contact: The Director, Division of Postgraduate Medical Education, Medical Sciences Building, University of Toronto, Toronto, Ontario, Canada, M5S 1A8.

SYMPOSIUM ON LATE CONSEQUENCES OF SUCCESSFUL CANCER THERAPY: PROVEN AND POTENTIAL

A Symposium on this topic will be held at the Sheraton Olympic Villas, Orlando, Florida, January 9–11, 1975.

The Symposium is sponsored by the Clinical Cancer Investigation Review Committee of the National Cancer Institute, and its Program Chairman is Giulio J. D'Angio, M.D., Memorial-Sloan Kettering Cancer Center, New York, New York.

The meeting is open to all physicians interested in cancer therapy. There is no registration fee.

For further information, please write to: Audrey E. Evans, M.D., Director, Division of Oncology, The Children's Hospital of Philadelphia, 18th and Bainbridge Streets, Philadelphia, Pennsylvania 19146.

POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

The New York Hospital-Cornell Medical Center is sponsoring a 4 day Postgraduate Course in Diagnostic Radiology at the King's Inn and Golf Club in Freeport, Grand Bahama Island, Bahamas, February 10–13, 1975.

Scientific sessions dealing with the gastrointestinal and genitourinary tracts, skeletal system, chest and pediatrics will be held from 8:30 A.M.–1:00 P.M. each day. Special emphasis will be placed on the anatomic and pathologic correlation.

For further information, please contact: John A. Evans, M.D., Department of Radiology, New York Hospital-Cornell Medical Center, 1300 York Avenue, New York, New York 10021.

REFRESHER COURSE IN CARDIAC RADIOLOGY

A Refresher Course in Cardiac Radiology will be held by the North American Society for Cardiac Radiology in Williamsburg, Virginia, March 2–6, 1975.

A distinguished faculty will conduct seminars covering the radiology of acquired and congenital heart disease and its clinical implications.

For further information, please write to: Klaus Ranninger, M.D., Medical College of Virginia, MCV Station, Box 2, Richmond, Virginia 23298.

INTERNATIONAL SOCIETY OF LYMPHOLOGY

The Fifth International Congress of Lymphology will be held in Buenos Aires, Argentina and Rio de Janeiro, Brazil, March 23–29, 1975.

Sessions dealing with the following topics will be presented: Lymphatics and Immunology; Lymphomas; Lymphatic Dissemination of Cancer; Lymphedema and Chylous Refluxes; Medical and Surgical Treatment; Basic Lymphology; Anatomy and Physiology of the Lymphatic System; Progress on Lymphangiography.

Deadline for abstracts is October 1974.

The President of the Society is P. Ruben Koehler, M.D., Department of Radiology, University of Utah Medical Center, Salt Lake City, Utah 84132.

For further details, please contact: Carlos M. Grandval, M.D., Austria 2626, Buenos Aires, Argentina, or Rubens C. Mayall, M.D., C.P. 1822, ZC-00 Rio de Janeiro, Brazil.

INTERNATIONAL SYMPOSIUM ON CANCER OF THE HEAD AND NECK

An International Symposium on Cancer of the Head and Neck sponsored by the

Foundation International Cooperation in the Medical Sciences will be held at Montreux, Switzerland, April 2–4, 1975.

The Honorary Committee is under the Chairmanship of L. Clodius, M.D., Zürich.

Those who intend to present a paper are kindly invited to submit an abstract of maximum 300 words before December 31, 1974 to the Program Secretary, at The Hague.

The Secretariat of the Symposium meeting is at Rue de la Paix 11, P.O. Box 236, 1820 Montreux Vd., Switzerland.

The U.S.A. information office is at The Upton Federal Building, 6609 Reisterstown Road, Baltimore, Maryland 21215.

It is with deep regret that we announce the death of John Paul Medelman, M.D., of White Bear Lake, Minnesota, distinguished member and past president of the American Roentgen Ray Society, in St. Paul, Minnesota on July 6, 1974.

BOOK REVIEWS

Radiologic Examination of the Urinary Tract. By Howard M. Pollack, M.D., Associate Professor of Radiology, Temple University School of Medicine, and Director, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. Reprinted from Karafin and Kendall's Urology, Volume I in Practice of Surgery. Cloth. Pp. 164, with 145 illustrations. Price, $8.50. Harper & Row, Publishers, Inc., 2350 Virginia Avenue, Hagerstown, Md. 21740, 1972.

This introductory text (180 pages) by Dr. Pollack is reprinted from Karafin and Kendall's Urology in Practice of Surgery. It deals with the conduct and performance of most of the uroradiologic examinations being performed for the study of the genitourinary tract.

This is not a book of pathologic diseases, but rather a concise informed text with a lucid readable style which provides an introduction to the subject of present day uroradiologic practice. The application of the various diagnostic modalities and the interpretation of the results of these examinations is based on the theme that each study has a particular purpose and that the choice of each procedure should be applied with an approach designed to suit the individual case.

This book should be read by all trainees in Radiology, Urology, and Nephrology for a concise summary of the anatomy, physiology, and current status of radiologic investigative procedures of the urinary tract. Certainly, all departments which run a training program in Radiology cannot afford to be without their own copy.

Richard C. Pfister, M.D.

Fraction Size in Radiobiology and Radiotherapy. Edited by Tsutomu Sugahara, M.D., Professor, Department of Experimental Radiology, Faculty of Medicine, Kyoto University, Japan; László Révész, M.D., Professor, Department of Tumor Biology, Karolinska Institutet, Medical School, Stockholm, Sweden; and Oliver C. A. Scott, Bt., British Cancer Council, London, England. Cloth. Pp. 236, with some illustrations. Price, $23.00. The Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, exclusive U. S. agents, 1974.

Several decades ago the physicist assumed his predestined role in the medical aspects of Radiology. The radiation physicist determined how radiation could be safely utilized and accepted the responsibility for teaching these developments to students of this fledgling specialty. It was only within the last decade, however, that the biologist was successful in producing his major contribution to Radiology —how radiation really works, that is, how it kills the cell. It is now the radiation biologist's turn to establish his role in the education of the radiologist; especially when the latter becomes a radiation oncologist.

This book contains the proceedings of a symposium on "The Size of Individual Dose Fractions in Radiobiology and Radiotherapy," which was held in Kyoto, Japan, on February 25th and 26th, 1972, and some invited papers by scientists and clinicians who did not participate, but whose work is closely related to the subject of the symposium. The majority of those who attended the symposium were Japanese, and it is their hope that this publication will give English-speaking readers some idea of what scientific endeavors are being done in Japan today.

Since it is the authors' belief that progress in medical radiobiology and the practice of radiotherapy will be more rapid if there is close contact between experimentalists and clinicians, the book begins with "The Place of Radiobiology in an Oncology Centre." The functions of the radiobiologist and his relationships with the other members (both clinical and experimental) of the oncology group are explained and their importance is stressed.

The major portion of this publication is divided into the following 4 main sections:

(1) "The Role of Cellular, Vascular and Other Stromal Reactions to Radiation in Determining Dose-Fractionation Regimes;"
(2) "Possible Alteration of Conventional Fractionation Due to the Use of Chemical Sensitizers, Protectors and High LET Radiation;"
(3) "The State of Oxygenation of Tumors and its Influence on the Choice of the Size of Individual Dose Fraction;" and
(4) "Clinical Trials."

Each section is composed of individual papers that explain in detail the radiobiologic aspects of the featured topic. These chapters are well written and contain numerous illustrations, tables, graphs and drawings that assist the reader in his understanding of data which are often quite complex, but most appropriate to present day problems in radiation oncology. The frequent orientation of the subject material to a clinical situation is a refreshing variance from many scientific publications of this type.

It is my opinion that *Fraction Sizes in Radiobiology and Radiotherapy* should be readily available to all students in radiation oncology, not only for its subject material, but for the complete list of references included in each chapter.

Paul M. Kroening, M.D.

BOOKS RECEIVED

Dorland's Illustrated Medical Dictionary. Twenty-fifth edition. Cloth. Pp. 1,748. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1974.

Abdominal Angiography. By W. Wenz, in collaboration with G. van Kaick, D. Beduhn and F.-J. Roth. Cloth. Pp. 217, with 183 figures. Price, $27.80. Springer-Verlag, New York, Inc., 175 Fifth Avenue, New York, N. Y. 10010, 1974.

Clinical Cardiac Radiology. By Keith Jefferson, M.A., F.R.C.P., F.F.R., Consultant Radiologist, National Heart Hospital and St. George's Hospital, London; Lecturer, Cardiothoracic Institute, University of London; and Simon Rees, M.A., M.R.C.P., F.F.R., Consultant Radiologist, National Heart Hospital and St. Bartholomew's Hospital, London; Lecturer, Cardiothoracic Institute, University of London, England. Cloth. Pp. 314, with many illustrations. Price, $57.75. Butterworths, 2265 Midland Avenue, Scarborough, Ontario, Canada M1P 4S1, 1973.

Indications and Alternatives in X-Ray Diagnosis: a Guide to the Effective Employment of Roentgenologic Studies in the Solution of Diagnostic Problems. By Melvyn H. Schreiber, M.D., Professor of Radiology, University of Texas Medical Branch, Galveston, Texas. Paper. Pp. 176, with some illustrations. Price, $11.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill. 62717, 1974.

Formulating X-Ray Techniques. Eighth edition. By John B. Cahoon, R.T., F.A.S.R.T., Assistant Professor, Duke University Medical Center; Technical Director, Programs in Radiologic Technology, Duke University Medical Center, Durham, Veterans Administration Hospital, Durham, N. C. Cloth. Pp. 381, with some illustrations. Duke University Press, Durham, N. C., 1974.

X-ray Anatomy of the Vascular System. By G. Luzsa, M.D., Chief Physician of the Radiological Department, City Hospital, Mosonmagyaróvár, Hungary. Cloth. Pp. 387, with 222 illustrations. Price, $27.00. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1974.

The Fundamentals of Radiological Science. By John Hale, Ph.D., Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pa. Cloth. Pp. 343, with some illustrations. Price, $14.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1974.

Postoperative Coronary Angiography. By Matti Valle, The First Department of the Institute of Diagnostic Radiology, Meilahti Hospital, University Central Hospital, Helsinki, Finland. Paper. Pp. 67, with many figures. Price, Sw.Kr. 40:–. Acta Radiologica Supplementum 333. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

Carcinoma of the Uterine Corpus: a Retrospective Survey of Individualized Therapy. By I. Joelsson, A. Samari, and H. L. Kottmeier, Department of Gynecology, Radiumhemmet, 104 01 Stockholm, Sweden. Paper. Pp. 63, with some figures. Price, Sw.Kr. 40:–. Acta Radiologica Supplementum 334. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

Metrizamide: a Non-Ionic Water-Soluble Contrast Medium; Experimental and Preliminary Clinical Investigations. Paper. Pp. 390, with some figures. Price, Sw.Kr. 50:–. Acta Radiologica Supplementum 335. Acta Radiologica, Box 2052, 103 12 Stockholm 2, Sweden, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

American Radium Society
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

Radiological Society of North America
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Portland-Hilton Hotel, Portland, Oregon, March 31–April 4, 1975.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Atlantic City, N. J., June 15–19, 1975.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following city during this year. Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers in June 1975.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1975 is September 30, 1974.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

The third examination is scheduled for Saturday, Sept. 21, 1974. Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017. The deadline for receipt of completed applications was June 1, 1974.

American Association of Physicists in Medicine
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D.C. 20010. Annual Meeting: Kansas City, Mo., Aug. 22–24, 1974.

American Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting: Sonesta Beach Hotel, Key Biscayne, Fla., Oct. 30–Nov. 4, 1974.

American Nuclear Society
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545. Winter Meeting: Washington, D.C., Oct. 27–Nov. 1, 1974.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

Fourteenth International Congress of Radiology
Rio de Janeiro, Brazil in 1977.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

Eleventh Inter-American Congress of Radiology
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aéreo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

Inter-American College of Radiology
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

Third Congress of the European Association of Radiology
Secretary General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland. Meeting: June 22–27, 1975.

Second Asian and Oceanian Congress of Radiology
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters 2161 Taft Ave., Manila, Philippines.
Meeting: Manila, Philippines, Nov. 10–14, 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society, Chapter ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

American Thermographic Society
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Mark M. Mishkin. The Graduate Hosp., Univ. of Pennsylvania, Philadelphia, Pa. 23rd Annual Meeting: San Diego, Calif., May 4–6, 1975.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Marshall Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on four Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Charles J. Masters, Maj. MC, Department of the Air Force, United States Air Force Hospital, Wiesbaden (USAFE) APO New York 09220.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joshua A. Becker, Downstate Medical Clin., 450 Clarkson Ave., Brooklyn, N. Y. 11203. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Franco, 45 Alexander St., Lockport, N. Y. 14094. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Daniel E. Lewis, Grant Hosp., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL TEXAS RADIOLOGY SOCIETY
Secretary-Treasurer, Dr. John M. Stoebner, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

CHICAGO RADIOLOGICAL SOCIETY, DIVISION OF THE ILLINOIS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

COLORADO RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

DELAWARE CHAPTER OF ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

EAST BAY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George N. Weston, 1515 Ygnacio Valley Rd., Walnut Creek, Calif. 94598. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Paul A. Mori, 800 Miami Rd., Jacksonville, Fla. 32207. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

GEORGIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER KANSAS CITY RADIOLOGICAL SOCIETY
President-Secretary, Dr. Rodger W. Lambie, 701 E. 63rd St., Ste. 108, Kansas City, Mo. 64110.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Richard E. Litt, 6125 S.W. 31st St., Miami, Fla. 33155. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

HAWAII RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

HEALTH PHYSICS SOCIETY
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

IDAHO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. John A. Knote, 716 Carrolton Blvd., West Lafayette, Ind. 47906.

IOWA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Bruce Saxe, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference: Century Plaza Hotel, Los Angeles, Calif., Jan. 31–Feb. 2, 1975.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Eli A. Etscovitz, 10 Stonewood Dr., Newton Centre, Mass. 02159.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bdg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

New England Roentgen Ray Society
Secretary, Dr. N. Thorne Griscom, Children's Hosp, 300 Longwood Ave., Boston, Mass. 02119. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 3:00 P.M. Annual Meeting.

New England Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference, New Orleans, La., Apr. 25–27, 1974.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

North American Society of Cardiac Radiology
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Pablo Ramos, Box 1618, Bismarck, N. D. 58501. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Mark D. Reiss, 5301 F Street, Sacramento, Calif. 95819. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

NORTHWESTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Tearle L. Meyer, 175 S. Merkle Rd., Columbus, Ohio 43215.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting: Washington Plaza Hotel, Seattle, Wash., May 10–12, 1974.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 22–24, 1974.

SAN ANTONIO-CIVILIAN MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President, Dr. Stanley G. Seat, 476 Prospect St., LaJolla, Calif. 92037. Meets the first Wednesday of each month at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION OF RADIOLOGY, NATIONAL MEDICAL ASSOCIATION
Chairman, Dr. Roy G. Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broad-

way, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, Vanderbilt University Hospital, Nashville, Tenn. 37232.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 111 Whitehall Bldg., 960 E. Third St., Chattanooga, Tenn. 37403. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton, St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. James O. Richardson, Jr., 280 Mamaronek Ave., White Plains, N. Y. 10605. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1 Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St.

Catherine St. West, Montreal 107, Que., Canada. Annual Meeting: Toronto, Ont., May 11–16, 1975.

EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

FACULTY OF RADIOLOGISTS
Honorary Secretary, Robert Morrison, Lincoln's Inn Fields, London WC2A 3PN, England.

MONTREAL RADIOLOGICAL STUDY CLUB
Secretary, Dr. W. Paul Butt, Montreal General Hospital Montreal, Que., Canada. Meets first Tuesday evening, October to April.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

SOCIÉTÉ CANADIENNE-FRANÇAISE DE RADIOLOGIE
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

THE HOSPITAL PHYSICISTS' ASSOCIATION
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

THE ROYAL AUSTRALASIAN COLLEGE OF RADIOLOGISTS
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

SOUTH AMERICA

COLÉGIO BRASILEIRO DE RADIOLOGIA
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

FEDERACIÓN ARGENTINA DE SOCIEDADES DE RADIOLOGÍA
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

SOCIEDAD ARGENTINA DE RADIOLOGÍA
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

SOCIEDAD COLOMBIANA DE RADIOLOGIA
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

SOCIEDAD ECUATORIANA DE RADIOLOGÍA
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

SOCIEDAD PERUANA DE RADIOLOGIA
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

SOCIEDAD DE RADIOLOGÍA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

SOCIEDAD DE RADIOLOGÍA DEL NORDESTE ARGENTINO
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

SOCIEDAD DE RADIOLOGÍA DE LA PLATA
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

SOCIEDADE DE RADIOLOGÍA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

SOCIEDAD DE ROENTGENOLOGÍA Y MEDICINA NUCLEAR DE LA PROVINCIA DE CÓRDOBA
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

SOCIEDAD DE RADIOLOGÍA, RADIOTERAPÉUTICA Y MEDICINA NUCLEAR, DE ROSARIO
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

SOCIEDAD SALTEÑA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

SOCIEDAD VENEZOLANA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

CONTINENTAL EUROPE

ÖSTERREICHISCHE RÖNTGEN-GESELLSCHAFT
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

SOCIÉTÉ ROYALE BELGE DE RADIOLOGIE
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

SOCIÉTÉ EUROPÉENNE DE RADIOLOGIE PÉDIATRIQUE
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Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

SOCIÉTÉ FRANÇAISE DE RADIOLOGIE MÉDICALE, MÉDECINE NUCLÉAIRE ET ÉLECTROLOGIE, and its branches: SOCIÉTÉ DU SUD-OUEST, DU LITTORAL MÉDITERRANÉEN, DU CENTRE ET LYONNAIS, DU NORD, DE L'OUEST, DE L'EST, DU MASSIF CENTRAL, D'OUTRE-MER ET D'ÉTRANGER. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
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Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

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Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

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Societas Radiologica Danica
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Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
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Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
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Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel. Nineteenth National Radiology Congress: Tel-Aviv, Israel, Nov. 6–8, 1974.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

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Association of Radiologists of West Africa
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Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

NECK AND CHEST

ARNAUD, A. *et al.:* Selective bronchial arteriography in hemoptysis 915
GOULON, M. *et al.:* Fatty embolism after repeated perfusions of lipid emulsions 915

ABDOMEN

GREENBAUM, E. I., and FREEDMAN, S.: Neoplasia of the colon over a long segment 915
FRIEDLAND, G. W. *et al.:* Diverting ileostomy for colonic Crohn's disease. Part 2: Radiological findings in the colon following diversion 916
NORA, P. F. *et al.:* Cholecystokinin cholecystography in acalculous gallbladder disease .. 916
COTTON, P. B., and BEALES, J. S. M.: Endoscopic pancreatography in management of relapsing acute pancreatitis 916
MILLER, T. A. *et al.:* Pancreatic abscess 917

GENITOURINARY SYSTEM

MOREAU, J.-F. *et al.:* Radiology of acute renal insufficiency 917
COOKE, G. M., and BARTUCZ, J. P.: Spontaneous extravasation of contrast medium during intravenous urography: report of fourteen cases and a review of the literature 917
HATELY, W., and WHITAKER, R. H.: How accurate is diagnostic ultrasound in renal disease? 918
KING, W. W. *et al.:* Current status of prostatic echography 918

SKELETAL SYSTEM

BASU, S. P.: Bone changes in leprosy: Dr. Diwan Chand Aggarwal Memorial Oration for 1972 919
JACOBS, P.: Osteolytic Paget's disease 919
MCELFRESH, E. C., and COVENTRY, M. B.: Femoral and pelvic fractures after total hip arthroplasty 919
MIRRA, J. M., and MARCOVE, R. C.: Fibrosarcomatous dedifferentiation of primary and secondary chondrosarcoma: review of five cases 920
BARRIER, J. *et al.:* Pubic osteitis and postpartum thrombophlebitis 920

BLOOD AND LYMPH SYSTEM

PINET, F. *et al.:* Dissections, fissurizations and ruptures of the aortic wall: contribution of aortography by arterial route; report of 69 observations 920
HIGHMAN, J. H., and O'SULLIVAN, E.: Isotope venography of pelvic veins 921
BLOOM, J. D. *et al.:* Defective limb growth as a complication of catheterization of the femoral artery 921
HENKIN, R. E. *et al.:* Radionuclide venography (RNV) in lower extremity venous disease .. 922
NIKAIDOH, H. *et al.:* Aortic rupture in children as a complication of coarctation of the aorta . 922
BLIZNAK, J., and STAPLE, T. W.: Radiology of angiodysplasias of the limb 923

ROENTGEN DIAGNOSIS

Neck and Chest

Arnaud, A., Giraud, R., Autran, P., Pasquier, J., and Clément, J. P. Artériographie bronchique sélective et hémoptysies. (Selective bronchial arteriography in hemoptysis.) *J. de radiol., d'électrol. et de méd. nucléaire*, Feb., 1974, *55*, 105–109. (Address: R. Giraud, Service Central de Radiologie, Hôpital Sainte-Marguerite, B.P. 29, 13274 Marseille Cedex 2, France.)

Selective bronchial arteriography is recognized as one of the significant procedures in the routine examination of the lungs in cases of hemoptysis.

Anomalies and abnormalities of the bronchial circulation are best evaluated in the light of the total clinical and radiologic findings. In the presence of hemoptysis, these findings may be considered in relation to one of the following:

1. When all conventional studies fail to reveal any abnormal findings—and routine chest roentgenography and bronchography are normal. There were 6 cases in this category.

A case in point is reported in which all conventional studies were normal. Bronchial arteriography demonstrated the presence of a voluminous artery in the right posterior basal segment, presenting an angiomatous appearance. There was injection of the pulmonary artery and vein at the same level. Resection of the right lower lobe resulted in a cure. The surgical specimen revealed the presence of bronchial pathology, not detected on the preoperative bronchogram.

2. When conventional studies reveal minor abnormalities with minimal bronchial changes, irregular segmental or tubular ectasia.

There were 9 cases in this group. An illustrative case is reported in which selective bronchial angiography demonstrated an area of hypervascularization, well beyond the abnormal area demonstrated on the bronchogram. Resection of the right lower lobe resulted in complete cure with no recurrence of the hemoptysis.

3. When conventional studies reveal multiple obvious lesions—selective bronchial angiography will determine which of these lesions is the source of hemoptysis, thus making definitive surgery possible.

Illustrative cases belonging to each of the 3 groups are presented. These include: minimal bronchiectasis; vascular anomalies; esophageal diverticula with accompanying minor bronchial changes.

This report is based on a study of 26 patients with severe hemoptysis on whom selective bronchial arteriography was performed. Definitive surgery was carried out in 10 cases without recurrence of symptoms.

Six reproductions of illustrative roentgenograms accompany this article.—*William H. Shehadi, M.D.*

Goulon, M., Barois, A., Grosbuis, S., and Schortgen, G. Embolie graisseuse après perfusions répétées d'emulsions lipidiques. (Fatty embolism after repeated perfusions of lipid emulsions.) *La Nouvelle Presse méd.*, Jan., 1974, *3*, 13–18. (Address: Pr M. Goulon, Hôpital Raymond Poincaré, 104 bd. Raymond Poincaré, F. 92380, Garches, France.)

Immediate reactions and intolerance to perfusions of lipid emulsions are more frequent, whereas late reactions after repeated and prolonged perfusions are rarely observed. These latter are known as the "fat overloading syndrome."

The authors present 1 such case with a typical "snow-flake lung" roentgenogram and all the clinical and laboratory findings. Then they summarize the findings of various authors on 9 similar cases.

It is their belief that the stabilizing agent of the emulsions is destroyed by antibodies formed during the repeated perfusions, thus precipitating the fat in the blood stream, and thereby causing these late severe embolic accidents. They are of the opinion that these antibodies form more frequently when lecithin derived from soya, rather than from egg yolk, is used in the preparation of the emulsions.—*Jirair N. Sarian, M.D.*

Abdomen

Greenbaum, Edward I., and Freedman, Sheldon. Neoplasia of the colon over a long segment. *Clin. Radiol.*, Oct., 1973, *24*, 416–426. (Address: Edward I. Greenbaum, M.D., Serra Memorial Hospital, 9449 San Fernando Road, Sun Valley, Calif. 91352.)

A series of well documented and nicely illustrated colonic lesions is presented to point out that neoplasia of the colon may present as long segment defects having tapered margins and largely intact mucosal patterns. Adenocarcinomas involving a long segment will usually be seen in the left colon, where the tumor tends to grow around the lumen and may remain superficial. Carcinoma of the right colon will usually be polypoid in nature and will extend along one wall of the bowel.

Tumors of varying cell-type—including scirrhous carcinoma, carcinoid, and lymphoma—will present an unusual bowel wall defect, and tumors invading from without will likewise present a different roentgen pattern. The type of bowel, "the nature of the soil," may produce yet another variability in the tumor pattern as is illustrated by carcinoma developing in ulcerative colitis.

Perforation of a carcinoma with a pericolic inflammatory mass or a carcinoma which has eroded a blood vessel with resultant hemorrhage into a long segment of bowel wall may result in a difficult differential diagnosis.—*Richard E. Kinzer, M.D.*

Friedland, Gerald W., Kohatsu, Shoichi, and Oberhelman, Harry A., Jr. Diverting ileostomy for colonic Crohn's disease. Part 2: Radiological findings in the colon following diversion. *Clin. Radiol.*, Oct., 1973, *24*, 435–439. (From: The Department of Radiology, Stanford Medical Center, Stanford, Calif., and Department of Surgery, Division of Gastrointestinal Surgery, Stanford Medical Center and the Palo Alto Veterans Administration Hospital, Palo Alto, Calif.)

Radiologic and clinical features of 14 patients both preceding and following diverting ileostomy are evaluated.

A previous paper on colonic Crohn's disease dealt with a grading system.

The authors note that radiologic findings in the colon following a diverting ileostomy for colonic Crohn's disease, as well as the findings in the colon after re-establishment of intestinal continuity, have not previously been described.

Fifteen patients were included in the study, although 1 had no preoperative barium enema examination available. There was complete clinical and radiologic agreement in 10 of the 14 patients, 9 of whom improved and 1 failed to improve. There was clinical improvement in 4 patients, who showed radiographic progression of the disease.

Five patients were studied after intestinal continuity had been re-established. Colonic haustra reappeared in all patients, but 2 patients had recurrent symptoms supported by radiologic reappearance of ulcers.

Six radiologic illustrations are found in this interesting report. The reader would have enjoyed more profuse illustration.—*Richard E. Kinzer, M.D.*

Nora, Paul F., McCarthy, William, and Sanez, Nicholas. Cholecystokinin cholecystography in acalculous gallbladder disease. *A.M.A. Arch. Surg.*, April, 1974, *108*, 507–511. (Address: Paul F. Nora, M.D., 6969 North Lincoln Avenue, Lincolnwood, Ill. 60646.)

The authors report their findings in a group of patients with clinical evidence of recurrent gallbladder disease in whom routine "work-up" examinations were essentially negative. Oral cholecystography with 3 gm. iopanoic acid showed visualization of the gallbladder for 24 hours to 7 days following a fatty meal, and was regarded as prolonged visualization.

Fourteen cases were studied in the original group. Each of these patients had 1 or more previous oral cholecystograms with a fatty meal and were reported as normal. These patients were treated medically, but had recurrent attacks with symptoms of gallbladder colic. Chronic cholecystitis was found in each case following cholecystectomy. Postoperative results were good in 12 cases, and fair in 2 others.

In a second series of 19 cases, cholecystokinin was used in patients with persistent and recurrent gallbladder disease, in whom oral cholecystography was normal. Cholecystokinin, 75 Ivy units, was injected intravenously after a resting roentgenogram of the gallbladder following ingestion of 3 gm. iopanoic acid the previous evening. Serial roentgenograms were made during the next 3 days. Less than 50 per cent contraction of the gallbladder on a 20 minute postinjection roentgenogram was considered an abnormal volume change. Other criteria indicating an abnormal response were: recurrence of symptoms; abnormal contractions in the neck or body of the gallbladder; and prolonged visualization of the gallbladder (36–108 hours in the positive cases).

Ten patients showed an abnormal cholecystokinin cholecystogram, and subsequently had a cholecystectomy. The histologic findings in these cases were: chronic cholecystitis (7); focal cholesterolosis (1); papilloma and cholesterolosis (1); and a normal gallbladder in 1 case.

The authors recommend 24 and 48 hour postcibal roentgenograms to evaluate prolonged visualization of the gallbladder. Cholecystokinin cholecystography is advised in symptomatic patients with a so-called normal oral cholecystogram.—*Paul M. Dassel, M.D.*

Cotton, P. B., and Beales, J. S. M. Endoscopic pancreatography in management of relapsing acute pancreatitis. *Brit. M. J.*, March, 1974, *1*, 608–611. (Address: P. B. Cotton, M.D., Middlesex Hospital, London W. 1, England.)

Endoscopic retrograde cholangiopancreatography (E.R.C.P.) has become a useful procedure in patients with jaundice and in those with pancreatic disease. This study was limited to 31 patients suffering from repeated attacks of acute pancreatitis. Only 5 were women, and 15 of the patients used alcohol rarely if at all. Seven patients regularly drank more heavily.

Pancreatograms were obtained in 25 of the 31 patients. Normal or minor "non-surgical lesions" were found in 13 patients. Twelve patients had major "surgical lesions." These included 4 with complete obstruction of the main pancreatic duct, 3 with stricture and pseudocyst, and 5 with juxtapapillary stricture. These findings seemed quite useful in determination of type of management to be offered these patients.

A normal pancreatogram does not rule out carcinoma and normal pancreatograms may be obtained even during acute pancreatitis.

E.R.C.P. is not a simple diagnostic test. The examiner needs much experience with the fiberoptic

endoscope and roentgenograms of good quality must be obtained. This is strictly a hospital procedure and should be carried out in the X-ray Department. An Olympus JFB duodenoscope was used during this study.—*Richard E. Kinzer, M.D.*

Miller, Thomas A., Lindenauer, S. Martin, Frey, Charles F., and Stanley, James C. Pancreatic abscess. *A.M.A. Arch. Surg.*, April, 1974, *108*, 545–551. (Address: S. Martin Lindenauer, M.D., Veterans Administration Hospital, 2215 Fuller Road, Ann Arbor, Mich. 48105.)

The authors have reviewed the medical records of 63 proved cases of pancreatic abscess occurring at the University of Michigan Medical Center hospitals over the past 36 years

There were 17 postoperative deaths among the 46 patients undergoing surgery for pancreatic abscess. Death occurred in all 17 cases in which pancreatic abscess was unsuspected and untreated. There were 47 males and 16 females. The average age was 46 years, but varied from 18 to 77 years.

Acute pancreatitis preceded abscess formation in 61 cases. Usually there was an initial response to acute pancreatitis with conservative management, but with recurrence of symptoms 10 days to 3 weeks later. Abscess formation was frequently misinterpreted as recurrent pancreatitis. In 15 per cent of the cases there was progression to abscess formation without an intermediate remission.

Conditions leading to pancreatitis in this series include: recent upper abdominal surgery; biliary tract disease; alcoholism; and a variety of uncommon factors.

The triad of abdominal pain, tenderness in the region of the abscess, and unexplained fever was important in establishing the diagnosis. An elevated leukocyte count was the most consistent abnormal laboratory finding.

Roentgenographic abnormalities were helpful in 87 per cent of these cases. Gas within the abscess was demonstrated in 10 patients. Other roentgenographic findings were evidence of subphrenic inflammation (35), and of an abdominal mass (18). Mass lesions presented as: displacement of the stomach; gastric outlet obstruction secondary to extrinsic compression; widened duodenal loop; and obliteration of the psoas muscle shadows.

The most common organisms found on cultures grown from the abscesses were *Escherichia coli*, *Klebsiella*, *Proteus* and *Staphylococcus aureus*.

Ultrasound was useful as a diagnostic procedure in 3 cases.

The most frequent complications of pancreatic abscess contributing to death were: pleural effusion; pneumonia; bleeding; renal failure; enteric fistula; and recurrent abscesses.—*Paul M. Dassel, M.D.*

Genitourinary System

Moreau, J.-F., Kleinknecht, D., Lachand, A.-T., and Michel, J.-R. Radiologie de l'insuffisance rénale aiguë. (Radiology of acute renal insufficiency.) *La Nouvelle Presse méd.*, Jan., 1974, *3*, 201–204. (Address: J.-F. Moreau, Hôpital Necker, 161, rue de Sèvres, F 75730 Paris Cedex 15, France.)

The main problem in acute renal insufficiency is to search for, determine, or rule out, the presence of urinary tract obstruction as an etiologic factor.

The contrast media which are presently used offer a wide margin of safety—acute renal insufficiency is no contraindication to their use.

High dose excretion urography with tomography should be adequate to outline and delineate the urinary tract and demonstrate the site of obstruction, if present. This dispenses with the need for retrograde urography and the hazards such a procedure carries. Additional radiologic studies, which may be needed to arrive at a diagnosis, include inferior vena cava venography and renal arteriography.

The authors use a dose of 2.5 ml./kg. of sodium and methyl glucamine diatrizoate or ioxithalamate. Routine standard projections are used; delayed films up to 24 hours are often necessary to adequately demonstrate the dilated portion of the urinary tract and the obstruction which has resulted in impairment of the renal excretory function.

Vicarious excretion of the contrast medium occurs through the liver and digestive tract eliminating 30–40 per cent of the contrast medium. This may be supplemented by a 12 hour session of hemodialysis which could eliminate up to 90 per cent of the administered dose of the contrast medium.

Six excellent reproductions of roentgenograms of urography, inferior vena cava venography, and renal arteriography, accompany this interesting article.—*William H. Shehadi, M.D.*

Cooke, G. M., and Bartucz, J. P. Spontaneous extravasation of contrast medium during intravenous urography: report of fourteen cases and a review of the literature. *Clin. Radiol.*, Jan., 1974, *25*, 87–93. (From: The Department of Diagnostic Radiology, St. Michael's Hospital, 30 Bond Street, Toronto 2, Ontario, Canada.)

Spontaneous extravasation refers to the radiologic demonstration of contrast material outside the renal collecting system with no demonstrable rupture of the kidney or pyelocalyceal system. To fulfill the requirements of this condition, there must have been no recent ureteric instrumentation, no previous surgery, no destructive renal lesion, no external compression, and no pressure necrosis of the ureter or pelvis by a calculus.

The condition is rare, the incidence being less than 1 in 1,000. Fourteen cases are reported, 11 of which involved the left kidney. Twelve were males all of adult ages. Sudden onset of flank pain was the predominant symptom.

The authors believed that a sudden increase in intrapelvic pressure was the basic etiologic factor. The site of rupture was not always apparent, but in some cases rupture through a middle or lower pole calyx was clearly visualized.

Cases have been reported in which peripelvic abscesses have developed, but it is concluded that spontaneous extravasation *per se* associated with renal colic is a relatively benign process.

Treatment is essentially supportive, and retrograde pyelography is usually unnecessary.—*Arch H. Hall, M.D.*

HATELY, WILLIAM, and WHITAKER, ROBERT H. How accurate is diagnostic ultrasound in renal disease? *Brit. J. Urol.*, Oct., 1973, *45*, 468–473. (From: Departments of Diagnostic Radiology and Urology, The London Hospital, London E1 2AD, England.)

None of the currently available methods of examining the prostate gland is entirely satisfactory for determining the total size and producing a complete survey of the diseases of the gland. For this reason, investigation of the possibilities of sonography was made. Out of a total of 66 patients, satisfactory tomographic sonograms were obtained in 60.

In 10 patients with benign hyperplasia and subsequent operation, the over-all dimensions, as shown surgically and by sonography, correlated well. In the presence of intracapsular, small carcinomas, false negative results were encountered frequently, although occasionally subtle variations in sound reflection suggested the presence of the neoplasm. When there was an invasive, extracapsular malignancy, several abnormalities in the sonogram were observed. The most common and characteristic finding was distortion and deformity of the prostatic contour, caused by extension of the malignant tissue beyond the fibrous capsule. The method was accurate in 77 per cent of these patients. In addition, the acoustical interfaces between normal and malignant tissue often produced greater attenuation of the ultrasonic beam, requiring higher receiver gains for definition. Further, larger, irregular echo patterns were encountered at these interfaces. Prostatic calculi simulated the changes due to intracapsular malignancies, but not the deformity of the capsule.

In inflammatory disease there are few changes in the reflectivity of the gland parenchyma, but in 2 cases the entire prostate was displaced from the colon by a sonolucent process histologically classified as granulomatous prostatitis.

The examination is carried out by introducing a shielded transducer into the rectum. The transducer is surrounded by degassed water contained in a thin rubber condom, which is inflated to fill the rectum, distending its walls and eliminating any air pockets. A series of patterns, made at 1 cm. increments beginning at the level of the bladder and seminal vesicles, produces cross sections of the gland and surrounding structures. The entire examination requires about 30 minutes and is painless.—*E. Frederick Lang, M.D.*

KING, WILLIAM W., WILKIEMEYER, R. MARK, BOYCE, WILLIAM H., and MCKINNEY, WILLIAM M. Current status of prostatic echography. *J.A.M.A.*, Oct., 1973, *226*, 444–447. (Address: William W. King, M.D., Department of Urology, University of Tennessee Medical Center, 800 Madison Avenue, Box 635, Memphis, Tenn. 38103.)

A series of 66 patients with urologic problems was investigated by sonography in conjunction with other methods of examination, and in 61 cases a definite opinion was expressed from the sonar findings and the final true situation became known. The accuracy rate was 87 per cent. The diagnosis was proved occasionally by operation, and frequently by puncture, aspiration, and contrast study, in the case of a cyst.

In 31 cases a cyst was confidently and correctly identified. In another, multiple cysts were correctly identified, but in 1, multiple cysts were found after a sonographic diagnosis of a solid tumor. Five patients had correctly diagnosed tumors, proved surgically. There were 8 patients with no excretion by intravenous urography, and all were correctly classified—4 with tumors, and 4 with hydronephrosis, although the basis for the obstruction could not be classified until operation.

The most serious error occurred when a mass lesion was thought to be cystic by sonography, but arteriography subsequently showed a vascular tumor. At the time of operation, the kidney was found to be partially cystic. In another patient a large pelvic tumor, which was readily demonstrated by intravenous urography, was not shown by sonography. This situation is in agreement with statements in the literature. Another error occurred when a small, scarred kidney was removed after being called hydronephrotic by sonography. In 1 patient, in whom sonar examination showed what appeared to be hydronephrosis, a perinephric abscess resulting from rupture of the renal pelvis in association with calculous obstruction was encountered at operation. In 3 patients sonograms did not disclose the presence of small (20 to 30 ml.) cysts; those cysts which were demonstrable were at least 50 ml. in capacity.

Since only 1 in 20 renal masses found at excretory urography is a carcinoma, it is highly desirable to avoid surgical exploration and even vascular catheterization, if possible, while keeping the examinations as safe and simple as is consistent with ac-

curacy. In the symptomatic patient with a normal excretory urogram, ultrasonic examination has little, if anything, to add. The main contribution of ultrasound is in the management of surgical problems of the kidney.—*E. Frederick Lang, M.D.*

Skeletal System

Basu, S. P. Bone changes in leprosy: Dr. Diwan Chand Aggarwal Memorial Oration for 1972. *Indian J. Radiol.*, Nov., 1972, *26*, 239–249. (Address: "Smriti," No. 728, Block "P," New Alipore, Calcutta-53, India.)

A group of 140 patients with leprosy was examined by conventional roentgenography, arteriography, venography, and lymphangiography. Of the total group, 18 were of the *lepromatous* variety, and 90 were *tuberculoid*.

In the patients with *lepromatous* leprosy a common bone abnormality is osteoporosis, probably due to the presence of the bacillus in the medulla. This is occasionally associated with erosion of the medullary border of the cortex, and this may progress to expansion of the cortex. The other manifestations of infection by the leprosy bacillus are enlarged nutrient foramina, honeycombing of the bone, and, most characteristic, bone cyst formation. These cysts are clear cut regions of destruction, and are seen in the medullary regions of small bones of the hands and feet. Subarticular bone erosion is the most common bone complication and begins at the articular margins and is usually associated with extensive soft tissue swelling. Without vigorous treatment the joint space soon becomes involved, the joint disintegrates, and deformity develops.

In patients with *lepromatous* leprosy the bone changes are caused by direct invasion of the bone. Needle biopsy was carried out in 7 of these cases and was positive in 4. Occasionally, sequestra develop in this type of involvement, and examination of one such fragment showed complete replacement of all cellular structure by calcific material.

Among the 90 patients with *tuberculoid* leprosy, the type of bone involvement was much more varied. In this variety, the bone changes are not due to direct invasion, but are the result of invasion of the nerves with subsequent nervous and vascular trophic disturbance. Needle biopsy in 12 of these cases yielded no positive findings. In general, the bone changes result from anesthesia and uncontrolled trauma, from infection in the anesthetized soft tissues, and from neurotrophic disturbance—the Charcot joint.

The changes observed are all nonspecific: distal tuft erosion, the most common; joint contractures; absorption of distal phalanges either in association with infection or with obliterating endarteritis; development of "pin and cup" joints, indistinguishable from those seen in collagen diseases; and amputation. In most of these varieties of bone involvement there is no osteoporosis, and occasionally this feature is striking. Most of the involvement is in the lower extremities, and occurs as a result of unnoticed trauma, infection, ulceration of the foot, and subsequent bone change.

In the 140 patients, the average duration of the disease was 18 years. The ages ranged between 16 and 52 years. All but 53 had had sulphone therapy for varying periods. Although the lower extremity was the part most frequently involved, other affected sites were the bones of the face, including the nose.—*E. Frederick Lang, M.D.*

Jacobs, Philip. Osteolytic Paget's disease. *Clin. Radiol.*, Jan., 1974, *25*, 137–144. (From: X-ray Departments, Birmingham Accident Hospital and Royal Orthopaedic Hospital, Birmingham, England.)

An unusual form of Paget's disease involves a large localized zone of bone destruction in long bones, similar to the well known osteoporosis circumscripta in the skull. An early osteolytic, monostotic lesion in a young person will cause diagnostic difficulties. The lesion represents an early, active stage of Paget's disease in a long bone. Later, consolidation occurs, and classical radiologic signs will be found.

Three well known signs of Paget's disease are particularly useful in reaching the correct diagnosis in this type of long bone lesion: (1) the almost invariable tendency for one end of the lesion to be subarticular; (2) enlargement of the affected bone; (3) the flame-shaped or V-shaped boundary between the normal and affected bone.

In the 5 case reports presented, 2 lesions were in the radius, 1 in the ulna, 1 in a proximal phalanx, and 1 in the pelvis.

If a solitary osteolytic lesion is seen in a long bone, a bone survey could be most helpful in arriving at the correct diagnosis, especially if classical Paget's disease was found elsewhere.—*Arch H. Hall, M.D.*

McElfresh, Edward C., and Coventry, Mark B. Femoral and pelvic fractures after total hip arthroplasty. *J. Bone & Joint Surg.*, April, 1974, *56-A*, 483–492. (Address: Edward C. McElfresh, M.D., c/o Section of Publications, 200 First Street, S.W., Rochester, Minn. 55901.)

The artificial hip joint may withstand considerable trauma. This report from the Mayo Clinic, however, reviews 11 fractures following total hip replacement occurring 4 to 20 months after operation; 3 pubic, 6 femoral, and fractures of both the acetabulum and femur in 1 patient.

These fractures can be placed in 3 groups: 1. Stress fractures apparently related to increased activity following hip replacement. There were 2 patients with pubic fractures unrelated to trauma. 2. Surgical defects in the cortex or insufficient cement around the tip of the femoral component, with a minor injury causing fracture. One patient

twisted her leg and fractured the femur at a point where the cement extruded through the shaft. Another patient stepped off a curb and fractured near a screw hole left from a removed plate and screws. 3. Trauma sufficient to produce fractures in a normal limb. Four patients had a fall or auto accident with resultant shaft fractures; 1 fractured the pubic ramus and 2 had both the femoral shaft and acetabulum fractured.

The authors found that fractures adjacent to the methylmethacrylate cement healed normally, if in good position.

Several suggestions are made which may prevent these fractures. In patients who have been disabled for some time or have osteoporosis, pubic stress fractures might be avoided by a postoperative rehabilitation program. In patients with defects in the femoral cortex from removed screws or penetration of the cortex at the time of surgery, it is suggested that a long stem prosthesis with adequate cement be used and care be taken that the cement does not protrude through a cortical defect. They also indicate that the cement should extend at least 2 cm. distal to the tip of the prosthesis.

Closed treatment of fractures is preferred since the prior surgery with reaming and heat may predispose to avascularity.

The case histories include 18 roentgenographic representations and there is discussion of therapy.—*Martha Mottram, M.D.*

Mirra, Joseph M., and Marcove, Ralph C. Fibrosarcomatous dedifferentiation of primary and secondary chondrosarcoma: review of five cases. *J. Bone & Joint Surg.*, March, 1974, *56-A*, 285–296. (Address: Joseph M. Mirra, M.D., The University of California at Los Angeles, Center for Health Sciences, Los Angeles, Calif. 90024.)

Review of 27 cases of well-differentiated chondrosarcoma at The University of California, Los Angeles, showed that 5 developed fibrosarcomatous dedifferentiation with no evidence of osteoid formation. Three of the chondrosarcomas developed in enchondromas, 1 in Ollier's disease, and 1 was a primary lesion

The presenting symptom in all cases was pain and tenderness without a history of trauma. Three patients had swelling of the affected limb and 2 of these had extension of tumor into the soft tissues. All patients had either elevated fasting blood sugar or abnormal glucose tolerance tests.

The mean age when fibrosarcoma was diagnosed in the chondrosarcoma was 57 years. Three tumors were in the femur and 2 in the humerus.

The 5 cases are reported in detail with roentgenographic reproductions and photomicrographs. The lesions showed multiple fine or coarse granular and linear calcifications with irregular lytic defects. The cortex was quite irregular in the illustrated cases. One roentgenogram showed Codman triangles. In areas of cortical bone destruction the tumor extended into the soft tissues. One patient had a pathologic fracture.

The gross and microscopic appearance was indicative of underlying enchondroma in 4 cases, with heavy calcification in the shaft and anaplasia in the chondrosarcomatous portions; 1 fibrosarcoma arose in a primary well-differentiated chondrosarcoma.

Pathologic examination showed that the chondrosarcoma and fibrosarcoma were closely associated; in some areas there was sharp delineation between them, and in others a gradual transition.

There is usually a highly malignant course after fibrosarcomatous dedifferentiation and widespread metastases developed in 4 of 5 patients within a year; these were spindle-cell or fibrosarcomatous lesions without osteoid, bone or cartilage formation.

In the patient with Ollier's disease there had been excision of a well-differentiated chondrosarcoma of the femoral neck with hip fusion at the age of 33 years. At the age of 52 years she developed chondrosarcoma in the distal femur, separate from the old lesion; there were several small foci of fibrosarcomatous dedifferentiation in the lower shaft. This patient had a more protracted course and died of generalized metastases within 5 years.—*Martha Mottram, M.D.*

Barrier, J., Grall, F., and Vigneron, N. Ostéite pubienne et thrombo-phlébite du post-partum. (Pubic osteitis and postpartum thrombophlebitis.) *J. de gynécol., obstét. et biol. de la repr.*, Sept., 1973, *2*, 737–743. (Address: Dr. J. Barrier, Hôpital Louis Mourier, Faculté Xavier Bichat, 178, rue des Renouillers, F. 92700 Colombes, France.)

Postpartum nonsuppurative acute pubic osteitis starts as a septicemia followed by a severe pain localized in the pubis.

Roentgen findings vary and the most important diagnostic signs are sensitivity to intensive antibiotic treatment and regression of the infection as monitored by sedimentation rate studies.

The 2 cases of acute pubic osteitis treated by the authors, developed pelvic thrombophlebitis subsequently. This is believed to be due to the same postpartum septicemia (anaerobic *Streptococci*). Hence, the advice is to be alert to detect early clinical signs of postpartum pelvic thrombophlebitis so as to start anticoagulant therapy in addition to antibiotics.—*Jirair N. Sarian, M.D.*

Blood and Lymph System

Pinet, F., Duquesnel, J., Amiel, M., Froment, J.-C., Clermont, A., and Rubet, A. Les dissections, fissurations et ruptures de la

paroi aortique: apport de l'aortographie par voie artérielle; à propos de 69 observations. (Dissections, fissurizations and ruptures of the aortic wall: contribution of aortography by arterial route; report of 69 observations.) *La Nouvelle Presse méd.*, March, 1974, *3*, 797–800. (Address: Pr F. Pinet, Hôpital Cardio-Vasculaire, B.P. Lyon Montchat 69394 Lyon Cedex 3, France.)

Based on their experience in 102 cases of dissection and 2 cases of rupture, the authors conclude that retrograde aortography *via* the femoral artery is the procedure of choice for opacification of the aorta.

They noted a preponderance of male patients (68 per cent) in their series. The age distribution was between 26 and 90, the majority being between 40 and 70 years of age.

Retrograde femoral arteriography (Seldinger technique) is performed whenever the femoral pulse is palpable. In the absence of a femoral pulse, percutaneous axillary arteriography is made.

The position and progress of the catheter should be carefully checked using test injection of a small amount of contrast material, thus avoiding entering a false channel or creating one in the aortic wall.

There were no noteworthy complications as a result of this procedure. The risks in performing the examination are minimal, compared with the value of the information gained.

In determining the type and site of dissection of the aorta, the authors use the classification of DeBakey, which is as follows:

1. The orifice or point of entry is at the ascending aorta, with the dissection extending beyond the bronchocephalic trunk, and which may involve the entire aorta (38 per cent)

2. Dissection limited to the ascending aorta (19 per cent)

3. The orifice, or point of dissection, is beyond the level of the left subclavian artery with dissection extending beyond the level of the diaphragm (18 per cent).

The authors add 4 types of their own based on the false channel observed in 74 cases. These are:

1. Intermediate location between DeBakey 1 and 2 (15 per cent)

2. Dissection beginning in the arch at the level of the great vessels (3 per cent)

3. Orifice located below the left subclavian artery, but with retrograde dissection up to the arch of the aorta (4 per cent)

4. Abdominal—the primary dissection is limited to the abdominal aorta (3 per cent).

Reproductions of 5 excellent roentgenograms and drawings accompany this interesting article.—*William H. Shehadi, M.D.*

Highman, J. H., and O'Sullivan, E. Isotope venography of pelvic veins. *Clin. Radiol.*, Jan., 1974, *25*, 125–143. (From: Department of Radiology, St. Mary's Hospital, London W. 2, England.)

Evaluation of the pelvic venous system is of value in patients with chronic varicose veins or leg edema. X-ray phlebography is accepted as the most accurate method of demonstrating the venous system of the pelvis and extremities. Radioisotopic examination of the pelvic venous system and lower extremities has had limited use.

Twenty-five cases with chronic varicose veins and leg edema were studied by isotope venography and the results were compared with x-ray phlebography in the same cases. A dose of 2 mc Tc^{99m} was injected simultaneously into the dorsum of each foot, and, subsequently, recordings were made over the pelvis using a gamma camera.

Abnormalities in the images included: (1) absence of normal vessels; (2) collateral channels, with the most common being a retropubic anastomosis; and (3) delayed isotope flow.

There were several cases in which the x-ray phlebogram appeared normal, but there was a delay in appearance of the isotope in the left iliac vein. The authors did not attach much significance to this in the absence of additional evidence of venous occlusion.

The pictorial definition of the isotope venograms was not as good as the x-ray phlebogram, but any lesion which affects the flow causing delay or obstruction with the formation of collaterals will be excellently demonstrated. The upper iliac vessels and the inferior vena cava are routinely demonstrated.

Isotope venography is recommended as a rapid and accurate outpatient screening procedure for the evaluation of the status of the pelvic venous system.—*Charles W. Cooley, M.D.*

Bloom, John D., Mozersky, David J., Buckley, Clifford J., and Hagood, Clyde O., Jr. Defective limb growth as a complication of catheterization of the femoral artery. *Surg., Gynec. & Obst.*, April, 1974, *138*, 524–526. (From: The General Surgery Service, Wilford Hall U. S. Air Force Medical Center, Lackland Air Force Base, Texas.)

Because of advances in cardiac surgical techniques, increasing numbers of young patients are being subjected to retrograde femoral artery catheterization procedures.

Although statistics vary widely, complications do occur. Three cases are detailed in which the common femoral and external iliac arteries became occluded and went unrecognized until retarded growth developed in the involved limb. The defects were cor-

rected by means of modified bovine arterial heterografts with good results.

The effect of altered blood flow on limb development is well documented, and the importance of a prolonged follow-up period after arterial catheterization is recommended. The presence of a palpable pulse and a warm extremity cannot be used to exclude arterial thrombosis unequivocally.

Adjunct methods of evaluation include: exercise testing; the use of an ultrasonic velocity detector; ankle pressures; and finally, arteriography.

In the 3 cases presented, retardation in limb growth was the first sign of arterial occlusion.—*Arch H. Hall, M.D.*

Henkin, Robert E., Yao, James S. T., Quinn, James L., III, and Bergan, John J. Radionuclide venography (RNV) in lower extremity venous disease. *J. Nuclear Med.*, March, 1974, *15*, 171–175. (Address: Robert E. Henkin, M.D., Department of Nuclear Medicine, Northwestern Memorial Hospital, Superior Street and Fairbanks Court, Chicago, Ill. 60611.)

The importance of lower extremity and pelvic venous thrombosis as the source of pulmonary embolism is well known, as are the dire results of pulmonary embolism. Contrast venography, ultrasound and the radioiodinated fibrinogen uptake have been used to study the problem.

The authors studied 53 cases with lower extremity venous disease or pulmonary embolism. Ultrasound studies were done on 50 per cent of the cases and 25 cases had contrast venography. Radionuclide venography (RNV) was done in the 53 cases using Tc^{99m}-human albumin microspheres and a gamma camera. The RNV was found to be more accurate than the ultrasound, and when combined with the ultrasound study there were no false negatives. There was 1 false negative and no false positives in the patients who had both RNV and contrast venography studies. Fifty-three per cent of the patients with pulmonary emboli and clinically normal lower extremities had abnormal RNV studies.

Vascular occlusion, collateral circulation and thrombi are accurately identified by RNV.

The RNV test is safe, accurate, easily performed and is useful in evaluating patients with pulmonary embolism.—*Charles W. Cooley, M.D.*

Nikaidoh, Hisashi, Idriss, Farouk S., and Riker, William L. Aortic rupture in children as a complication of coarctation of the aorta. *A.M.A. Arch. Surg.*, Dec., 1973, *107*, 838–841. (Address: Dr. Hisashi Nikaidoh, 2300 N. Children's Plaza, Chicago, Ill. 60614.)

The natural history of undiagnosed or untreated cases of coarctation of the aorta of the adult type is death, on the average before the age of 35 years, due to a complicating rupture of the aorta, bacterial endocarditis or endo-aortitis, congestive heart failure, or intracranial hemorrhage.

Early diagnosis and surgical treatment removes the life threatening aspects of coarctation.

The authors report that at their hospital 325 patients with coarctation of the aorta have been treated surgically since 1946. During this period there were 3 cases in children of aortic rupture associated with the coarctation. Two of these involved mycotic false aneurysms secondary to bacterial endo-aortitis, and the other was a dissecting aneurysm of the ascending aorta which ruptured into the pericardial cavity.

Aortic rupture without infection appears to be more common than the one induced by infection, and occurs on the average in the third decade of life.

The case being reported by the authors was a 7 year old boy, probably the youngest reported, and this may have been related to the blunt trauma the patient sustained falling from the front steps of his home. This usually takes the form of a dissecting aneurysm secondary to hyaline degeneration and a decrease in elastic tissue in the media of the wall of the aorta near the site of rupture. The ascending aorta is usually considered to be the most common location, but others have found an equal distribution in the pre-stenotic and post-stenotic segments of the coarctation. The dissecting aneurysm eventually ruptures into the pericardial sac, pleural cavity, esophagus or bronchial lumen.

Aortic rupture secondary to bacterial infection is due to a mycotic false aneurysm caused by direct bacterial invasion of the aortic wall from endo-aortitis. This occurs more frequently in the post-stenotic segment and the 2 major organisms are *Staphylococci* and *Streptococci*. These mycotic aneurysms usually progress to rupture into the pleural cavity, the esophagus, or the bronchial lumen. These commonly occur in boys in the second decade, with the ages of the 2 boys being reported, however, being $5\frac{1}{2}$ and $10\frac{1}{2}$ years, respectively.

Clinically, the symptoms and physical findings are those due to subacute or acute bacterial infection, with pyogenic emboli to those organs and extremities below the level of the coarctation.

The chest roentgenograms show the typical findings of coarctation, and, in addition, a rapidly expanding soft tissue density in the left hilus.

Surgically, the major technical difficulty is the frequent sudden uncontrollable hemorrhage which may occur early during the dissection through rupture of the aneurysm. For this reason the false aneurysm and the surrounding structures should not be disturbed, until measures such as extracorporeal circulation, hypothermia and circulatory arrests are taken to manage massive bleeding from the aorta.—*Donald M. Monson, M.D.*

Bliznak, Johnny, and Staple, Tom W. Radiology of angiodysplasias of the limb. *Radiology*, Jan., 1974, *110*, 35–44. (Address: Johnny Bliznak, M.D., Mallinckrodt Institute of Radiology, Washington University School of Medicine, 510 South Kingshighway Boulevard, St. Louis, Mo. 63110.)

This report deals with a series of 18 patients studied at the Mallinckrodt Institute of Radiology between 1963 and 1972. Plain film roentgenograms were reviewed with regard to a soft tissue mass and soft tissue calcifications. Comparisons were made of the length, diameter, periosteal reaction, bone erosion and remodelling patterns, and the growth and development of the affected extremity. The findings were found to be valuable in determining the extent of the lesions, as filling of the vessels at angiography sometimes was found to be incomplete. Soft tissue calcifications were present in one half of the patients studied, and were found more commonly in lesions with arterial components.

The angiographic classification devised by the authors was found helpful to the surgeon in the preoperative approach to these lesions and is listed as follows: (a) local arteriovenous malformations; (b) diffuse arteriovenous malformations; (c) small vessel malformations; and (d) venous malformations.

a. *Local arteriovenous malformations* were usually associated with considerable hemodynamic changes as exemplified by loud bruits (indicating rapid flow), and shunting. Arteriography was considered mandatory in these patients. The filming sequence necessary to demonstrate these lesions consisted of 2 exposures per second for a minimum of 10 seconds.

b. *Diffuse arteriovenous malformations* were noted to usually be extensive, requiring a large field film changer to demonstrate. The striking finding in these lesions was that of greatly dilated veins, resulting in a clinical varicose appearance. Hence, a diffuse vascular malformation should always be considered in the differential diagnosis of a patient with varicose veins and gangrene of the tips of the digits.

c. *Small vessel malformations* were often accompanied by the development of adjacent periosteal new bone formations. These lesions were best demonstrated by arterial injection using a rapid film changer. Late films were also necessary, as the malformations typically were best demonstrated in the capillary phase, and additionally were characterized by the appearance of large draining veins in the venous phase.

d. *Venous malformations* in this series ranged in size from 2 cm. to extensive lesions involving an entire limb. They were characterized by extremely slow flow, so that arteriography was not often helpful, adequate visualization being defeated by the dilution effect and the fact that some feeding vessels may be thrombosed as well. Venography was considered mandatory and often multiple simultaneous injections at different sites were necessary to demonstrate the lesion.

In summary the authors concluded that the presence of a bruit, digital gangrene with or without varicose veins, or roentgen evidence of vascular calcification was much more common in arterial malformations than in venous malformations, and indicated that the lesion had an arterial component. Any patient suspected of having a vascular malformation despite a normal arteriogram should undergo venography. If the venous site cannot be visualized from the arterial side, either the feeding vessels and/or many of the venous channels are thrombosed or the large volume of blood in the malformation is causing dilution of contrast material delivered from the arterial side. If venography fails to demonstrate the lesion, direct injection into any palpable portion of the malformation should demonstrate vascular spaces, often to a rather surprising extent. Venous dysplasias were best demonstrated by venography. Arteriography was considered essential in patients with varicose veins due to arteriovenous malformations. Surgical results have been best in those patients with localized lesions. However, even in these cases, infiltration of muscles vital to function may leave the patient with a useless limb. Small vessel lesions with extensive infiltration into a group of muscles tend to recur at the border of the resection and extend proximally from this point. Angiographic demonstration of the entire course of these vessels not only helps in planning surgery, but also provided a good indication as to the prognosis.—*David E. Marley, M.D.*

SUBJECT INDEX TO VOLUME 121

(E) = editorial (ab) = abstract

ABDOMEN, forgotten operative fields in: radio-clinical study (ab), 211
ABDOMINAL organs, three-dimensional imaging of, with ultrasound, 883
ABSCESS, pancreatic (ab), 917
tubo-ovarian; spectrum of radiologic abnormalities due to (ab), 702
ADRENAL tumors simulating intrarenal lesions, 518
ALPORT'S syndrome, hereditary nephritis, angiographic features of, 539
AMERICAN RADIUM SOCIETY, Fifty-sixth Annual Meeting of (E), 890
Heuristic gems from (E), 653
AMERICAN ROENTGEN RAY SOCIETY, Preliminary Program, 436
Section on Instruction, 661
Seventy-fifth Annual Meeting of (E), 431
ANAL canal, large diverticulum of; case report and review of literature on anal canal and rectal diverticula, 344
ANEURYSM, dissecting; with particular reference to lower intercostal and renal arteries; branch involvement at aortography in (ab), 219
ANEURYSMS, abdominal aortic; continuous ultrasound "B" scanning of, 626
dissecting, of aorta; roentgenographic evaluation of, 617
ANGIODYSPLASIAS of limb; radiology of (ab), 923
ANGIOGRAM, renal, in tuberculosis (ab), 460
ANGIOGRAPHIC anatomy, left ventricular, of ostium primum defect in adult, 597
demonstration of absence of atrial septal defect in presence of partial anomalous pulmonary venous connection, 591
diagnosis and treatment by closed aspiration; pyogenic liver abscess, 388
features of Alport's syndrome; hereditary nephritis, 539
study; intrahepatic arterial portal communications, 384
ANGIOGRAPHY; acute renal failure after, 544
cinecoronary, improved accuracy of, angulated views in sagittal plane for, 565
in unilateral renal bleeding with negative urogram, 508
negative, reliability of; in clinically suspected splenic rupture (ab), 700
of liver and spleen in Hodgkin's disease, 789
of upper extremity; pharmacologic aids in, 150
recognition without; pericapsular renal veins (ab), 462
selective, of systemic arterial supply to lungs in planning surgical correction of pseudotruncus arteriosus, importance of, 606
selective pharmacodynamic, in diagnosis of carcinoma of pancreas (ab), 466
AORTA; aortic rupture in children as complication of coarctation of (ab), 922
roentgenographic evaluation of dissecting aneurysms of, 617
AORTOGRAPHY by arterial route, contribution of: dissections, fissurizations and ruptures of aortic wall; report of 69 observations (ab), 921
branch involvement at, in dissecting aneurysm, with particular reference to lower intercostal and renal arteries (ab), 219
double catheter technique in, 634
APPENDIX, normal; frequency of visualization of, with barium enema examination, 312
ARCUATE ligament, syndrome of (ab), 703
ARTERIAL, intrahepatic, portal communications; angiographic study, 384
systemic, supply to lungs in planning surgical correction of pseudotruncus arteriosus; importance of selective angiography of, 606
ARTERIOGRAPHIC differentiation of intrahepatic and extrahepatic masses; pitfalls in, 420
signs of primary hyperparathyroidism; report of 10 cases (ab), 457
symptoms of renal tuberculosis (ab), 701
ARTERIOGRAPHY and phlebography of limbs in planning shunt and fistula operations in patients with chronic renal failure, 551
coronary and cardiac catheterization; radiation reduction by means of low pulse-rate fluoroscopy during, 636
coronary, in left main coronary artery disease, 583
"lower" gastrointestinal tract hemorrhage: new concepts based on, 357
selective bronchial, in hemoptysis (ab), 915
ARTERIOSUS, pseudotruncus; importance of selective angiography of systemic arterial supply to lungs in planning surgical correction of, 606
ARTERY, femoral; defective limb growth as complication of catheterization of (ab), 922
maxillary, in craniofacial pathology, 124
ARTHROGRAPHY in rupture of supra-patellar bursa with pseudocyst formation, 89
knee, synovial recesses in, 86
of lateral meniscus, 79
ARTHROPLASTY, total hip; femoral and pelvic fractures after (ab), 919
ASPIRATION cannula, new, introduction of, and review of 541 consecutive studies; painless myelography (ab), 216
Aspergillus fumigatus, its pathogenic action on lung (ab), 210
ATRIAL septal defect in presence of partial anomalous pulmonary venous connection; angiographic demonstration of, 591

Barium enema examination; frequency of visualization of normal appendix with, 312
study, water double contrast; new method of roentgen examination of colon, 326
Barrett's esophagus, diagnosis of, with radioisotopes, 716
Battered child syndrome; patterns of injury and significance of uncommon fractures in, 143
Benign hemorrhagic renal cyst; nephrotomography, renal arteriography, and cyst puncture (ab), 461
Biliary calculi, residual, extraction of; in x-ray department (ab), 212
Biopsy needle, new, and its clinical use, 854
percutaneous needle localization of clustered mammary microcalcifications prior to, 839
precision needle, of breast at mammography; device for, 843
Bladder, uroepithelial tumors of, calcification in; report of 5 cases and survey of literature, 827
Blastomycosis, pulmonary, acute; clinical features of (ab), 698
Bone changes in leprosy: Dr. Diwan Chand Aggarwal Memorial Oration for 1972 (ab), 919
demineralization studies, roentgenographic, of Gemini space flights; review of, 1
imaging; comparison of Tc^{99m} polyphosphate and F^{18} for, 184
similar, manifestations of scleroderma and rheumatoid arthritis, 35
Book Reviews
The Radiology of Joint Disease. By D. M. Forrester, and John W. Nesson, 200
Complications of Diagnostic Radiology. By John F. Weigen, and Sydney F. Thomas, 200
Radiology of the Newborn and Young Infant. By Leonard E. Swischuk, 446
Coronary Angiography. By Harold A. Baltaxe, Kurt Amplatz, and David C. Levin, 687
The Aortic Arch and Its Malformations: with Emphasis on the Angiographic Features. By Wade H. Shuford, and Robert G. Sybers, 687
Radiologic Examination of the Urinary Tract. By Howard M. Pollack, 905
Fraction Size in Radiobiology and Radiotherapy. Edited by Tsutomu Sugahara, László Révész, and Oliver C. A. Scott, 905
Bowel, small, following pyloroplasty and vagotomy (ab), 699
Brain scanning, what is role of, subdural hematoma; in its diagnosis? (ab), 456
Breast carcinomas; analysis of 462, 846
Bronchiolar carcinoma; review of 152 patients (ab), 465
Bronchocutaneous fistula as late complication of permanent epicardial pacing, 614
Bronchoscopy, prognostic value of, in primary lung cancer; new perspective for old procedure (ab), 459
Buscopan, esophageal varices; enhanced visualization of, 232

Calcaneus, solitary unicameral bone cyst of; review of twenty cases (ab), 703
Calcification in Cooper's ligament, 107
in uroepithelial tumors of bladder; report of 5 cases and survey of literature, 827
soft tissue, in infancy; extravasation of calcium gluconate as cause of, 109
Calcifications, intracranial, in infancy (ab), 210
Calcium gluconate as cause of soft tissue calcification in infancy; extravasation of, 109
Calculi, residual biliary, extraction of; in x-ray department (ab), 212
Calvarial, multiple, "Doughnut Lesions"; case report, 121
Calvarium; primary lymphangioma of, 118
Cancer of lung; results of split-course radiation therapy in, 754
of prostate; radiation therapy and pelvic node dissection in management of, 832
primary lung, prognostic value of bronchoscopy in; new perspective for old procedure (ab), 459
thyroid; unusual concentration of radiopertechnetate in patient with, 714
Carcinoid tumors of colon (ab), 212
Carcinoma, bronchiolar; review of 152 patients (ab), 465
of colon following ureterosigmoidostomy; increased incidence of, 806
of pancreas; selective pharmacodynamic angiography in diagnosis of (ab), 460
vocal cord; influence of fractionation on acute and late reactions in, 748
Carcinomas, breast; analysis of 462, 846
Catheterization, cardiac and coronary arteriography; radiation reduction by means of low pulse-rate fluoroscopy during, 636
of femoral artery, defective limb growth as complication of (ab), 922
Cervical esophageal webs—roentgen-anatomic correlation: observations on pharyngoesophagus, 221
spine and dysphagia; osteophytes of (ab), 463
Charnley total hip replacement, painful, 61
Cholangiography; percutaneous transhepatic (ab), 213
Cholecystography, cholecystokinin, in acalculous gallbladder disease (ab), 916
use of C-terminal octapeptide of cholecystokinin in, 380
Cholecystokinin cholecystography in acalculous gallbladder disease (ab), 916
Choledocho-pancreatography, endoscopic retrograde; review with report of collaborative series (ab), 213

Chondrosarcoma, primary and secondary; fibrosarcomatous dedifferentiation of: review of five cases (ab), 920
Christmas disease; bilateral pseudotumors of pelvis in patient with; with notes on localization by radioactive scanning and ultrasonography, 173
Chronic pancreatic pleural effusions (ab), 210
Cinecoronary angiography, improved accuracy of; angulated views in sagittal plane for, 565
Cirrhotic liver, macro-regenerating nodule in; radiologic features and differential diagnosis, 401
Clavicle, condensing osteitis of; new entity, 17
Cleidocranial dysostosis; review of 40 new cases, 5
Clindamycin (cleocin) colitis, 301
"Clipping injury" fracture of epiphysis in adolescent football player; occult lesion of knee, 69
Clustered mammary microcalcifications prior to biopsy: percutaneous needle localization of, 839
Colitis, clindamycin (cleocin), 301
Collagenous sprue, 275
Colon; carcinoid tumors of (ab), 212
carcinoma, increased incidence of; following ureterosigmoidostomy, 806
left; seat-belt injuries of (ab), 212
neoplasia of; over long segment (ab), 915
new method of roentgen examination of; water double contrast barium enema study, 326
"Colonic cocktail"; oral examination of colon, 318
Computer processing of data; dual camera studies of pulmonary function with, 761
Condensing osteitis of clavicle; new entity, 17
Continuing medical education; implications concerning recertification, re-examination and relicensure (E), 192
Cooper's ligament, calcification in, 107
Coronary artery disease, left main; coronary arteriography in, 583
left, and its branches; new lordotic projection for improved visualization of, 575
Craniofacial pathology; maxillary artery in, 124
Crohn's disease, colonic; diverting ileostomy for; Part 2: Radiological findings in colon following diversion (ab), 916
surgical treatment of; review of 120 cases (ab), 465
Cruciate ligament injuries; use of xeroradiography in, 94
C-terminal octapeptide of cholecystokinin in cholecystography; use of, 380
Cyst, bone, solitary unicameral, of calcaneus; review of twenty cases (ab), 703
puncture, nephrotomography, and renal arteriography; benign hemorrhagic renal cyst (ab), 461
renal, updated approach to diagnosis of; renal ultrasonography (ab), 701
Cystathionine synthase deficiency, clinical-roentgenologic correlations; homocystinuria due to, 45

Defect, distal anterior femoral metaphyseal; anatomic variant that may simulate disease, 101
Diabetic osteoarthropathy; clinical and roentgenographic observations in 90 cases, 22
Diagnoses, analysis of confidence level of, established by sequential roentgenographic investigation; roentgenographic assessment of asymptomatic renal lesions (ab), 214
Diagnosis, angiographic, and treatment by closed aspiration; pyogenic liver abscess, 388
differential and radiologic features; macroregenerating nodule in cirrhotic liver, 401
differential, of brain lesions by gallium scanning (ab), 456
of carcinoma of pancreas; selective pharmacodynamic angiography in (ab), 460
of perirenal and subcapsular masses; anatomic-radiologic correlation, 523
of renal cyst; renal ultrasonography: updated approach to (ab), 701
roentgen, of so-called intralobar sequestration of lung (ab), 458
subdural hematoma—what is role of brain scanning in its? (ab), 456
Diagnostic value of hypotonic duodenography, 256
Diaphragms, nonobstructive, or rings of gastric antrum in adults, 236
Diastematomyelia and congenital spine deformities (ab), 702
Disease, acalculous gallbladder; cholecystokinin cholecystography in (ab), 916
anatomic variant that may simulate; distal anterior femoral metaphyseal defect, 101
Christmas; bilateral pseudotumors of pelvis in patient with; with notes on localization by radioactive scanning and ultrasonography, 173
colonic Crohn's, diverting ileostomy for; Part 2: Radiological findings in colon following diversion (ab), 916
Crohn's, surgical treatment of; review of 120 cases (ab), 465
Hodgkin's, and malignant lymphoma; extended mantle radiotherapy in, 772
Hodgkin's; angiography of liver and spleen in, 789
lower extremity venous; radionuclide venography (RNV) in (ab), 922
Niemann-Pick; radiological findings in (ab), 218
osteolytic Paget's (ab), 919
Paget's; technetium 99m labeled diphosphonate bone scanning in, 177
renal, obstructive; megacalyces; further observations and differentiation from, 473
Diseases, diffuse, of small intestine presenting with

malabsorption; roentgenographic classification of, 283
Disparaesophageal hernias and acquired paraesophageal; complications of hiatal hernia repair, 248
Dissections, fissurizations and ruptures of aortic wall; contribution of aortography by arterial route; report of 69 observations (ab), 921
Disseminated intravascular coagulation (DIC); roentgen appearance of (ab), 219
Diverticula, giant duodenal, 334
multiple, or diverticulosis of ureter; rare radio-clinical syndrome (ab), 462
Diverticulosis or multiple diverticula of ureter; rare radio-clinical syndrome (ab), 462
Diverticulum, giant sigmoid, 338
large, of anal canal; case report and review of literature on anal canal and rectal diverticula, 344
"Doughnut Lesions," multiple calvarial; case report, 121
Dual camera studies of pulmonary function with computer processing of data, 761
Duodenal diverticula, giant, 334
hematoma; radiologic findings in (ab), 459
Duodenography, hypotonic; diagnostic value of, 256
Dura, lamina; lore and lure o' (ab), 697
Dysostosis, cleidocranial; review of 40 new cases, 5
Dysphagia and cervical spine; osteophytes of (ab), 463

Echography, prostatic; current status of (ab), 918
Ectopia, gallbladder; review of anomalies of position, 396
Ectopic ossification following total hip replacement; incidence and method of classification (ab), 703
Editorials
Continuing Medical Education: Implications concerning Recertification, Re-Examination and Relicensure, 192
Seventy-fifth Annual Meeting of the American Roentgen Ray Society, 431
Heuristic Gems from the American Radium Society, 653
Fifty-sixth Annual Meeting of the American Radium Society, 890
Effusions, pancreatic pleural, chronic (ab), 210
Embolism, fatty, after repeated perfusions of lipid emulsions (ab), 915
Emulsions, lipid; fatty embolism after repeated perfusions of (ab), 915
Endoscopic retrograde choledocho-pancreatography; review with report of collaborative series (ab), 213
Enterocolitis, those other types of, 291
Enterobiliary fistulas, spontaneous (ab), 212
Epicardial pacing, permanent; bronchocutaneous fistula as late complication of, 614
Epiphysis, fracture of, "Clipping injury" in adolescent football player; occult lesion of knee, 69
Esophageal varices by Buscopan; enhanced visualization of, 232
webs, cervical—roentgen-anatomic correlation; observations on pharyngoesophagus, 221
Esophagus, Barrett's, diagnosis of; with radioisotopes, 716
Extrahepatic and intrahepatic masses; pitfalls in arteriographic differentiation of, 420
Extravasation of calcium gluconate as cause of soft tissue calcification in infancy, 109
spontaneous, of contrast medium during intravenous urography: report of fourteen cases and review of literature (ab), 917

F^{18} and Tc^{99m} polyphosphate, comparison of, for bone imaging, 184
Femoral metaphyseal defect, distal anterior; anatomic variant that may simulate disease, 101
Fibrosarcomatous dedifferentiation of primary and secondary chondrosarcoma: review of five cases (ab), 920
Fifty-sixth Annual Meeting of American Radium Society (E), 890
Fistula, bronchocutaneous, as late complication of permanent epicardial pacing, 614
vesicoenteric, occult; detection and localization of, 811
Fistulas, enterobiliary, spontaneous (ab), 212
Fracture, "Clipping injury," of epiphysis in adolescent football player; occult lesion of knee, 69
Fractures, femoral and pelvic, after total hip arthroplasty (ab), 919
uncommon, in battered child syndrome; patterns of injury and significance of, 143

Galactography, mammography, and pneumocystography in detecting occult carcinomas of breast; significance of (ab), 466
Gallbladder disease, acalculous; cholecystokinin cholecystography in (ab), 916
ectopia; review of anomalies of position, 396
localized deformity of, secondary to hepatic mass lesions, 412
Gallium scanning; differential diagnosis of brain lesions by (ab), 456
Gangliosidosis (GM_1); re-evaluation of vertebral deformity, 155
Gastric antrum, nonobstructive mucosal diaphragms or rings of; in adults, 236
carcinoma patients; clinical evaluation of preoperative liver scintigraphy in, 720
Gastrointestinal tract, "lower," hemorrhage; new concepts based on arteriography, 357

Gemini space flights; review of roentgenographic bone demineralization studies of, 1
Glucagon; hypotonic roentgenography with, 264

(HEDSPA), Tc^{99m} diphosphonate, value of; as skeletal scanning agent, 735
Hematoma, duodenal; radiologic findings in (ab), 459
subdural—what is role of brain scanning in its diagnosis? (ab), 456
Hematuria, post-traumatic; selective pharmacoangiographic control of (ab), 463
Hemoptysis, selective bronchial arteriography in (ab), 915
Hemorrhage, "lower" gastrointestinal tract; new concepts based on arteriography, 357
renal, spontaneous; roentgenographic manifestations of (ab), 461
Hepatic mass lesions; localized deformity of gallbladder secondary to, 412
Hernia "En Masse"; spontaneous reduction of, 252
Hiatal hernia repair, complications of; acquired paraesophageal and disparaesophageal hernias, 248
Hickey Lecture, 1974; radiotherapy of tumors of urinary tract, 467
Hip joint, artificial, from roentgenologic standpoint (ab), 702
replacement, complications of; roentgen evaluation, 55
replacement, total, ectopic ossification following; incidence and method of classification (ab), 703
replacement, total, painful Charnley, 61
Hodgkin's disease and malignant lymphoma; extended mantle radiotherapy in, 772
angiography of liver and spleen in, 789
Homocystinuria due to cystathionine synthase deficiency; clinical-roentgenologic correlations, 45
Hyperparathyroidism, primary; arteriographic signs of; report of 10 cases (ab), 457
Hypotonic duodenography; diagnostic value of, 256
roentgenography with glucagon, 264
Hysterosalpingography in infertility, 642

I.A.E.A. Catalogue of Radiation Dosimetry Data, 901
Ileostomy, diverting, for colonic Crohn's disease; Part 2: Radiological findings in colon following diversion (ab), 916
Imaging, three-dimensional, of abdominal organs with ultrasound, 883
Infancy; intracranial calcifications in (ab), 210
Infertility; hysterosalpingography in, 642
Injury, patterns of, and significance of uncommon fractures in battered child syndrome, 143
Intestinal complications of radiotherapy (ab), 464
Intestine, small; inflammatory pseudotumor of, 305
Intra-arterial papaverine; contrast enhancement of mesenteric and portal veins using, 374
Intracranial calcifications in infancy (ab), 210
Intrahepatic and extrahepatic masses; pitfalls in arteriographic differentiation of, 420
arterial portal communications; angiographic study, 384
lesions, focal; use of multiple radionuclide imaging to differentiate, 728
Intralobar sequestration, so-called, roentgen diagnosis of; of lung (ab), 458
Intrarenal lesions; adrenal tumors simulating, 518
Isodose curves, computer generated, for high energy x-ray machines; experimental verification and time-saving strategies, 865
Isotope venography of pelvic veins (ab), 921

Kaposi's sarcoma; experience with ten cases, 793
Knee, lesion of, occult, "Clipping injury" fracture of epiphysis in adolescent football player, 69

Lamina dura; lore and lure o' (ab), 697
Leprosy, bone changes in: Dr. Diwan Chand Aggarwal Memorial Oration for 1972 (ab), 919
Lesion, focal intrahepatic; use of multiple radionuclide imaging to differentiate, 728
Lesions, brain, differential diagnosis of, by gallium scanning (ab), 456
hepatic mass; localized deformity of gallbladder secondary to, 412
intrarenal; adrenal tumors simulating, 518
Linear accelerators, high energy, in radiotherapy; design of x-ray targets for, 873
Lipohemarthrosis, 97
Lipomatosis, pelvic; report of three cases (ab), 220
Liver scintigraphy, preoperative, in gastric carcinoma patients; clinical evaluation of, 720
Lordotic projection, new, for improved visualization of left coronary artery and its branches, 575
Lung; *Aspergillus fumigatus;* its pathogenic action on (ab), 210
cancer of; results of split-course radiation therapy in, 754
so-called intralobar sequestration of; roentgen diagnosis of (ab), 458
superior sulcus tumor of; orthopedic aspects and early diagnosis of (ab), 698
Lymphangioma, primary, of calvarium, 118
Lymphoma, malignant and Hodgkin's disease; extended mantle radiotherapy in, 772

Macro-regenerating nodule in cirrhotic liver: radiologic features and differential diagnosis, 401
Malignant lymphoma and Hodgkin's disease; extended mantle radiotherapy in, 772
Mallory-Weiss syndrome (ab), 699
Mammary microcalcifications, clustered, prior to biopsy; percutaneous needle localization of, 839
Mammography; device for precision needle biopsy of breast at, 843
galactography, and pneumocystography in de-

tecting occult carcinomas of breasts; significance of (ab), 466
Masses, intrahepatic and extrahepatic; pitfalls in arteriographic differentiation of, 420
perirenal and subcapsular, diagnosis of; anatomic-radiologic correlation, 523
Meckel; os supra petrosum of, 139
Medical education, continuing; implications concerning recertification, re-examination and relicensure (E), 192
Megacalyces; further observations and differentiation from obstructive renal disease, 473
Megacolon, toxic; considerations on (ab), 211
Melanoma, metastatic; urographic manifestations of, 801
Melorheostosis (ab), 218
Meniscus, lateral; arthrography of, 79
Mesenteric and portal veins using intra-arterial papaverine; contrast enhancement of, 374
Metacarpal sign (ab), 464
Metastases, pulmonary; results after resection of (ab), 458
Metastatic melanoma; urographic manifestations of, 801
Mucosal diaphragms, nonobstructive, or rings of gastric antrum in adults, 236
striations of upper urinary system; review of 58 cases (ab), 215
Myelography, painless; introduction of new aspiration cannula and review of 541 consecutive studies (ab), 216

National Council on Radiation Protection and Measurements (NCRP), 900
Research Council: Committee on Radiology, Division of Medical Sciences; Picker Foundation Awards, 1974–1975, 897
Neonatal transplacental rubella syndrome; its effect on normal maturation of diaphysis, 166
Neoplasia of colon over long segment (ab), 915
Neoplastic and cultured normal cells; thermal enhancement of radiosensitivity using, 860
Nephrotomography, renal arteriography, and cyst puncture; benign hemorrhagic renal cyst (ab), 461
Niemann-Pick disease; radiological findings in (ab), 218

Oral examination of colon; "colonic cocktail," 318
Orbit, venography of; analytical report of 413 cases (ab), 456
Ossification, ectopic, following total hip replacement; incidence and method of classification (ab), 703
Os supra petrosum of Meckel, 139
Osteitis, condensing, of clavicle; new entity, 17
pubic, and postpartum thrombophlebitis (ab), 920
Osteoarthropathy, diabetic; clinical and roentgenographic observations in 90 cases, 22
Osteoid osteoma, multifocal; case report and review of literature, 103
Osteolytic Paget's disease (ab), 919
Osteoma, multifocal osteoid; case report and review of literature, 103
Osteomyelitis, subacute; serpiginous tract: sign of (ab), 704
Osteophytes of cervical spine and dysphagia (ab), 463
Ostium primum defect in adult; left ventricular angiographic anatomy of, 597

Paget's disease, osteolytic (ab), 919
technetium 99m labeled diphosphonate bone scanning in, 177
(Pancoast) tumor, superior sulcus, of lung; orthopedic aspects and early diagnosis of (ab), 698
Pancreatic abscess (ab), 917
pleural effusions, chronic (ab), 210
Pancreatitis, relapsing acute; endoscopic pancreatography in management of (ab), 916
Pancreatography, endoscopic, in management of relapsing acute pancreatitis (ab), 916
Paraesophageal, acquired, and disparaesophageal hernias; complications of hiatal hernia repair, 248
Pathogenic action; *Aspergillus fumigatus*, on lung (ab), 210
Pathologic and roentgenographic findings in surgical patients; inferior vena caval extension of renal carcinoma; lost cause?, 819
Pathology, craniofacial; maxillary artery in, 124
Pelvic bony outlet, size of, in renal agenesis, 159
destructions; progressive post-traumatic (ab), 217
lipomatosis, report of three cases (ab), 220
node dissection and radiation therapy, in management of cancer of prostate, 832
veins, isotope venography of (ab), 921
Percutaneous needle localization of clustered mammary microcalcifications prior to biopsy, 839
transhepatic cholangiography (ab), 213
Perirenal and subcapsular masses, diagnosis of; anatomic-radiologic correlation, 523
Peutz-Touraine-Jeghers syndrome (ab), 700
Pharmaco-angiographic, selective, control of post-traumatic hematuria (ab), 463
Pharmacologic aids in angiography of upper extremity, 150
Pharyngoesophagus, observations on; cervical esophageal webs—roentgen-anatomic correlation, 221
Phlebography and arteriography of limbs in planning shunt and fistula operations in patients with chronic renal failure, 551
Picker Foundation Awards, 1974–1975; National Research Council; Committee on Radiol-

ogy, Division of Medical Sciences, 897
Pneumobilia, "innocent"; report of case and review of literature (ab), 459
Pneumoconiosis in women (ab), 210
Pneumocystography, mammography, and galactography, in detecting occult carcinomas of breast; significance of (ab), 466
Portal vein, ventral, 369
veins and mesenteric, using intra-arterial papaverine; contrast enhancement of, 374
Post-traumatic pelvic destructions, progressive (ab), 217
Precision needle biopsy of breast at mammography; device for, 843
Preliminary Program: Seventy-fifth Annual Meeting of American Roentgen Ray Society, 436
Prostate, cancer of; radiation therapy and pelvic node dissection in management of, 832
Prostatic echography, current status of (ab), 918
Pseudocyst formation; arthrography in rupture of suprapatellar bursa with, 89
Pseudotruncus arteriosus; importance of selective angiography of systemic arterial supply to lungs in planning surgical correction of, 606
Pseudotumor, inflammatory, of small intestine, 305
Pseudotumors, renal (ab), 215
Pulmonary blastomycosis, acute; clinical features of (ab), 698
function, dual camera studies of; with computer processing of data, 761
metastases, results after resection of (ab), 458
venous connection, partial anomalous; angiographic demonstration of absence of atrial septal defect in presence of, 591
Pyelonephritis, xanthogranulomatous, 500
Pyloroplasty and vagotomy; small bowel following (ab), 699
Pyogenic liver abscess; angiographic diagnosis and treatment by closed aspiration, 388

Radiation reduction by means of low pulse-rate fluoroscopy during cardiac catheterization and coronary arteriography, 636
therapy and pelvic node dissection in management of cancer of prostate, 832
therapy, split-course, in cancer of lung; results of, 754
Radioactive scanning and ultrasonography, bilateral pseudotumors of pelvis, in patient with Christmas disease; with notes on localization by, 173
Radio-clinical study; forgotten operative fields in abdomen (ab), 211
Radioisotopes; diagnosis of Barrett's esophagus with, 716
Radiologic abnormalities due to tubo-ovarian abscess; spectrum of (ab), 702
features and differential diagnosis; macroregenerating nodule in cirrhotic liver, 401
findings in duodenal hematoma (ab), 459
studies in investigation of causes of total hip replacement failures (ab), 217
Radiological findings in Niemann-Pick disease (ab), 218
Radiology of acute renal insufficiency (ab), 917
of angiodysplasias of limb (ab), 923
Radionuclide imaging, multiple, use of, to differentiate focal intrahepatic lesion, 728
venography (RNV) in lower extremity venous disease (ab), 922
Radiopertechnetate, unusual concentration of; in patient with thyroid cancer, 714
Radiosensitivity, thermal enhancement of, using cultured normal and neoplastic cells, 860
Radiotherapy; design of x-ray targets for high energy linear accelerators in, 873
extended mantle, in Hodgkin's disease and malignant lymphoma, 772
intestinal complications of (ab), 464
of tumors of urinary tract; Hickey Lecture, 1974, 467
Rectal diverticula and anal canal, case report and review of literature on; large diverticulum of anal canal, 344
Renal, acute, failure after angiography, 544
agenesis; size of pelvic bony outlet in, 159
angiogram in tuberculosis (ab), 460
arteriography, nephrotomography, and cyst puncture; benign hemorrhagic renal cyst (ab), 461
bleeding, angiography in, with negative urogram, 508
carcinoma, inferior vena caval extension of; lost cause? roentgenographic and pathologic findings in surgical patients, 819
cyst, benign hemorrhagic; nephrotomography, renal arteriography, and cyst puncture (ab), 461
cyst; renal ultrasonography: updated approach to diagnosis of (ab), 701
disease, how accurate is diagnostic ultrasound in? (ab), 918
disease, obstructive; megacalyces: further observations and differentiation from, 473
failure, chronic; arteriography and phlebography of limbs in planning shunt and fistula operations in patients with, 551
hemorrhage, spontaneous; roentgenographic manifestations of (ab), 461
insufficiency, acute; radiology of (ab), 917
lesions, asymptomatic, roentgenographic assessment of; analysis of confidence level of diagnoses established by sequential roentgenographic investigation (ab), 214
pseudotumors (ab), 215
tuberculosis; arteriographic symptoms of (ab), 701
ultrasonography; updated approach to diagnosis of renal cyst (ab), 701

veins, pericapsular; recognition without angiography (ab), 462
Retrogastric space, evaluation of; normal appearance and variation, 348
Rheumatoid arthritis and scleroderma; similar bone manifestations of, 35
Roentgen-anatomic correlation, cervical esophageal webs; observations on pharyngoesophagus, 221
appearance of disseminated intravascular coagulation (DIC) (ab), 219
diagnosis of so-called intralobar sequestration of lung (ab), 458
evaluation; complications of total hip replacement, 55
examination of colon, new method of; water double contrast barium enema study, 326
features of skeletal involvement in small pox (ab), 218
features of synovial sarcomas (ab), 466
Roentgenographic and pathologic findings in surgical patients; inferior vena caval extension of renal carcinoma; lost cause?, 819
assessment of asymptomatic renal lesions; analysis of confidence level of diagnosis established by sequential roentgenographic investigation (ab), 214
bone demineralization studies of Gemini space flights; review of, 1
classification of diffuse diseases of small intestine presenting with malabsorption, 283
evaluation of dissecting aneurysms of aorta, 617
findings in urinary tract tuberculosis; 10 year review, 487
investigation, sequential; roentgenographic assessment of asymptomatic renal lesions; analysis of confidence level of diagnoses established by (ab), 214
manifestations of spontaneous renal hemorrhage (ab), 461
Roentgenography, hypotonic, with glucagon, 264
Rubella syndrome, neonatal transplacental; its effect on normal maturation of diaphysis, 166
Rupture, aortic, in children as complication of coarctation of aorta (ab), 922

Sagittal plane, angulated views in, for improved accuracy of cinecoronary angiography, 565
Sarcoma, Kaposi's; experience with ten cases, 793
Sarcomas, synovial; roentgen features of (ab), 466
Scan, liver; conservative management and liver trauma (ab), 460
Scanning agent, skeletal; value of Tc^{99m}-diphosphonate (HEDSPA) as, 735
Paget's disease; technetium 99m diphosphonate of bone, 177
thyroid, quantitative; reliable thyroid function test, 705
Scleroderma and rheumatoid arthritis; similar bone manifestations of, 35
Seat-belt injuries of left colon (ab), 212
Section on Instruction, American Roentgen Ray Society, 661
Serpiginous tract; sign of subacute osteomyelitis (ab), 704
Seventy-fifth Annual Meeting of American Roentgen Ray Society (E), 431
Sigmoid diverticulum, giant, 338
Skeletal imaging, pitfalls in; Tc^{99m} polyphosphate, 739
involvement in small pox; roentgen features of (ab), 218
Small pox; roentgen features of skeletal involvement in (ab), 218
Spine deformities, congenital; diastematomyelia (ab), 702
Splenic rupture, clinically suspected; reliability of negative angiography in (ab), 700
Split-course radiation therapy in cancer of lung; results of, 754
Sprue, collagenous, 275
Subcapsular and perirenal masses, diagnosis of; anatomic-radiologic correlation, 523
Subclavian steal, congenital, 559
Suprapatellar bursa, rupture of, with pseudocyst formation; arthrography in, 89
Syndrome, Alport's, angiographic features of; hereditary nephritis, 539
Mallory-Weiss (ab), 699
neonatal transplacental rubella; its effect on normal maturation of diaphysis, 166
of arcuate ligament (ab), 703
Peutz-Touraine-Jeghers (ab), 700
rare radio-clinical; multiple diverticula or diverticulosis of ureter (ab), 462
Synovial sarcomas; roentgen features of (ab), 466

Tc^{99m}-diphosphonate (HEDSPA), value of, as skeletal scanning agent, 735
polyphosphate and F^{18} for bone imaging; comparison of, 184
polyphosphate skeletal imaging; pitfalls in, 739
Technetium 99m labeled diphosphonate bone scanning in Paget's disease, 177
Thermal enhancement of radiosensitivity using cultured normal and neoplastic cells, 860
Thrombophlebitis, postpartum, and pubic osteitis (ab), 920
Thyroid cancer; unusual concentration of radiopertechnetate in patient with, 714
scanning, quantitative; reliable thyroid function test, 705
Thyroidography and thyrolymphography (ab), 697
Thyrolymphography and thyroidography (ab), 697
Toxic megacolon; considerations on (ab), 211
Tracheomalacia, acquired (ab), 457
Transhepatic cholangiography; percutaneous (ab), 213
Transplacental rubella syndrome, neonatal; its

effect on normal maturation of diaphysis, 166
TRAUMA, liver; conservative management and liver scan (ab), 460
TUBERCULOSIS; renal angiogram in (ab), 460
renal; arteriographic symptoms of (ab), 701
urinary tract; roentgenographic findings in: 10 year review, 487
TUBO-OVARIAN abscess; spectrum of radiologic abnormalities due to (ab), 702
TUMORS, adrenal, simulating intrarenal lesions, 518
carcinoid, of colon (ab), 212
of urinary tract; radiotherapy of, Hickey Lecture, 1974, 467
uroepithelial, of bladder, calcification in; report of 5 cases and survey of literature, 827

ULTRASONOGRAPHY, bilateral pseudotumors of pelvis in patient with Christmas disease; with notes on localization by radioactive scanning and, 173
renal; updated approach to diagnosis of renal cyst (ab), 701
ULTRASOUND "B" scanning, continuous, of abdominal aortic aneurysms, 626
diagnostic; how accurate is, in renal disease? (ab), 918
three-dimensional imaging of abdominal organs with, 883
UPPER extremity; pharmacologic aids in angiography of, 150
URETER, multiple diverticula or diverticulosis of; rare radio-clinical syndrome (ab), 462
URETERAL obstruction in patients with uterine prolapse (ab), 463
URETEROSIGMOIDOSTOMY; increased incidence of carcinoma of colon following, 806
URINARY system, upper; review of 58 cases, mucosal striations of (ab), 215
UROEPITHELIAL tumors of bladder, calcification in; report of 5 cases and survey of literature, 827
UROGRAM, negative; angiography in unilateral renal bleeding with, 508
UROGRAPHIC manifestations of metastatic melanoma, 801
UROGRAPHY, intravenous; spontaneous extravasation of contrast medium during; report of fourteen cases and review of literature (ab), 917
UTERINE prolapse; ureteral obstruction in patients with (ab), 463

VAGOTOMY and pyloroplasty, small bowel following (ab), 699
VARICES, esophageal, by Buscopan; enhanced visualization of, 232
VENA caval extension, inferior, of renal carcinoma; lost cause? roentgenographic and pathologic findings in surgical patients, 819
VENOGRAPHY, isotope, of pelvic veins (ab), 921
of the orbit; analytical report of 413 cases (ab), 456
radionuclide, (RNV) in lower extremity venous disease (ab), 922
VENTRICULAR angiographic anatomy, left, of ostium primum defect in adult, 597
VERTEBRAL deformity, re-evaluation of; gangliosidosis (GM_1), 155
VESICOENTERIC fistula, occult; detection and localization of, 811
VOCAL cord carcinoma; influence of fractionation on acute and late reactions in, 748

WORKSHOPS on Federal Diagnostic X-Ray Standard; Department of Health, Education, and Welfare, 441

XANTHOGRANULOMATOUS pyelonephritis, 500
XERORADIOGRAPHY in cruciate ligament injuries; use of, 94
X-RAY machines, high energy, computer generated isodose curves for; experimental verification and time saving strategies, 865
Standard; Workshops on Federal Diagnostic: Department of Health, Education and Welfare, 441
targets for high energy linear accelerators in radiotherapy, design of, 873

AUTHOR INDEX TO VOLUME 121

*=Original Article

Adar, Rafael, *384
Allen, William E., III, *124
Ameratunga, Bertie, 218
Amiel, M., 921
Anderson, William J., 210
Antoine, John E., *118, *634
Antonovic, Ruza, *583
Appelman, Henry, *839
Archer, Bernard T., 697
Arger, Peter H., *97
Arkles, L. Barry, *705
Arnaud, A., 915
Asher, W. Michael, 701
Autran, P., 915

Babenco, George O., *412
Bader, Lewis M., *348
Baglin, A., 457
Baltaxe, Harold A., *575, *591, *606
Barois, A., 915
Barrier, J., 920
Barry, William F., Jr., *806
Bartrum, Royal J., Jr., *883
Bartucz, J. P., 917
Basu, S. P., 919
Batata, Mostafa A., *832
Beachley, Michael C., *500, *617
Beales, J. S. M., 916
Beattie, Edward J., Jr., 465
Beeckman, P., 211
Ben-Menachem, Yoram, *388
Berdon, Walter E., *159
Bergan, John J., 922
Berger, Paul E., *109
Bernhard, J. D., 459
Berry, Harrison M., Jr., 697
Bilaniuk, Larissa T., 456
Biramje, Aspi, 460
Bismuth, V., 457
Blaisdell, F. William, 219
Blanc, P., 211
Blanton, David E., *396
Blery, M., 457
Bliznak, Johnny, 923
Bloch, Peter, *865
Bloom, John D., 922
Blumgart, L. H., 213
Bogokowski, Harry, *384
Borushok, Michael J., *559
Bourassa, Martial G., *565
Bowerman, Jack W., 703
Boyce, William H., 918
Brady, Luther W., 890
Brandborg, Lloyd L., *275
Bream, Charles A., *396
Brill, Paula W., *45
Brisset, J. M., 457
Brogdon, B. G., 192
Brooker, Andrew F., 703
Brooks, John R., 699
Brower, Anne C., *17
Brown, C. Selby, 217
Brown, Henry W., 697
Bryk, David, *103
Buckley, Clifford J., 922
Buddemeyer, E. Ulric, *811
Burwood, R. J., 213

Cameron, John L., 210
Campbell, Robert E., *61
Campbell, William L., *305
Capdeville, R., 215
Carmody, Philip W., *634
Carr, David T., *487
Casarella, William J., *357
Cassan, P., 217
Castronovo, Frank P., Jr., *177
Castroviejo, I. Pascual, 210
Catalano, Dino, 463
Chernish, Stanley M., *264
Chiotelis, E., *735
Chuang, Vincent P., *539
Chynn, K. York, 216
Clark, Robert, *518
Clay, Claude, 456
Cleary, Robert E., *642
Clément, J. P., 915
Clements, James L., Jr., *221
Clermont, A., 921
Clouse, Melvin E., *22
Coates, Geoffrey, 456
Cohen, Leroy E., *301
Cole-Beuglet, C., *626
Constable, William C., *754
Constandinidis, C., *735
Constantinidis, M., *735
Cooke, G. M., 917
Cotton, P. B., 916
Coussement, A., 211
Coussement, N., 211
Coventry, Mark B., 919
Cox, George W., *221
Crocker, Allen, 218
Cuaren, Alfredo, *716
Cunningham, C. D. B., 210
Currarino, Guido, *139
Czekala, Zbigniew, 701

Dalinka, Murray K., *79, 704
Dallemand, Serge, *338
Davies, G. T., 213
Davis, A. Ross, *69
Davis, Lawrence W., *865
Del Fava, Raymond L., *312
Del Regato, J. A., *467
DeMots, Henry, *583
Depisch, D., 700
Deutsch, Victor, *384
Dietz, Geral, *69
Dische, Renate, *166
Dolinskas, Carol, *61
D'Orsi, Carl J., *94
Dorst, John P., *559
Dotter, Charles T., *583
Dow, Richard, *839
Dudley, H. A. F., 699
Duncan, Adah M., *89
Duquesnel, J., 921

Ebert, Paul A., *606
Edeiken, Jack, 704
Ehrlich, Frank E., 459
Eisenbrey, Bradley A., *150
Elguezabel, Alberto, *103
Elkin, Milton, 463
El-Mahdi, Anas, *754
Engelbrecht, E., 702
Engle, Mary Allen, *606
Englert, M., 464
Euler-Rolle, J., 700
Evans, John A., *523

Fagan, Charles J., *143
Farman, Jack, *338
Faure, C., 211
Feinberg, Samuel B., *283, *291
Feist, J. H., 457
Felder, Peter A., 460
Felippone, Nestor, 210
Fellows, Richard A., *159
Fendler, J. P., 457
Fleurquin, A. G., 220
Flood, Thomas, *22
Follett, David A., *357
Fontaine, Y., 211
Forbes, Charles D., *173
Fortier-Beaulieu, M., 215
Freedman, Sheldon, 915
Frey, Charles F., 917
Friedland, Gerald W., 916
Fritel, D., 457
Froment, J.-C., 921
Fulton, Richard E., *544

Galloway, Shelby J., *357

Gaull, Gerald E., *45
Gauthier, N., 215
Gaux, J. C., 457
Geslien, George E., *739
Ghaed, Nassar, *739
Ghahremani, Gary G., *236
Gilday, David L., 456
Giraud, R., 915
Gittes, Ruben F., *473, 701
Glavinović, M. I., *873
Glickman, Morton G., *420
Gohel, Vijay K., *79, 704
Golbey, Robert B., 465
Goldberg, Henry I., *275
Goldberg, Henry P., *606
Goldberg, Marvin E., *761
Goldenberg, David, 456
Goldman, Arnold M., *388
Goldman, Howard, *508
Goldman, Stanford M., 463
Goldstein, Harvey M., *801
Goodman, Daniel J., *597
Gordon, Felipe, *716
Gosink, Barbara B., 701
Goulon, M., 915
Govoni, A. F., *344
Govoni, Antonio F., 463
Grabstald, Harry, *832
Grall, F., 920
Gramm, Herbert F., *22
Grant, Mary, *173
Green, William M., *305
Greenbaum, Edward I., 915
Greenspan, Adam, *103
Greig, William R., *173
Griem, M. L., *772
Griffiths, Harry J., *94
Grollman, J. H., Jr., *636
Grosbuis, S., 915
Gupta, S. K., 218
Guttadauria, Maria, *35

Hagood, Clyde O., Jr., 922
Hahn, E., *860
Hammerman, Kenneth J., 698
Harell, George, *369
Hartman, Glen W., *487
Hately, William, 918
Hattery, Robert R., *487
Haven, James J., 702
Hawkins, I. F., Jr., 460
Heidelberger, Kathleen P., *109
Henkin, Robert E., 922
Highman, J. H., 921
Hilaris, Basil S., *832
Himmelfarb, Elliot, *252
Holm, Hans H., *883
Holman, Colin B., 431
Horowitz, Alfred L., 466,
Hoyt, Terry, *248
Hugh, A. E., 210
Huguier, M., 465
Hunter, James G., Jr., *614

Idriss, Farouk S., 922
Itzchak, Yacov, *384

Jackman, Roger J., 461
Jackson, B. T., 464
Jacobs, Philip, 919
Jaconette, J. Richard, 462
Jarvis, J. Luther, *5
Joffe, Norman, *412
Johns, H. E., *873
Johnson, Douglas E., *801
Johnson, Merrill C., *739
Johnson, Philip M., *728
Johnson, Thomas H., 457
Jones, Stanley, *69
Jonsson, K., *789
Judkins, Melvin P., *583

Kádas, I., 466
Kaminsky, Stephen, *801
Kaufer, Herbert, 698
Kawahira, Kenjiro, *720
Kay, Roger, *150
Keats, Theodore E., *5, *17, *101, *107, *614
Kempf, F., 459
Keyes, J. W., Jr., *184
Khafagy, Medhad M., 465
Kier, E. Leon, *124
Kim, J. H., *860
Kim, S. H., *860
King, William W., 918
Kinkabwala, Mahesh, *401
Kirsh, Marvin M., 698
Klatte, Eugene C., *642
Kleinhaus, Uriel, 699
Kleinknecht, D., 917
Knapp, Robert W., 459
Knickerbocker, W. J., 217
Kogutt, Marvin S., *143
Kohatsu, Shoichi, 916
Kolár, J., 217
Kollins, Stephen A., *487
Kopperman, Mordecai, *118
Kosaras, B., 466
Kronenberg, Richard S., 698, *761
Krook, Lennart, *166
Kyaw, Myo M., *248

Lachand, A.-T., 917
Lachman, Ralph, 218
Lagaard, Sheldon M., 702
Lally, James F., *79
Lamoureux, Jacques, *714
Landa, Luis, *716
Landman, S., *184
Lane, Karen, *865
Lang, Erich K., 214
Laurent, Y., 462
Lawrie, B. W., 213
Lee, George, 456
Lee, Jack G., *575
Lee, L. H., *854
Legg, Merle, *22
Lemaitre, G., 215
Leopold, George R., 701
Lequesne, M., 217
Lespérance, Jacques, *565
Lester, Richard G., *806
Leucutia, Traian, 653
Levant, Jonathan A., *380
Levin, Aaron R., *591, *606
Levin, David C., *575, *591, *606
Lewicki, Ann M., 699
Light, Wolfe, 212
Lindenauer, S. Martin, 917
Liu, Chung-I, *232
Lodwick, Gwilym S., *55
Loeffler, R. Kenneth, *748
Loken, Merle K., *761
Loygue, J., 465
Lunderquist, A., *789

Macgregor, Alexander M. C., 460
Malafosse, M., 465
Malta-Bey, G., 465
Mandel, Stanley R., *396
Mandl, W., 700
Mann, Steven G., *793
Marcove, Ralph C., 920
Mareschal, J. L., 215
Marks, J. E., *772
Marks, Richard D., Jr., *754
Martini, Nael, 465
Márton, Z., 466
Masterton, J. P., 699
Matsuura, Keiichi, *720
McCarthy, William, 916
McCullough, David L., *819
McElfresh, Edward C., 919
McGinnis, Kenneth D., 462
McKinney, William M., 918
McKinnon, C. Malcolm, *518
Meissner, K., 700
Melamed, Myron R., 465
Membreno, Alejandro A., 699
Meng, Chien-Hsing, 463
Meyers, Morton A., *523
Michel, J. R., 215
Michel, J.-R., 917
Middleton, Philip J., *634
Mikita, John J., 457
Millard, Jack R., *334
Miller, Ira D., 459
Miller, Roscoe E., *264
Miller, Stephen W., *177, *827
Miller, Thomas A., 917

Miller, Wallace T., *97
Minagi, Hideyo, 219
Mirra, Joseph M., 920
Mitty, Harold A., *45, *508
Moe, John H., 702
Montgomery, Charles E., *86
Moran, E. M., *772
Moreau, J.-F., 917
Moss, Albert A., *275
Moule, Brian, *173
Mozersky, David J., 922
Mozes, Mark, *384
Mühlow, Anders, *843
Mulder, D. S., *626
Mullen, Joseph T., 459
Mullins, Maurice F., *55
Muñoz, Rodrigo, *716
Muroff, Lawrence R., *728

Németh, A., 466
Nielsen, S. Skogstad, *883
Nikaidoh, Hisashi, 922
Nora, Paul F., 916
Nordahl, David L., *374
Novy, Stanley B., *388
Nunez, Eladio, *166

Oberhelman, Harry A., Jr., 916
Oberkircher, Paul E., *97
O'Donohue, Neil, *166
Oh, Kook Sang, *559
Olbert, F., 458
Oon, C. L., 213
O'Rourke, Terry, *839
O'Sullivan, E., 921
Ozonoff, M. B., *121

Padovani, J., 215
Palubinskas, Alphonse J., *420
Papadimitriou, J., *735
Parvey, Louis S., *252
Pasquier, J., 915
Peeters, F. L. M., 460
Pendergrass, Henry P., *177
Penn, W., 217
Pepper, H. Walter, *275
Petersen, Loren, *642
Petitclerc, Robert, *565
Pfister, Richard C., *827
Phillips, Jerry C., 702
Pinet, A., 215
Pinet, F., 921
Pinsky, Steven M., *739
Pister, James D., *301
Plainfossé, M. C., 215
Pochaczevsky, Rubem, *318, *326
Podgoršak, E. B., *873
Pollock, Edward J., *357
Pollack, Howard M., 461
Ponto, Richard A., *761
Popky, George L., 461
Popovsky, Julio, 460
Port, Frederick K., *544
Potsaid, Majic S., *177
Poranski, Andrew K., *109
Preisching, A., 700
Preissig, Randall S., *806
Prentice, Colin R. M., *173
Printen, Kenneth J., 212
Prokop, Edward K., *811
Putman, Charles E., 219

Quinn, James L., III, 922

Rabinowitz, Jack, *252
Rabinowitz, Jack G., *35, *155, *338, *401
Rahimtoola, Shahbudin H., *583
Ram, M. D., 697
Ramírez-Degollado, José, *716
Randall, Patricia A., *597
Ranniger, Klaus, *500, *617
Rappaport, A. S., *551
Rasmussen, S. Nørby, *883
Rawlinson, J. A., *873
Rennell, Charles L., *256
Resnick, Donald, 466
Reuter, Stewart R., *539
Reydman, Melvin, 460
Riker, William L., 922
Riley, Lee H., Jr., 703
Robbins, Alan H., *374
Robinson, Robert A., 703
Rodda, Bruce E., *264
Rogers, Lee F., *69
Roka, R., 700
Romani, Francesco, 703
Rösch, Josef, *583
Rosen, Yale, *338
Rosenak, Bernard D., *264
Roth, Franz-Joseph, *500, *617
Rothman, Richard H., *61
Rothman, Stephan L. G., *124
Royen, Peter M., *121
Rubet, A., 921
Russo, Rodolfo, 210

Sacher, Michael, *155
Safaie-Shirazi, Siroos, 212
Sakover, Raymond P., *312
Salmon, P. R., 213
Saltiel, Jacques, *565
Sanez, Nicholas, 916
Sarosi, George A., 698
Schechner, Stephen A., 459
Schmoller, H. J., 700
Schortgen, G., 915
Schroeder, John S., *597
Schuit, K. E., *107
Schulman, Joseph, 218
Scott, J. E., 212
Scruggs, Hugh, *754
Seaman, William B., *305, *357
Segura, Joseph W., *487
Shanser, Jon D., *275, *420
Shennan, John, 212
Shrago, Gary G., *518
Siemsen, Jan K., 456
Skinner, David B., 210
Skucas, Jovitas, *264
Slover, William P., *334
Smith, Chadwick F., 703
Smith, Charles J., *301
Smith, Ronald W., 703
Smulewicz, Jan J., *344
Sobredo, Enrique Eduardo, 210
Sos, Thomas A., *575, *591, *606
Spengler, Dan M., 698
Srivastava, T. P., 218
Stanley, James C., 917
Staple, Tom W., 923
Steinfeld, John R., *107
Stevens, G. Melvin, 461
Stigsby, Bent, *883
St. John, D. J. B., 699
Stoloff, Irwin L., 459
Strand, Roy, 218
Strauss, H. William, *811
Sturdevant, Richard A. L., *380
Sutton, Richard N., *55
Suwandschieff, N., 700
Sweet, Donald E., *17
Swischuk, Leonard E., *143
Syme, James, 219

Tabár, L., 466
Talner, Lee B., *473, 701, *819
Tavernier, J., 215
Taxin, Richard N., *357
Tay, Daniel, *591
Tegtmeyer, Charles J., *614
Tenner, R., 702
Thomas, M. Lea, *551
Thrall, James H., *739
Threatt, Barbara, *839
Tongio, J., 459
Torres, William E., *221
Tosh, Fred E., 698
Tountas, C., *735
Tully, Timothy E., *283, *291
Twersky, Jack, *35, *338

Ulreich, Sidney, *401
Ultmann, J. E., *772

Van De Velde, E., 211
Vezeridis, M., *735
Vignaud, Jacqueline, 456
Vigneron, N., 920

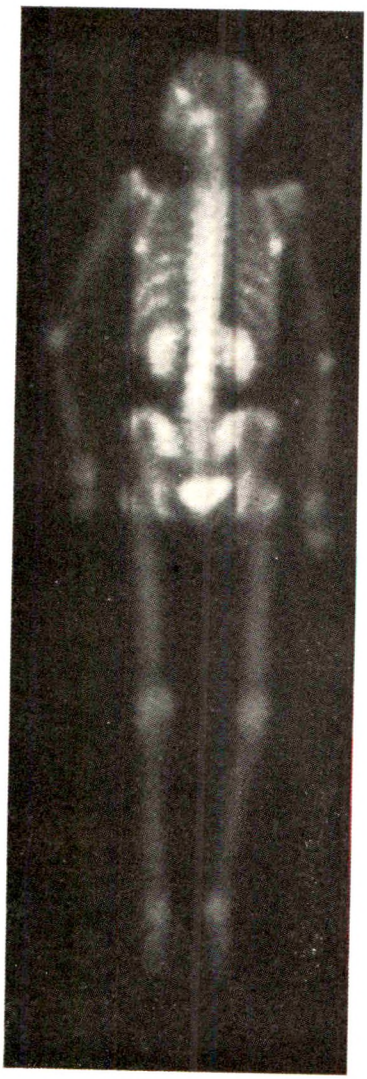
Vose, George P., *1

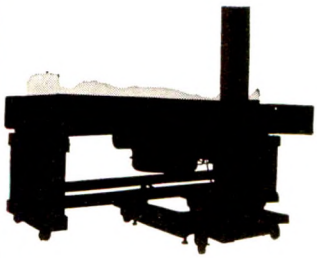
Wagner, Henry N., *811
Wagoner, Richard D., *544
Wallace, Sidney, *388, *801
Warter, P., 459
Watanabe, Katsushi, *720
Watson, Robin Caird, 466
Waxman, Alan D., 456
Weber, D. A., *184
Weens, H. Stephen, *221
Weinberg, Arthur, *139
Wells, Ralph F., *301
Wellwood, J. M., 464
Whalen, Joseph P., *166, *348, *523
Whitaker, Robert H., 918
White, Robert I., Jr., *559
Whitmore, Willet F., Jr., *832
Wicke, L., 458
Widrich, Warren C., *374
Wiener, Stephen N., 460
Wilkiemeyer, R. Mark, 918
Williams, Lawrence E., *761
Willich, von E., 464
Wilner, Harvey I., *150
Wilson, G. A., *184
Wilson, Robert J., 457
Winchester, Patricia, *166
Wing, A. J., *551
Winsberg, F., *626
Winter, Robert B., 702
Wolfe, John N., *846
Wolfman, Richard, *348
Wolfstein, Ralph, 456

Yao, James S. T., 922
Yeomans, N. D., 699
Yu, S. F., 213
Yune, Heun Y., *642

Zagnoli, Paolo, 703
Zeskind, H. Jay, 462
Zike, Wilbur L., 212
Ziter, Fred M. H., *334
Zuidema, George D., 210
Zwintz, E., 458, 458

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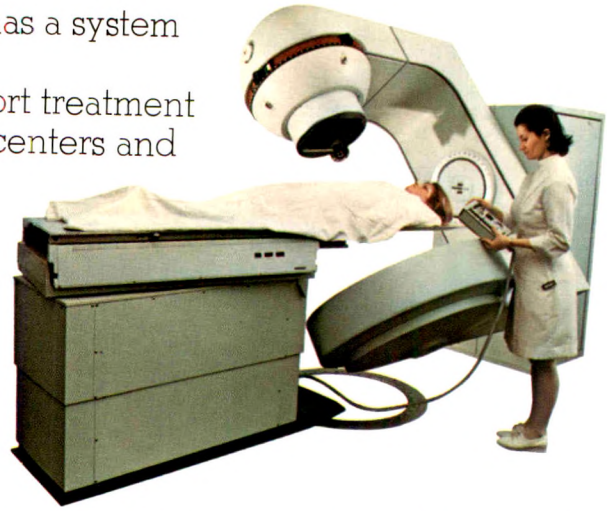
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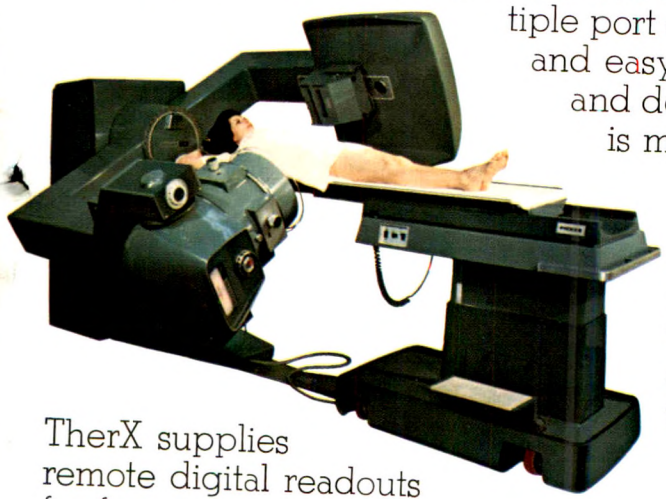


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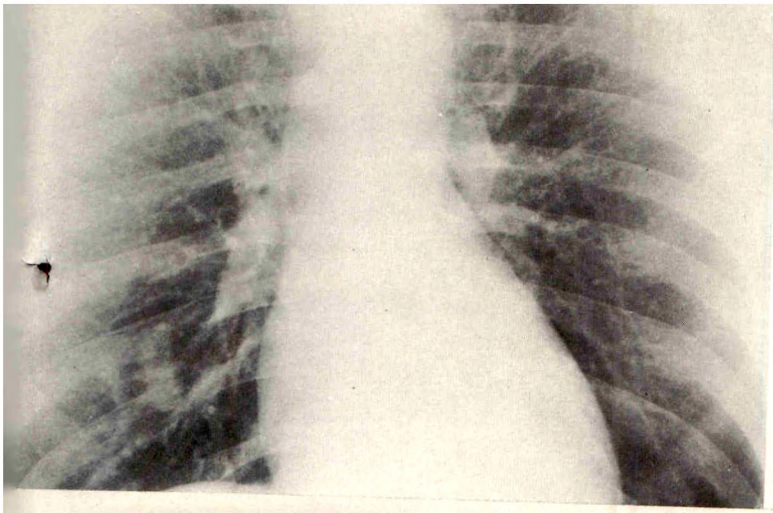
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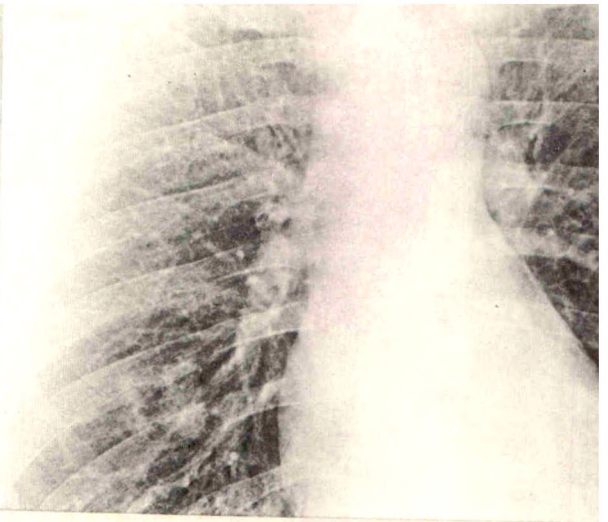
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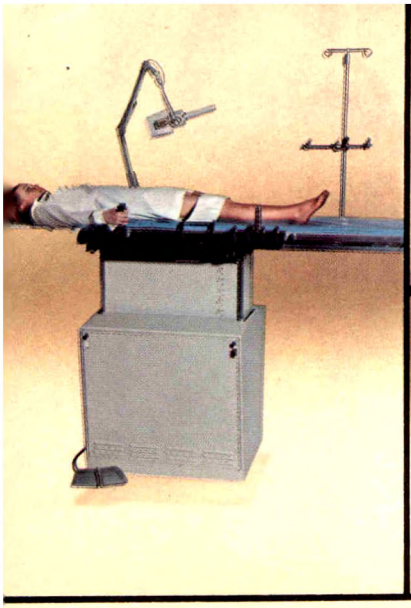
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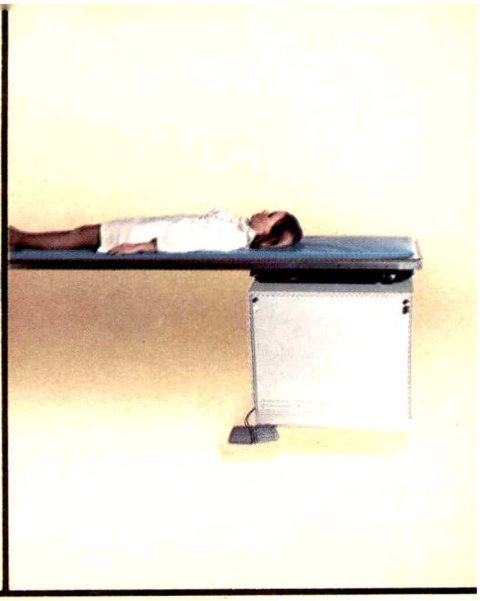
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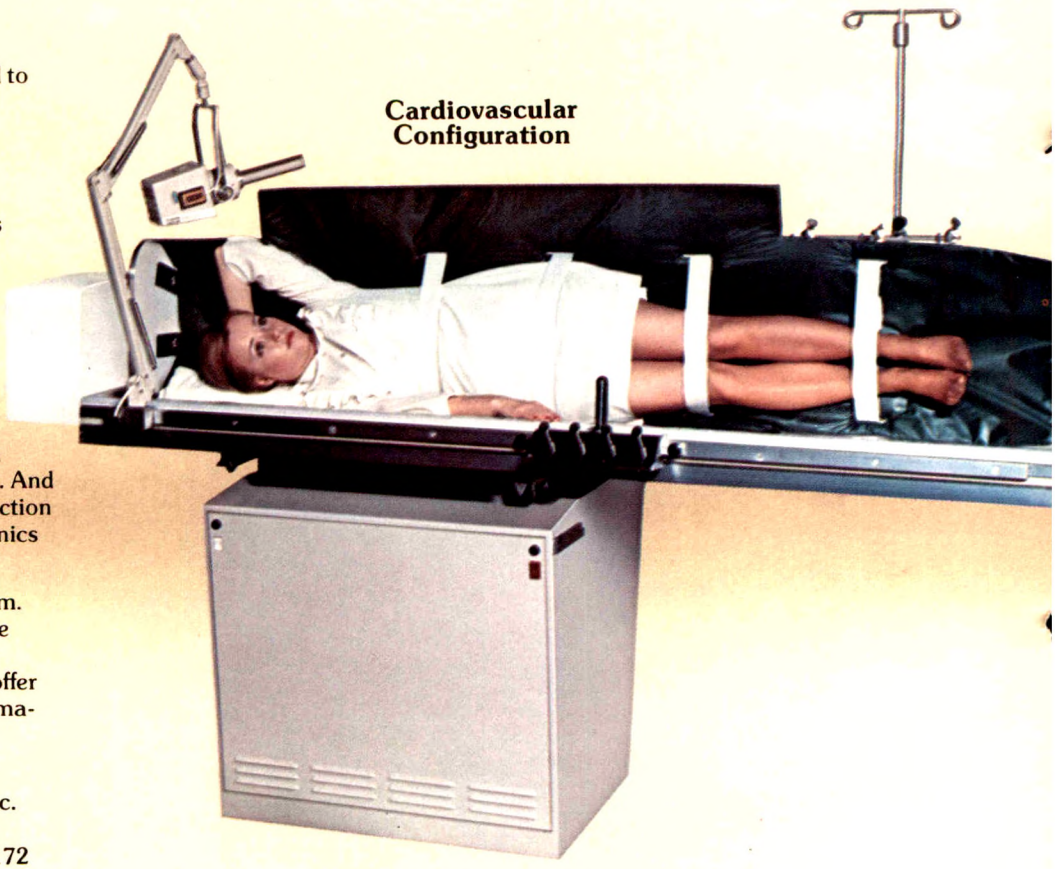
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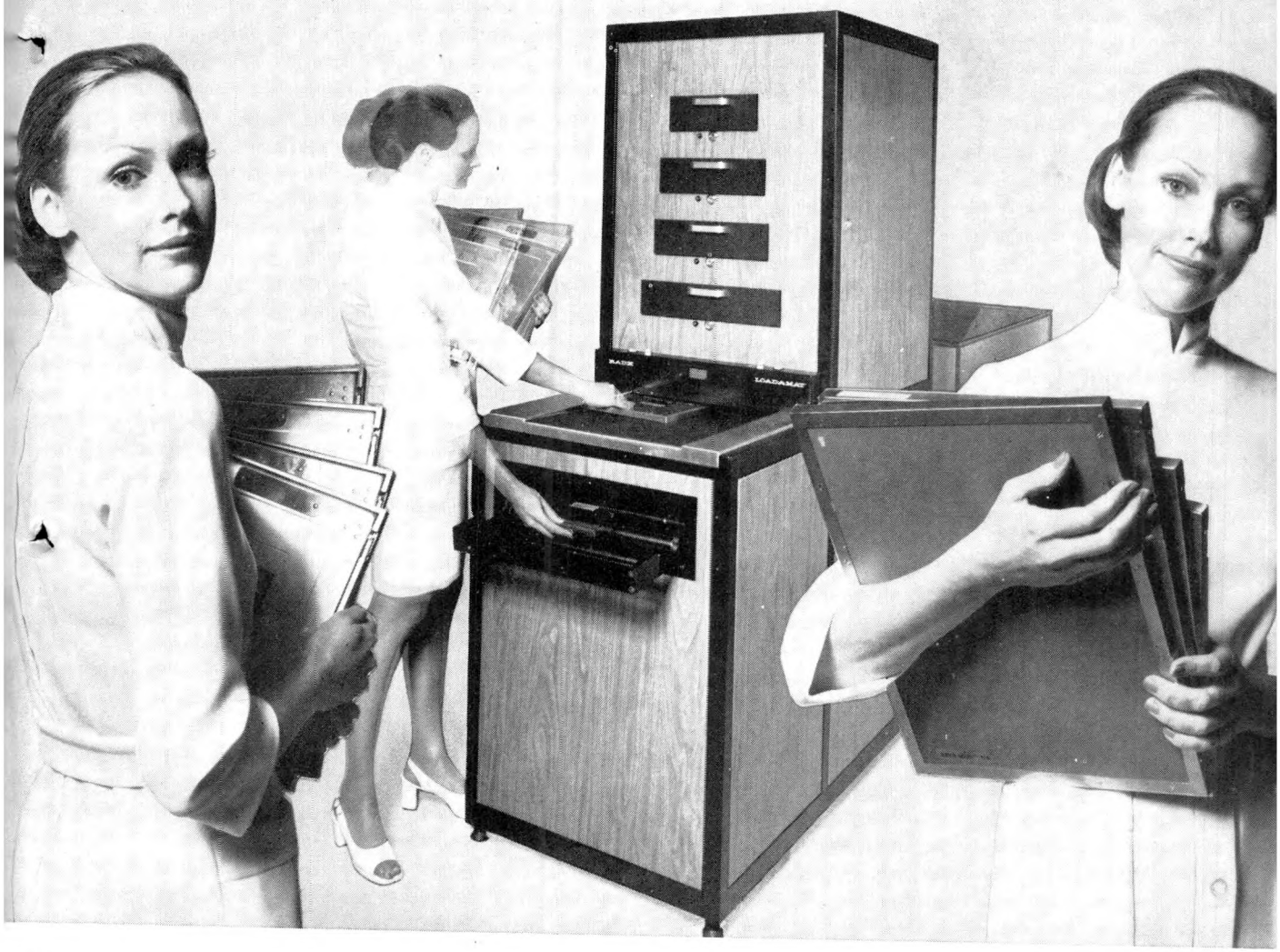
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To view and quantify patient count information in black and white or fully functional color, Maxiscan can be combined with GE's Videodisplay and Processing Unit. Images are displayed on a video monitor; count data is stored in the unit's electronic memory, and can be manipulated to enhance desired details and to aid interpretation and diagnosis. Enhanced VDP data may be played back to Maxiscan and recorded on 14 x 17 inch film. Scans can also be recorded on cassette tape for off-line playback and teaching purposes. Count information, obtained from any scanner or camera, can be transmitted from one VDP to another over regular telephone lines.



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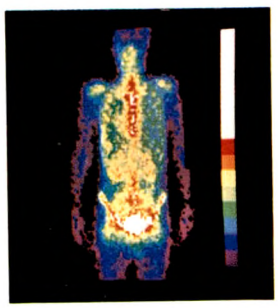
Here's the information hospitals are getting with Maxiscan...

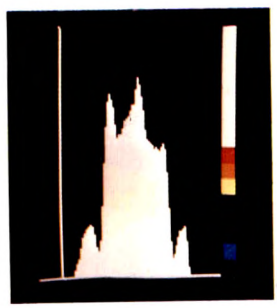
Hospitals report scanning performance like this from the Maxiscan system:

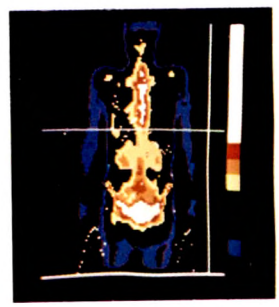
These reproductions of scans, from clinical examinations, illustrate the range of diagnostic information possible with Maxiscan and the Videodisplay Processor.

A GE motion picture demonstrates the full capability of both units. Ask your GE representative to schedule a desk top showing, at your convenience.

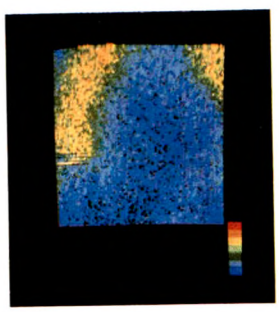


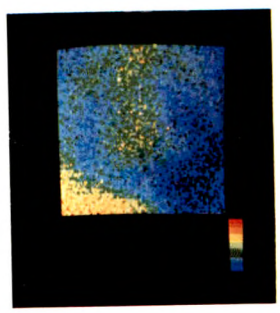


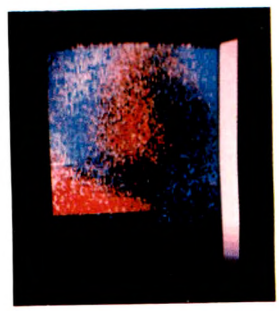




These three images, from a single whole body scan, demonstrate how manipulation of data stored in the VDP electronic memory can enhance desired details and aid diagnosis. The isotope used was ^{99m}Tc Polyphosphate. At left, an anterior view displays raw, unmanipulated data from the memory. At right, smoothed data is shown with a Y axis electronic slice through the area of suspicion. The count profile superimposed over this image and shown separately, center, confirms greater uptake on the right side. The photorecorded image showed only a suspicion of greater isotope uptake.





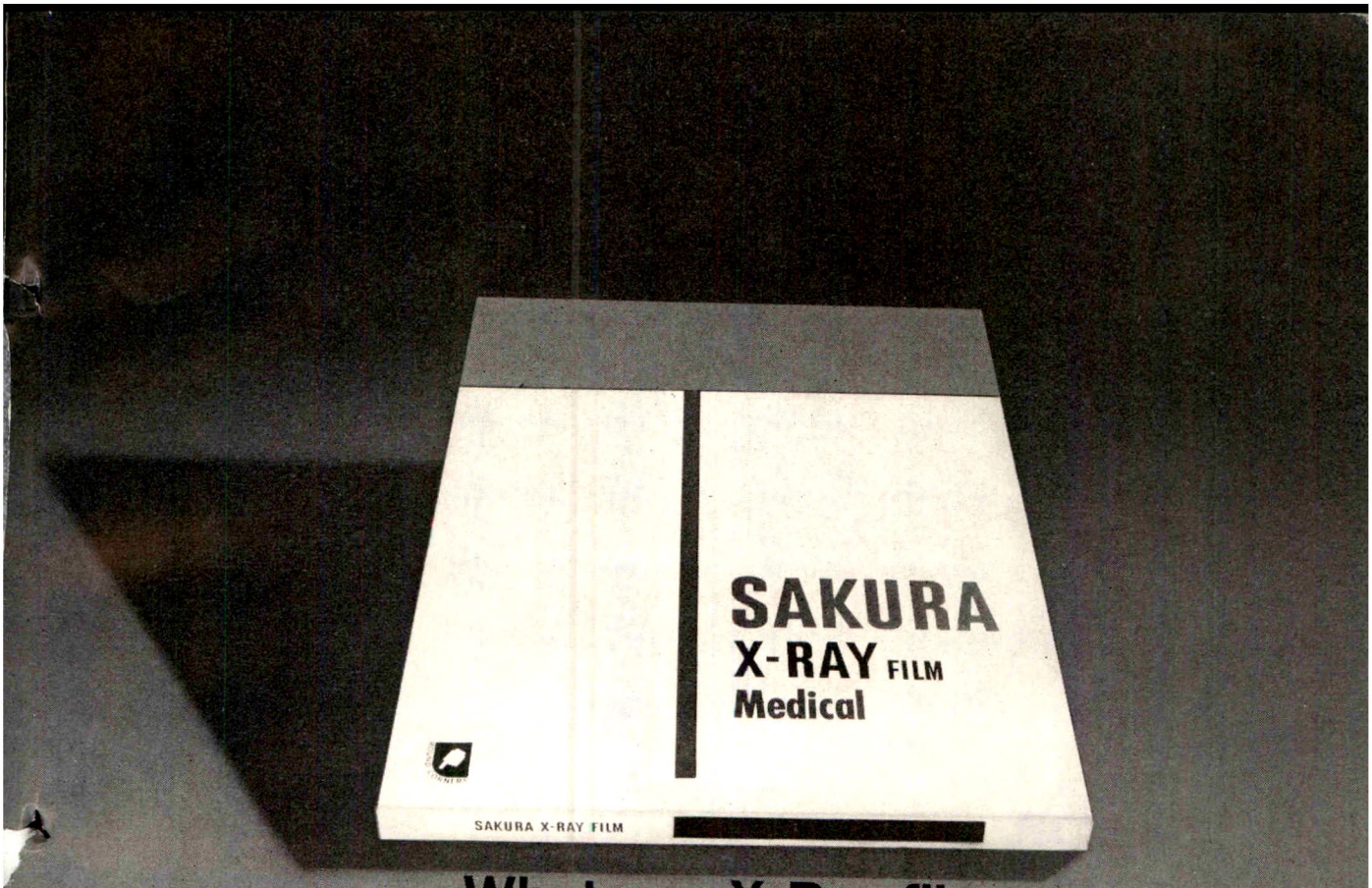


In a case of suspected pericardial effusion, a transmission scan (left) of the chest was obtained using an Iodine 131 source. An emission scan (center) of the same region was simultaneously obtained with the same probe, 15 minutes after an intravenous injection of ^{99m}Tc labeled albumin. The heart and liver are outlined. Note how the intracardiac activity (central area of center scan) fails to fill the large mediastinal shadow (central blue area of left scan). This discrepancy, between heart size and that of the mediastinum, is more easily seen when these two scans are superimposed (right); a technic easily accomplished on the VDP. The resulting diagnosis, a large pericardial effusion which appears to be predominantly left-sided, was confirmed by the aspiration of 1800 ml. of fluid from an encysted pericardial effusion.

Scans courtesy of Dr. M. J. Chamberlain, University Hospital, London, Ontario.

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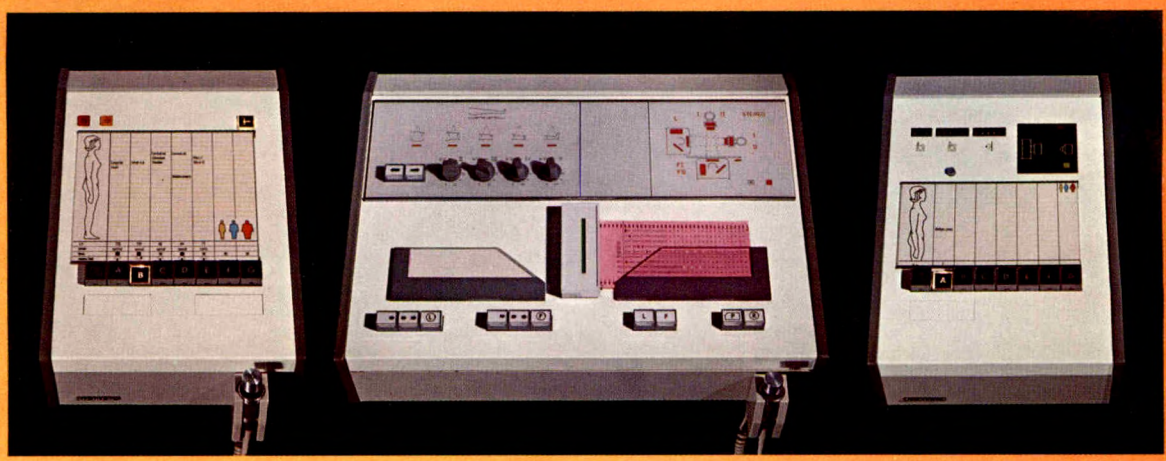
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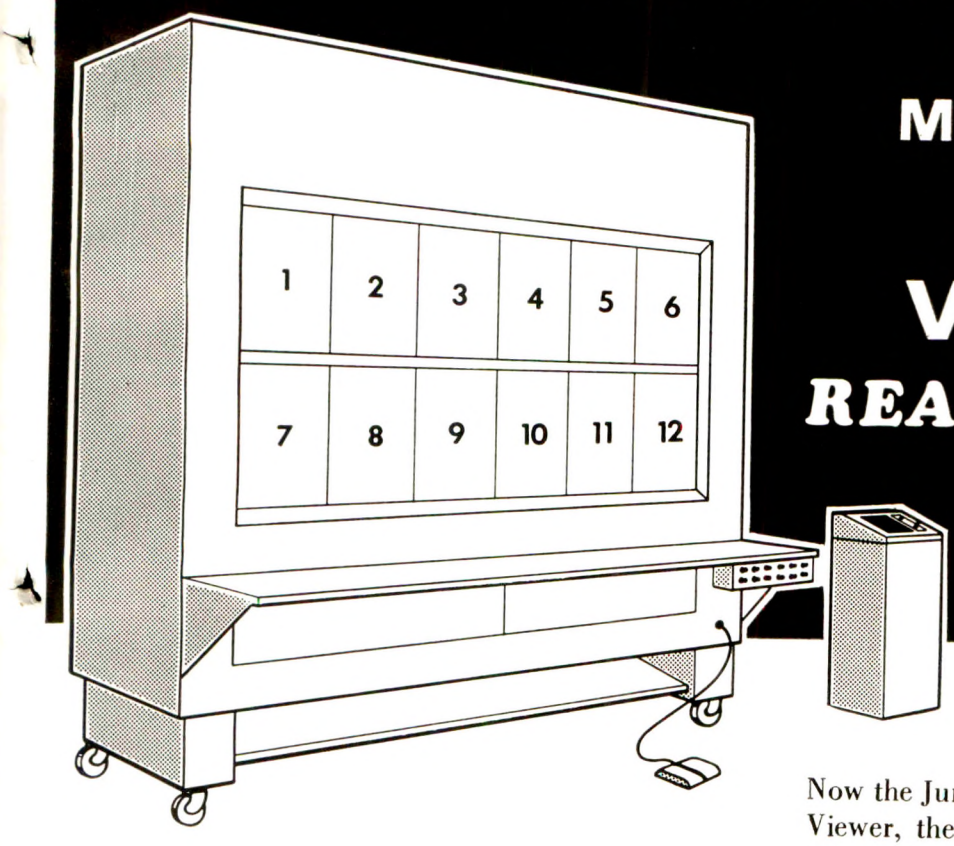
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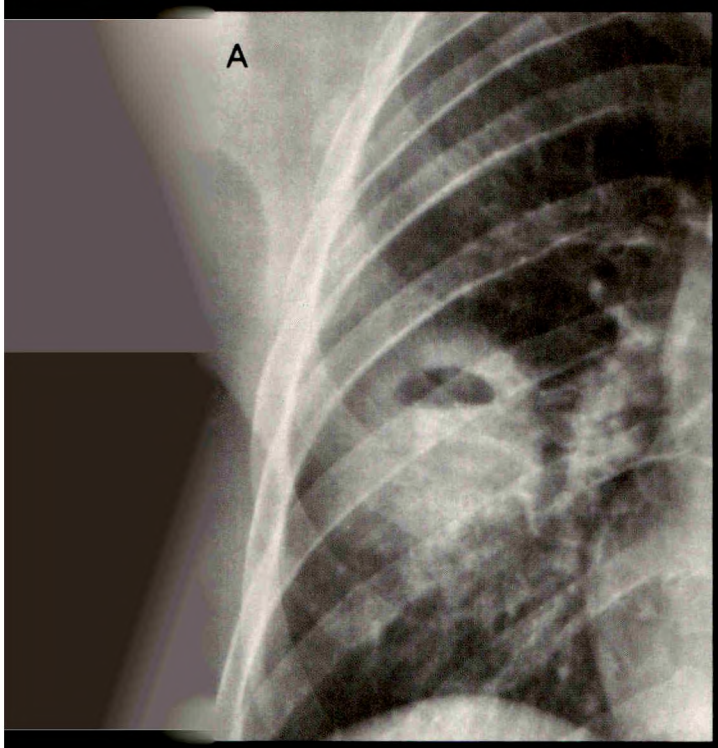
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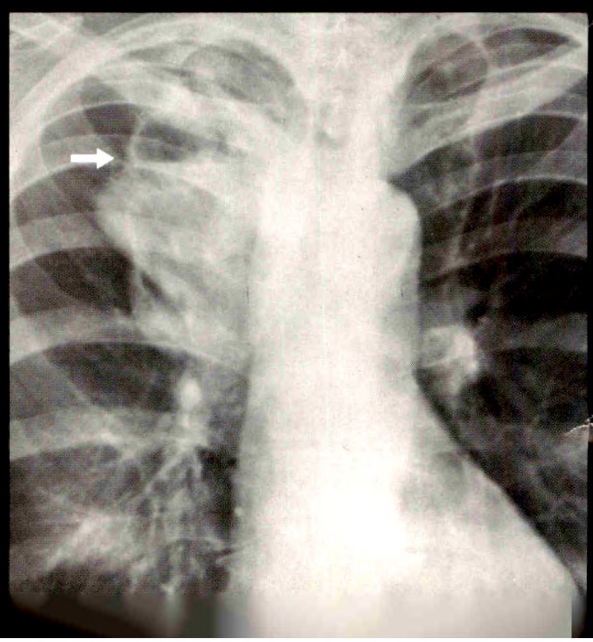
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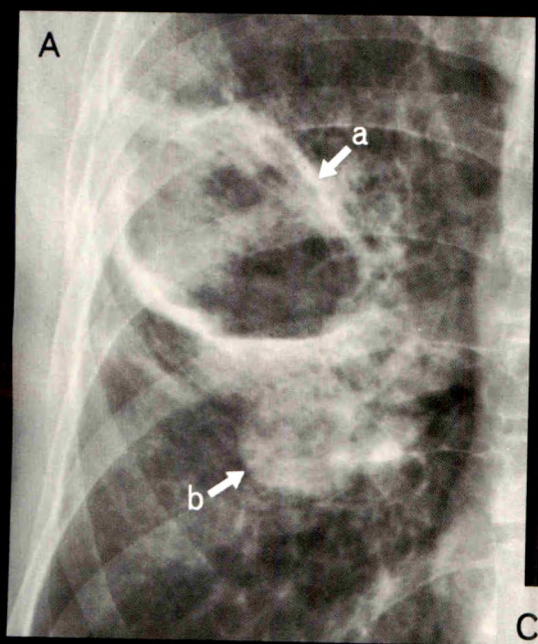


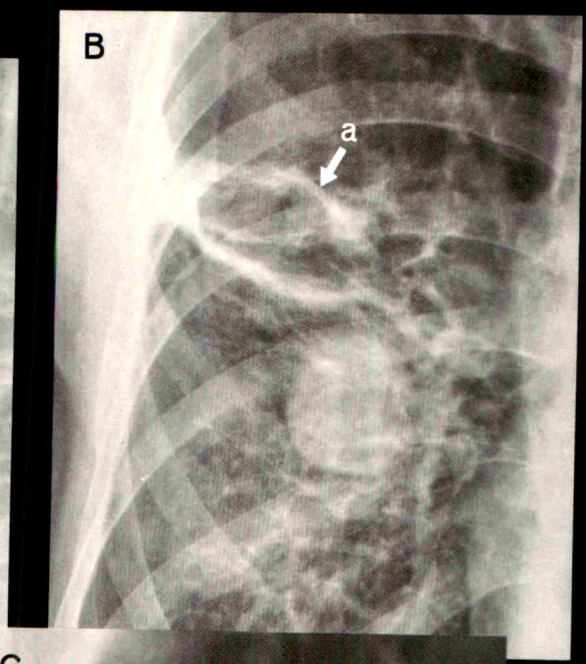


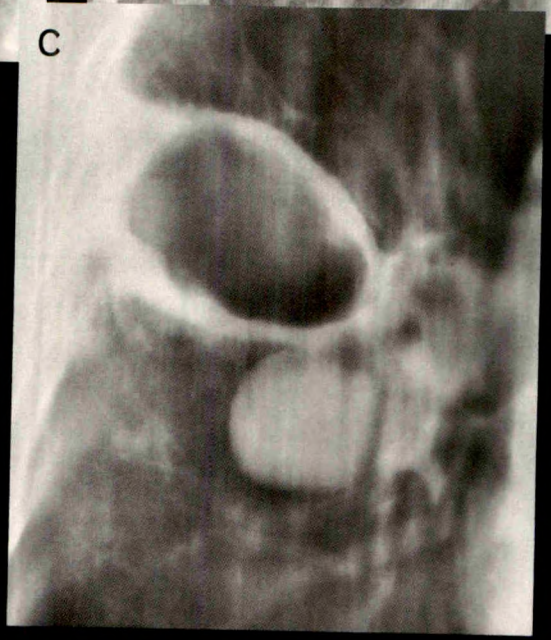
Case II—This 50-year-old man complained of chills, fever, and rusty sputum for many months. Whereas but one cavity with a fluid level is demonstrated here (arrow), his squamous cell carcinoma proved to contain several separate cavities.



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Case III—This 55-year-old man, a heavy smoker, complained of right-side chest pain, dyspnea, and a productive cough for several months.

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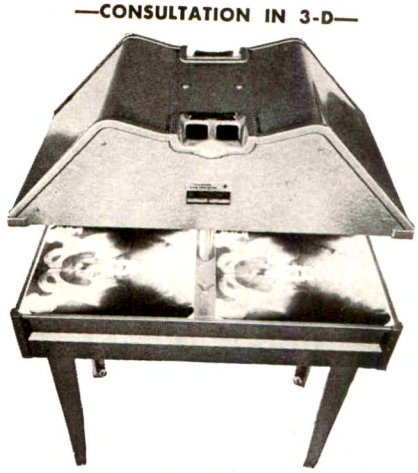
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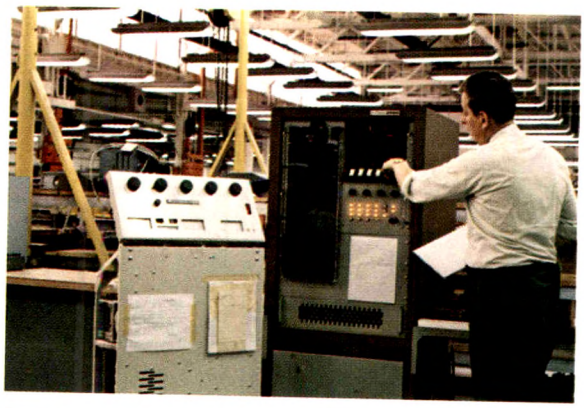


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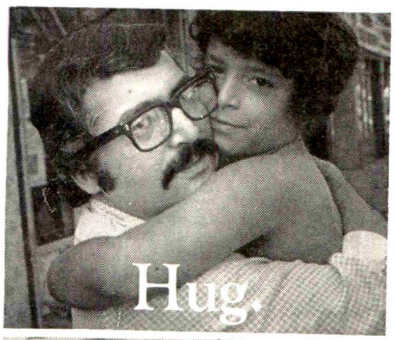
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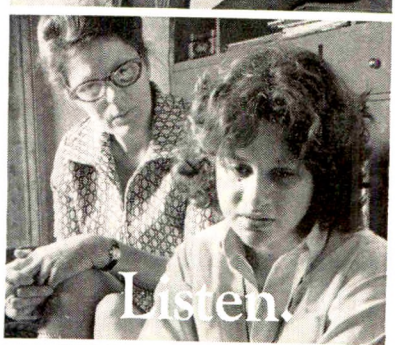
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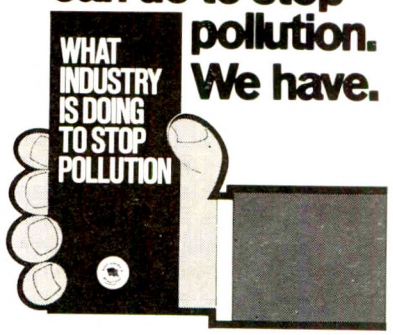
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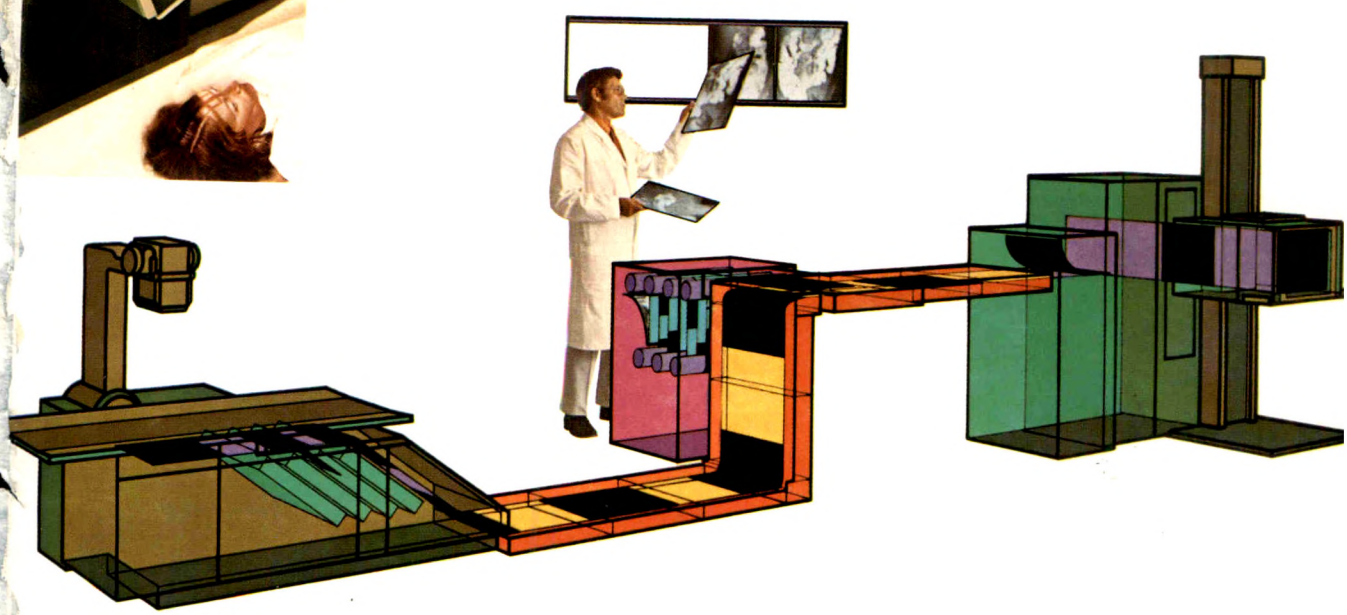


Picker then followed up with the Rapido®, the first practical application of the cassetteless concept to horizontal Bucky radiography. With the RapX™ modular film conveyor, these systems handle about 70% of the patient load in a busy radiology department. And they do it without cassettes and with superior radiographic quality.

Picker cassetteless radiography is the proven way to go all-cassetteless. And Picker cassetteless radiography is fully backed by Picker service to keep your busy department busy.

Picker Corporation, 595 Miner Road, Cleveland, Ohio 44143





Another reason to buy from...

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INDEX TO ADVERTISERS

Alderson Research Laboratories xx
Applied Radiation Corporation ..... x, xi
Atomic Energy of Canada, Ltd. ..... Inside Front Cover
CGR Medical Corporation ..... xxxviii, xxxix
Eastman-Kodak ..... xxxiv, xxxv
Elema-Schonander ix
E-Z Em xvi
General Electric ..... xxvi, xxvii, xxviii
John F. Kennedy Medical Center xxxii
Liebel-Flarsheim xv
Low X-Ray ..... xxiv, xxv
Machlett Laboratories viii
Donald McElroy, Inc. xiv
Micro X-Ray Recorder xxxvii
Ortho Pharmaceuticals ..... Back Cover
Picker Corporation ..... xviii, xix, xlii, xliii
Procter & Gamble ..... xii, xiii, xiv
Purdue-Frederick v
Radx xxiii
Sakura Medical Corporation ..... i, xxix
Schneeman Electronics xxxvii
Siemens Corporation ..... xxx, xxxi
Searle Radiographics ..... xvii, Inside Back Cover
S & S X-Ray xxxiii
3-M Company ..... vi, vii
Thomas, Charles C, Publisher ..... xxxii, xl
Xonics, Inc. ..... xxi, xxii

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Searle puts it all together...with the new Micro Dot Imager.

A new Multi Imager that produces up to 80 images on a single film.

It's taken us some time but at last we can offer Pho/Gamma users a display system that puts it all together. Gone is the expensive and tediously inaccurate pulling of Polaroids. Gone is the unreliable and complicated 35 or 70 mm mechanical transport system.

The new Micro Dot Imager electronically minifies and manipulates the images across the CRT screen and displays them on a choice of three different conventionally sized X-ray films... handled and processed with conventional techniques.

In addition the new Micro Dot Imager provides the following exclusive benefits.

Clinically Oriented

- Choice of either 5 x 7 or 8 x 10 X-ray film sizes as well as the competitively available 11 x 14 film size.
- Built in whole body imaging with choice of each view presented in dual intensity on the film to facilitate diagnosis or the more economical two views with single intensity.
- Highest cine sequential time per frame resolution of up to 80 frames/study.
- Organ-specific push buttons automate, standardize and speed the proper exposure settings for routinely performed studies.

Simplified Operation

- Light emitting diode (LED) display indicates system status and exposures available for format selected as well as exposures remaining on the individual film.
- Absolute exposure control insures consistent day to day and week to week exposure levels on a separate but built in high resolution, high uniformity CRT.

Economical Operation

- A variety of film sizes guarantees the lowest operational cost of any imager offered.
- System designed lightweight, low cost cassettes with future daylight unloading capability.
- Built in view-box saves space as well as steps.

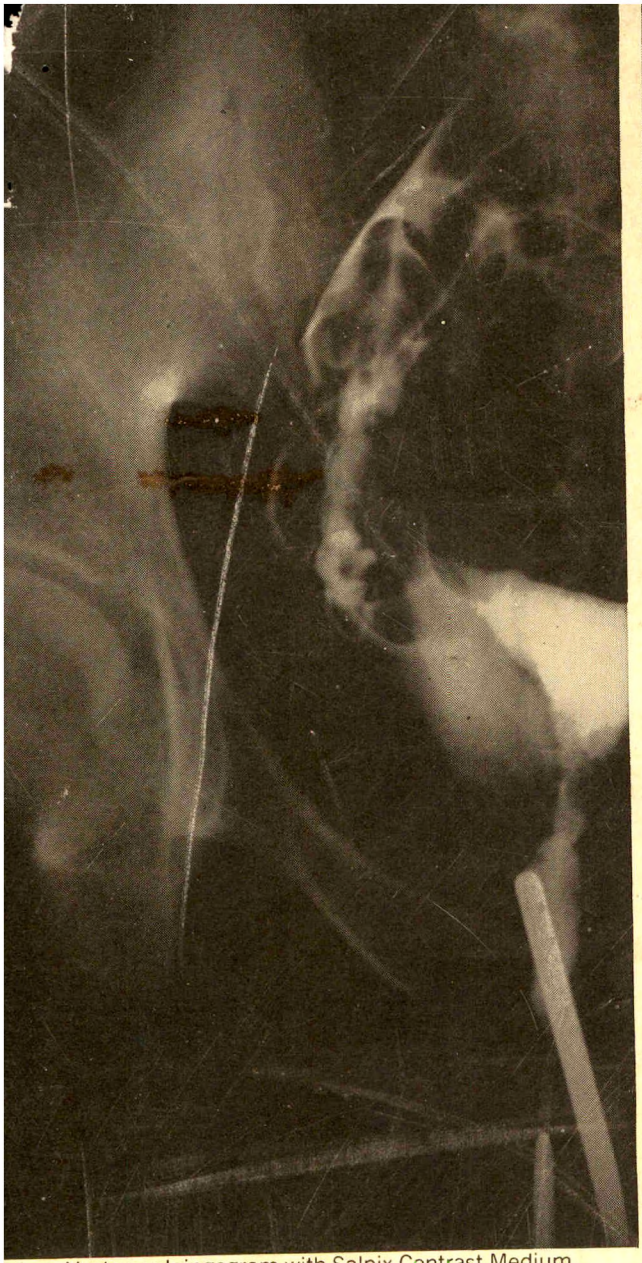
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CM-342



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